Asia Research Institute
Working Paper Series No. 205

Exploring Reverse Causational Effect of Fertility on the Infant Mortality Decline in India

P. Arokiasamy
Department of Development Studies
International Institute for Population Sciences, India

Srinivas Goli
Giri Institute of Development Studies, India
sirispeaks2u@gmail.com
(Corresponding author)

Mohd Shannawaz
International Institute for Population Sciences, India

August 2013

The **ARI Working Paper Series** is published electronically by the Asia Research Institute of the National University of Singapore.

Citations of this electronic publication should be made in the following manner: Author, "Title," ARI Working Paper, No. #, Date, www.nus.ari.edu.sg/pub/wps.htm. For instance, Smith, John, "Ethnic Relations in Singapore," ARI Working Paper, No. 1, June 2003, www.ari.nus.edu.sg/pub/wps.htm.

Asia Research Institute
National University of Singapore
469A Tower Block #10-01,
Bukit Timah Road,
Singapore 259770
Tel: (65) 6516 3810
Fax: (65) 6779 1428
Website: www.ari.nus.edu.sg
Email: arisec@nus.edu.sg

The Asia Research Institute (ARI) was established as a university-level institute in July 2001 as one of the strategic initiatives of the National University of Singapore (NUS). The mission of the Institute is to provide a world-class focus and resource for research in the Asian region, located at one of its communications hubs. ARI engages the social sciences broadly defined, and especially interdisciplinary frontiers between and beyond disciplines. Through frequent provision of short-term research appointments it seeks to be a place of encounters between the region and the world. Within NUS it works particularly with the Faculty of Arts and Social Sciences, Business, Law and Design, to support conferences, lectures, and graduate study at the highest level.

Exploring Reverse Causational Effect of Fertility on the Infant Mortality Decline in India

INTRODUCTION

The long-term progress in India's demographic transition since its onset in the late 1960s, particularly the fertility decline over the past half a century, is known to be mainly the consequence of proactive implementation of family planning programme. A clear demonstration of this claim is that India's recent fertility transition was driven by the rising contraceptive prevalence and major fertility declines in the 1990s and 2000s among women who are poor and illiterate (Bhat, 2002; McNay et al., 2003). This turnaround contrasts the earlier preoccupation with women's education and socioeconomic conditions as key determinants of fertility decline necessitating a steady shift of focus to research on the study of reciprocally initiated positive contribution of fertility decline to the improvement in the health of women and children (Arokiasamy, 2009). The best illustration of this is, the Indian states of Andhra Pradesh and Orissa, which display clear evidence of faster fertility decline than the infant mortality decline in the past decades (James, 1999; IIPS and Macro Internationals, 1992-2006; Office of Registrar General of India, 1971-2007). Such exceptional contra trends suggest a fertility decline as a potential driver of infant mortality decline during the current phase of India's fertility transition. Despite such steady reversal of causal connections, evidence based assessment of effect of 'fertility on child mortality, hypothesis has remained dormant vis-à-vis the long-term engagement with 'child mortality effect on fertility' hypothesis: This paper therefore, seeks to systematically assess the reverse causation hypothesis of fertility effect on infant mortality decline in the Indian context; one amongst those countries, that has experienced a steady fertility decline with incommensurate progress in socioeconomic structural conditions.

THEORETICAL BACKGROUND AND RATIONALE

Though demographers continue to show great interest in studying the relationship between fertility and child mortality, the literature is scarce in terms of evidence based analysis of the interrelationship among these two key variables. The prevailing demographic theoretical assumption states that high fertility is a biological and behavioural response to high mortality. Under this premise, a large number of past studies have contended that high levels of childhood mortality are closely tied to high fertility.

The classical demographic transition theory postulated that reduction in early childhood mortality has been the critical stimulus for the onset of fertility decline, driving forward the process of demographic transition. This assumption is also manifested in the theory of change and response, child survival and child replacement hypotheses amongst others (Davis, 1945, 1963; Taylor et al., 1976; Temkin-Greener and Swedlund, 1983). However, many studies have held contrary inferences, where evidences of the reverse sensational effect of fertility decline on infant mortality reduction has been sequentially documented during the middle stages of the demographic transition (Thorne and Green, 1976; LeGrand and Phillips, 1996; Woods et al., 1998; Arokiasamy, 2007).

The notion of reverse causation is based, in large part, on the belief that the decline in fertility may systematically alter the patterns of family building, birth spacing, parity and mother's age at childbirth-in ways that are potentially beneficial to the mother's and children's health (Thorne and Green, 1976; Woods et al., 1998). However, Bongaarts (1987), contested this point of view arguing that the presumption of a strong, positive effect of fertility decline on infant survival, acting through

these mechanisms, was unwarranted. As LeGrand and Philips (1996), have pointed out, Bongaarts argument was focused on two dimensions: first, while fertility reduction clearly alter the demographic patterns of childbearing, the net effect of these changes on infant survival is not necessarily beneficial[1]. Secondly, he argued that the simple statistical association between the pattern of childbearing and infant–health risks may overstate the importance of variables such as mother's age and birth intervals. Other factors correlated with these variables may be responsible for much of their apparent effects. Several researchers have strongly contested this uncertainty hypothesis, drawing attention to a great deal of research that has concentrated on improving our understanding of the causal linkages between birth spacing, mother's age, parity and children's health risks (Hobcraft, 1987; Trussel, 1988; Potter, 1988).

Recent evidence in developing countries suggests that during the middle course of demographic transitions high mortality could be a measured response to high fertility, instead of a stimulus, parallel to what the long-term trends demonstrated in the context of developed countries experience. The developed countries' experience could have been no exception to this. Sanderson's (2000), a study based on panel data analysis for England for the period 1880-1940 showed that infant mortality decline seemed to be an important cause of fertility decline between 1880 and 1910 but not between 1910 and 1940. However, he concluded somewhat reassuringly that the cause of fertility decline in the modern world may be different, at least to some extent, compared to those in the original demographic transition. However, he was silent on a particular causal factor of mortality decline.

Several other studies[2] that focused on the effect of family planning on infant mortality decline point out that the biological and economic effects of high fertility on mortality may well be recognized via close spacing of births, marriages at young ages, poor nutritional status and low per capita household availability of resources due to large family size. The death of a child has been found to be often followed by a shorter interval to the next birth, which may be explained in terms of a behavioural choice, that is, volitional replacement or else by a biological process, the fact that the mother stops breastfeeding and, thereby, is able to conceive the next child sooner than otherwise (Cleland and Sathar, 1984; Curtis et al., 1993). A short birth interval, in turn, seems to increase the mortality risk of the next child in the family, because the mother has not recuperated physiologically from the previous birth. And this risk becomes exaggerated at shorter birth intervals amongst higher order births. In the course of these family building processes, the incidence of infant deaths consistently increased with the rise in fertility level resulting in reverse causation effect. On the other hand, improved access to use of family planning methods would enable women to reduce closely spaced births, limit childbearing, and reduce their chances of having a baby who dies in infancy (Bongaarts and Potter, 1983; Preston, 1985; Pritchett, 1994; Rutstein, 2005).

The two conflicting theoretical arguments are akin to the customary population debate concerning a two-way sectional association between population growth and economic development. Most past studies majorly dealt with the negative consequences of population growth on economic growth (Coale and Hoover, 1958). In recent times, however, on a positive note, researchers have focused on evaluating the evidence of potential positive benefits of demographic variables. Two such critical areas include: a) studies devoted to measuring the demographic transition effect on the

[1] The proportion of births that are of high parity or born to teenage or relatively older women, all correlated with high infant mortality, tends to rise as fertility falls.

[2] Contemporary expert views on the debate whether how far family planning and consequent fertility decline effect the child mortality and maternal mortality has been documented by PRB (2009) and Rhonda et al. (2009).

demographic dividend (Bloom and Williamson, 2000) and b) the renewed effort to measure true benefits of both direct and indirect effects of fertility decline on women and child health, poverty and social welfare (Arokiasamy, 2009; Hongladarom, et al., 1987; Payne, 2004). The lack of progress in understanding the range of mortality-fertility linkage through a dynamic theoretical paradigm has led to failure in recognizing the true repercussions of fertility decline in reaping the potential health and development benefits.

Persistent failure to address reverse causation hypothesis is likely to mask the true casual connections, underplay fertility effect on mortality, and overestimate the child mortality effect potentially in the later phase of fertility decline. Exploring the way in which biological and behavioural factors shape this relation at the family level is crucial for understanding the causal pathways of demographic transition. India's long history of proactive family planning programmes and the varying pace of demographic transition across the states provide a perfect setting to unfold the two-way causal connections between fertility and child mortality trends. Surprisingly, there is a virtual void of evidence based assessment of demographic transition in India that deals with the potential effects of fertility decline in lowering child mortality. In an effort to fill this gap, in this paper we delve into evidence of 'fertility effect on infant mortality' hypothesis and explore the pattern of infant mortality differentials by fertility related determinants.

SOURCES OF DATA

Data from multiple sources: the Sample Registration System (Office of Registrar General of India, 1971-2007), Census of India (1971-2001) and the Centre for Monitoring Indian Economy (CMIE, 1971-2001) are used to explore the macro level reverse causal effect of fertility on infant mortality. Data from the recent National Family Health Survey-3 (IIPS and Macro International, 2005-06) are used for micro level analysis to explore the causal connections of fertility effect on infant and child mortality indicators.

Specification of the Statistical Models

The analysis of this paper is structured in three sections. First, we examined macro level trend data to understand the way in which trends in fertility and mortality decline are possibly interconnected coinciding with the onset point of fertility decline in India. Additionally, we estimated ordinary least square regression and multivariate panel data regression models with time-trend and cross-sectional (by states) to examine the effect of fertility decline on disaggregated childhood mortality indicators decline. Second, we used micro data (NFHS-3) to explore a number of potential reverse causational pathways of fertility decline on child mortality decline. We estimated Cox proportional hazard models to evaluate the relative importance of fertility vis-à-vis other mortality determining factors on childhood mortality. Thirdly, we constructed graphic plots of Kaplan-Meier estimation of the survival function and the hazard function of micro data to distinguish the effect of the determinants of fertility level on the infant mortality level.

Panel data regression model

We estimated panel data regression model on time trend over and cross-sectional across data to examine reverse causational effect of fertility on infant mortality. A panel data regression model differs from regular time-series or cross-sectional data regression because of double subscript on its variables (Baltagi, 2005). The general form of panel data regression is represented by the equation:

$$y_{it} = \alpha_i + X_{it}\beta + u_{it} \quad i = 1 \ldots \ldots \ldots N;\ t = 1 \ldots \ldots \ldots T$$

With *i* denoting individuals, households, state etc. and *t* denoting time. The *i* subscript, therefore, denotes the cross-sectional dimension, whereas *t* denotes the time-series dimension, α_i is a scalar, *β* is Kx1 vector, X_{it} is the it^{th} observation on *K* explanatory variables and u_{it} is the error component. We estimated both the fixed and random effects models to assess the effect of fertility on infant mortality.

The fixed effects model is an adequate specification when focusing on a specific set of *N* individuals.The fixed effects model is denoted as:

$$y_{it} = \alpha_i + X_{it}\beta + u_{it}$$
$$u_{it} = \mu_i + v_{it}$$

Where , u_{it} Represent individual-specific time-invariant effects. In this case, the μ_i are assumed to be fixed parameters to be estimated and the remainder disturbances stochastic with v_{it} independent and identically distributed IID (0*, σ2*). The X_{it} are assumed independent of the v_{it} for all *i* and *t.* There are too many parameters in the fixed effects model and the loss of degrees of freedom can be avoided if the μ_i can be assumed random. In this case μ_i ~IID (0*, σ2*),v_{it}~IID (0,*σ*2) and the μ_i are independent of the v_{it}. In addition, the X_{it} are independent of the μ_i and v_{it}, for all *i* and *t*.

The random effects model is an appropriate specification if we are drawing *N* individuals randomly from a large population. Two stipulations are important to note: a) if the unobserved variable does not change over time, then any change in the dependent variable must be due to influences other than the fixed characteristics and b) when time-series cross-sectional data is used, the interpretation of the beta coefficients would be-for a given country, as *X* varies *across time* by one unit, *Y* increases or decreases by *β* units.

Pooled observations of cross-section data across states of India over three time periods of 1981, 1991 and 2001 – the period of major fertility decline in most states – have been used. The *β* coefficients of panel data regression models have been estimated with their respective set of predictors of infant mortality rate. The predictors included in the models are: total fertility (TFR) and relevant covariates such as female literacy rate, per capita state domestic product (SDP), and percentage of males employed in non-agricultural sector, female work participation rate percentage of urban population, sex ratio of the population and period dummy variables of 1981-91 and 1991-2001.

Kaplan-Meier estimation of survival function and hazard function

Kaplan-Meier estimates of the survival function are used to plot survival curves in order to depict the proportion of children who would survive a given length of time by fertility related characteristics of the mother. Kaplan-Meier estimate of survival function is expressed as the survivor function (S):

$$S(t) = \frac{number\ of\ individuals\ surviving\ longer\ than\ t}{total\ number\ of\ individuals\ studied} = \Pr(T > t)$$

Where *t* is time, *T* is a random variable denoting the time of death. Cumulative hazard function plots have also been generated to show the probability a child will experience death within a small time interval, given that the child has survived until the beginning of the interval. This is interpreted as the risk of dying at time t by fertility characteristics of the mother. The hazard function is denoted H (*t*), and is defined as the event rate at time *t* conditional on surviving until time *t* or later (that is, $T \geq t$), H *(t)* can be estimated using the following equation:

$$H(t) = \frac{Number\ of\ infant\ or\ children\ experiencing\ deaths\ in\ interval\ begining\ at\ t}{(Number\ of\ individual\ surviving\ at\ time\ t)\ X\ (interval\ width)} = \frac{S'(t)dt}{S(T)}$$

Cox proportional hazard regression model

Cox proportional hazard model (Cox and Oakes 1984) is estimated to determine the effect of fertility related indicators on child mortality indicators using NFHS-3 data. Infant and child mortality are the respective dependent variables for model estimation of the effect of fertility indicators on child mortality indicators. Mother's fertility related indicators used in the models as independent variable (x) include: age of the mother at child birth, birth interval for successive birth(s) and parity at sterilization. Related socioeconomic indicators are incorporated as control variables in the model estimation of absolute effect of fertility on child mortality indicators.

The mathematical form of the hazard model is expressed in the following equation

$$h_i(t) = h_0 \exp\ (\beta_1 x_{i1} + \beta_2 x_{ik} \ldots\ldots\ldots\ldots\ldots\ldots\ldots \beta_k x_{ik})$$

where, '*i*' is a subscript for observation, and the '*x*'s are the covariates (e.g. age of the mother, parity at sterilization, birth order, birth interval and sex of the child). The quantity h_0 (*t*) is the baseline or an underlying hazard function and corresponds to the probability of dying when all explanatory variables are zero. The baseline hazard function is analogous to the intercept in ordinary regression (since $\exp^0=1$)

The regression coefficient $\beta_{parity\ at\ stergilization}$ gives the proportional change that can be expected in the hazard, related to the change in the explanatory variable (parity at sterilization). The Cox proportional regression model assumes that the hazard of death at time t for the children of women with parity more than two (z) is proportional to the hazard of the children of mothers with parity two or less than two (y) by the same factor ψ at every time t; mathematically expressed as following equation

$$h_z(t) = \psi h_y(t)$$

Where h_z *and* h_y, the hazards (probabilities of dying) are for the two groups of children and ψ is the hazard ratio. If ψ>1, the hazard of child death is higher for children of mothers with more than two parities relative to those with two or less than two parties. If ψ<1 or ψ =1, the hazard of child death is smaller or equal for children of mothers with more than two parities relative to those children whose mothers have two or less than two parties, in which case parity of the mother would have not shown any effect (ψ =1) or may be positively related to child deaths.

RESULTS

Trends in Fertility and Mortality Rates in India: An Exploration of Reverse Causation

In India's demographic transition history, the year 1971 represents a most critical onset point of fertility decline as the ensuing period was a period of substantial and irreversible fertility decline (figure 1), while during the same period, mortality decline was not as sharp as fertility decline. This pattern prevailed more or less during the following decade with the trend line suggesting that the birth rate has been declining faster than the death rate (Figure 1).

Figure 1. Trend in Crude Birth Rate (CBR) and Crude Death Rate (CDR) of India, during 1971-2007

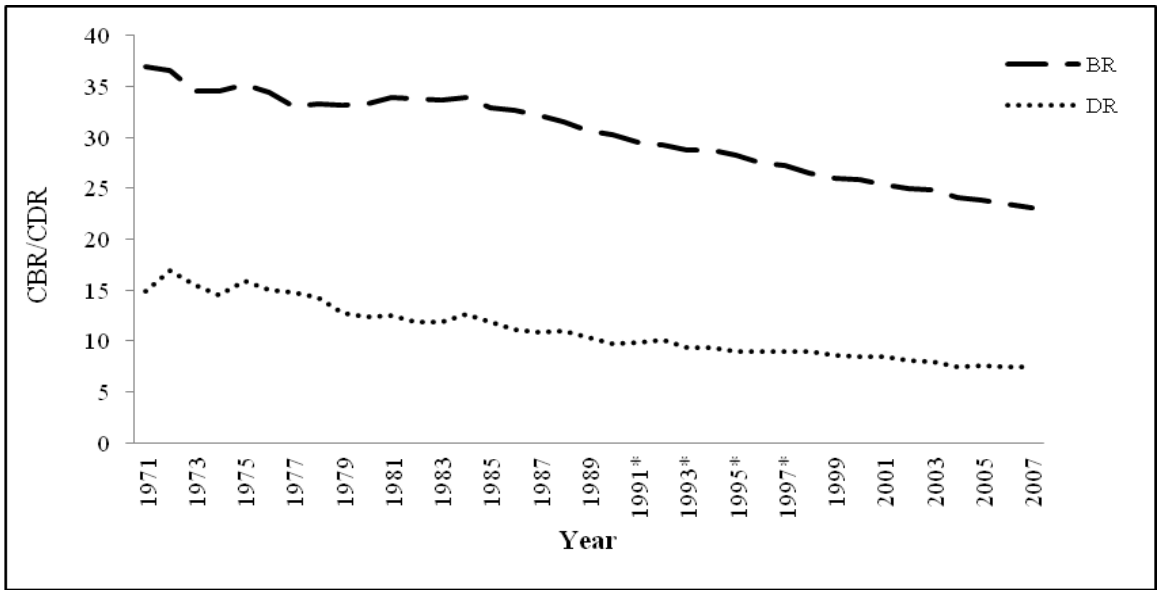


Figure 2 shows comparative trends in fertility and disaggregated infant mortality indicators for India. Trend lines reveal that at the initial stage, most indicators of the infant mortality component show a slower pace of decline than the decline in total fertility rate (TFR). During 1971-1981, the total fertility rate showed steady decline while critical components of child mortality indicators, such as neonatal mortality (NNMR) and perinatal mortality rate (PNMR) were plateauing or even rising. This trend signifies that the sustained fertility decline in India following its onset in 1971 was more likely to be a causal factor of mortality decline. However, in mid stages of the demographic transition in the 1980s and 1990s, a two-way causal relationship is a more plausible outcome, implying that both fertility and infant mortality had mutually impinging effects.

Figure 2. Comparative Trends in TFR and Disaggregated Indicators of Infant Mortality for India, 1971-2007

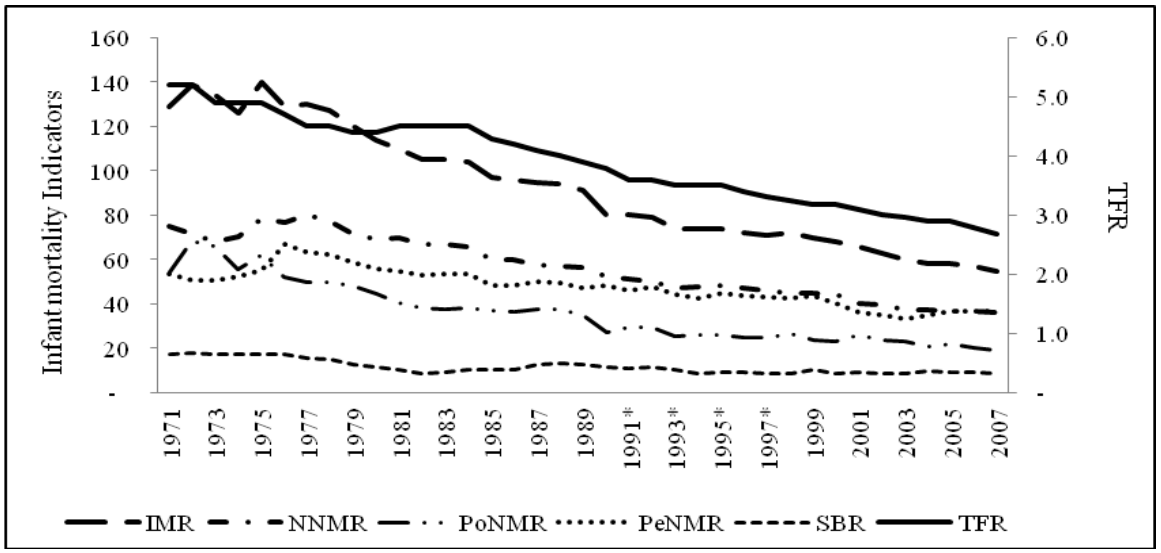
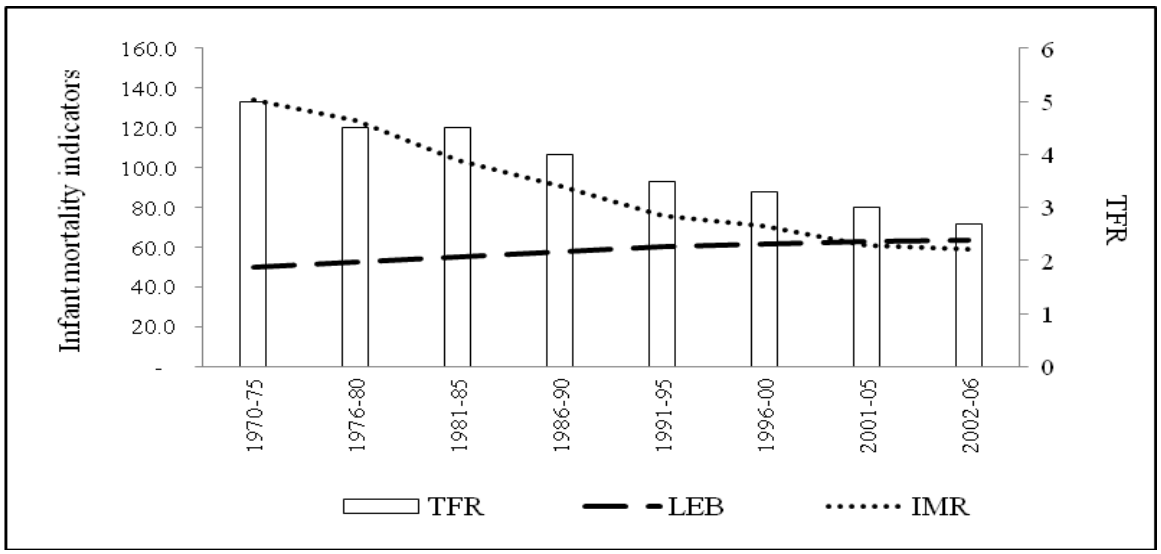


Figure 3 displays comparative trends in TFR, life expectancy at birth (LEB) and IMR. Trends point to a sharper decline in TFR preceding IMR decline and the rise in LEB. However, for the recent time period of 1991-2001, both fertility and mortality decline were stagnant with more visible stagnation of IMR than TFR; consequently, the rise in life expectancy was also seen to be stagnant.

Figure 3. Trends in TFR, Life Expectancy at Birth and Infant Mortality Rate for India, 1970-75 to 2002-2006



We further examined trends in high (child mortality) risk births. Births to mothers of younger or older age group mothers and higher order births are important demographic determinants that sustain high infant mortality. Births to mothers in younger or older ages and births of higher order births are termed as high child mortality risk births (Bingley et al. 2000); as such births tend to increase the proportion of underweight children, children with birth defects and deliveries with complications all of which add to a greater risk of infant deaths and stillbirths.

Figure 4 and 5 suggest a number of intriguing trends and patterns. Infant mortality decline accelerated from 1981 (figure 3) and this was more likely to be the result of dramatic reduction in the share of high risk births - child bearing at lower age and higher order births - in the mid 1970s. Childbearing after age 30 and above declined rapidly in successive years. Similarly, the proportion of births of higher order also declined sharply during the same period. The trend provides indispensable clue that risky births, which often lead to infant mortality has been declining with declining numbers of births to mothers at younger or older age groups and births of higher order in subsequent years. Therefore, the acceleration of infant mortality during the1980s and 1990s are more likely to be the outcome of a reduction in the number of births among mothers of younger or older ages and higher order births. In the same milieu, the stagnation in IMR decline in later stages, particularly since 1995 is more likely the failure in the momentum of the decline in the proportion of high child mortality risk births of the total live births. Figure 5 and 6 shows that a considerable share of births in India continue to be high child mortality risk births.

Figure 4. Trends in Age-Specific Fertility Rates (ASFR) in India, 1971-2007

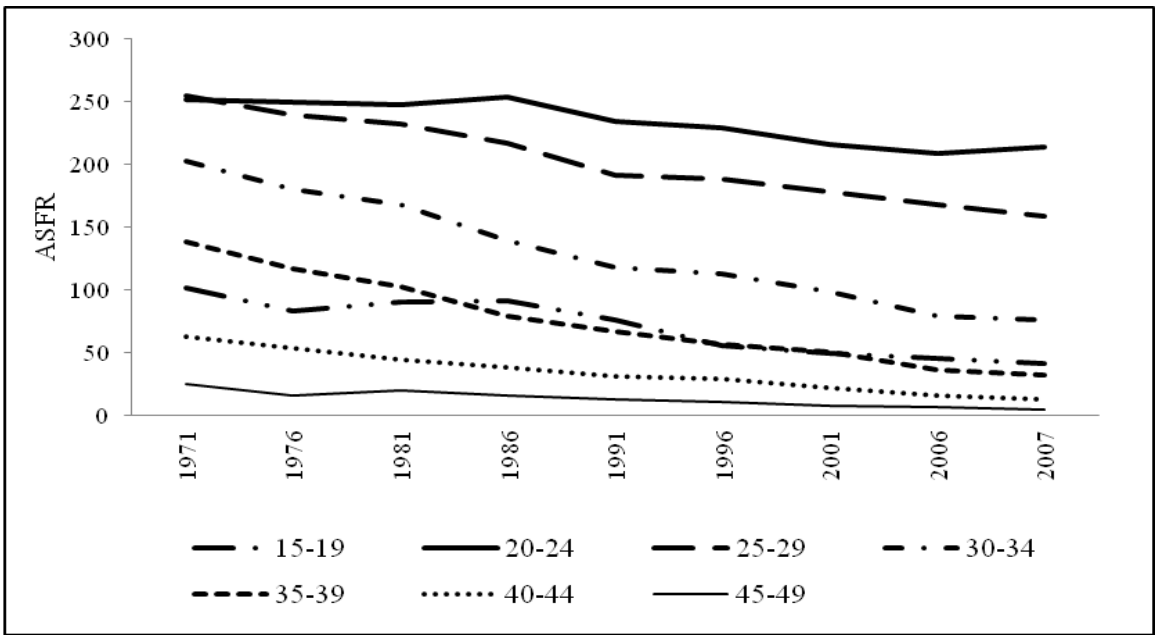
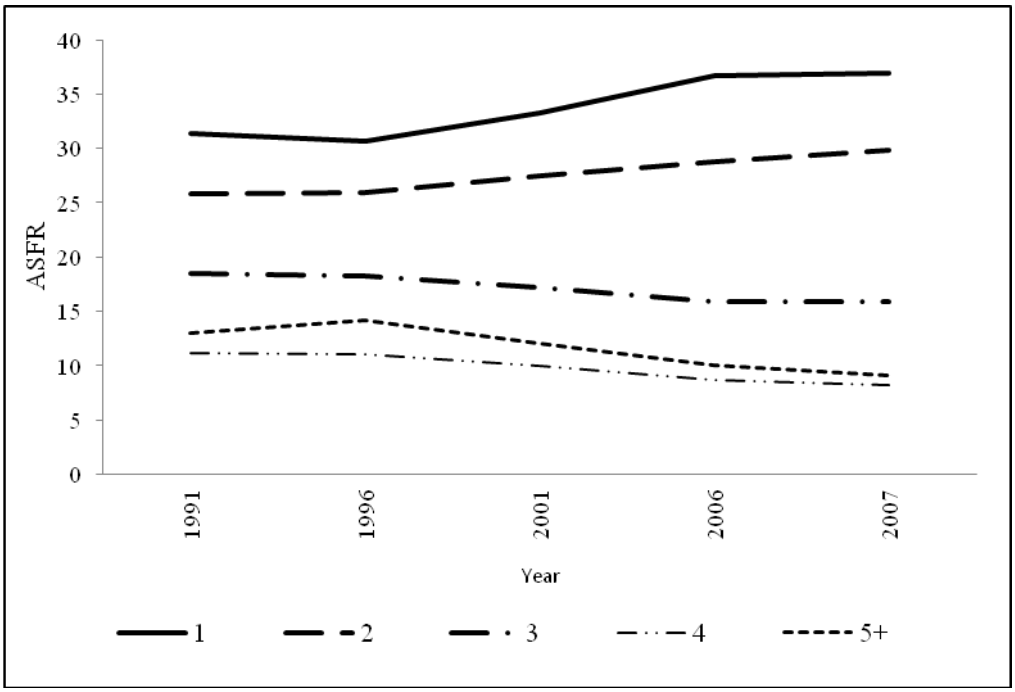
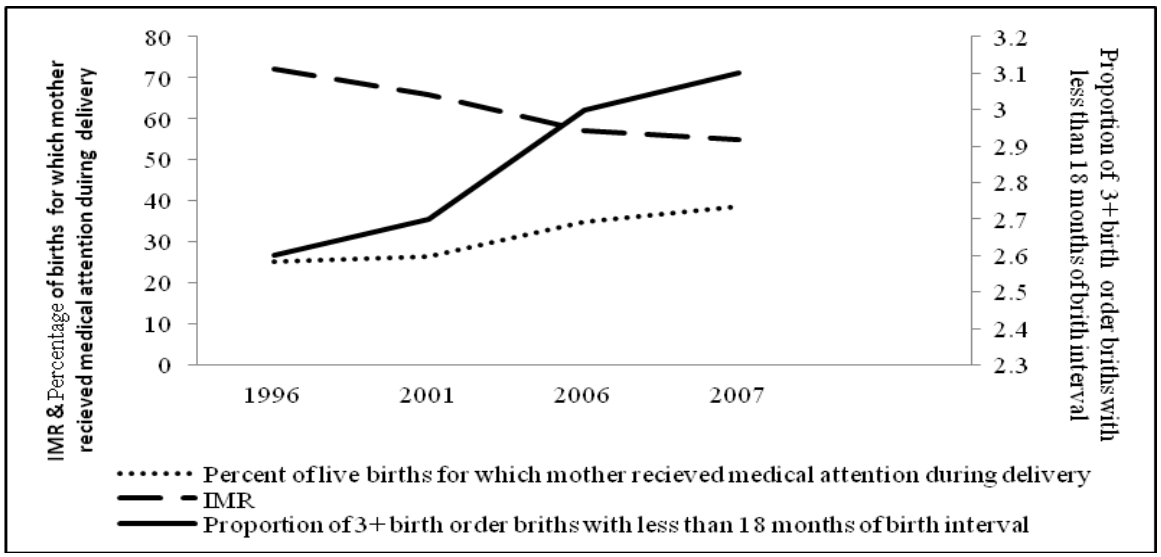


Figure 5. Percent Distribution of Live Births by Birth Order during 1991-2007



To understand the proportionate change in high child mortality risk births over the period, we assessed the trends in the percentage distribution of live births by birth order. In 1991, 13 percent of births were high child mortality risk births,[3] which fell to 8.4 percent in 1996. Corroboratively, during this period the trend indicates an accelerated decline in infant mortality even with just modest access to health care facilities to newborn children and their mothers (Office of Registrar General of India 2007). Contrastingly, however, since 1996, the proportion of high child mortality risk births, indicated a steady rise from 9 percent in 2001 to 11 percent in 2006 and 13 percent in 2007. This is most likely the reason why, during this period, in spite of the steady rise in health care facilities for newborn children and their mothers, infant mortality declined at a slower rate. These trends point to the fact that achieving sustained reductions in the share of higher order births is inevitable for curtailing childhood mortality rates during the mid to the last stages of demographic transition.

Figure 6. Trends in Percentage of Live Births by Type of Medical Attention (Institutional) Received by the Mother at Delivery, India and Bigger States, 1991-2007 at Interval of 5 Years



Causal Pathways of Fertility and Mortality Relationship: OLS Regression Analysis

First we examined linear relationship between fertility and key child mortality indicators. We estimated ordinary least squares (OLS) regression model to see the effect of the decline in TFR on different mortality indicators in India between 1971 and 2007. We regressed the lagged effect of fertility data on health and mortality data of the corresponding forward time points. The results of OLS regression of TFR on life expectancy at birth (LEB) showed a negative gradient of LEB on TFR. Similarly, TFR level indicated a positive gradient on disaggregated infant mortality indicators: infant mortality rate (IMR), the neonatal mortality rate (NNMR), postnatal mortality rate (PoNMR) and still birth rate (SBR); the decline in TFR showed a declining trend in all theses disaggregated child mortality indicators (figure 7).

[3] Present study, we are using concept 'high child mortality risk births' which refers to those births that were born at 3+ birth order with less than 24 months of birth intervals.

Figure 7. OLS regression scattered plots of the Relationship between Total Fertility Rate (TFR) and Mortality Indicators in India during 1971-2007

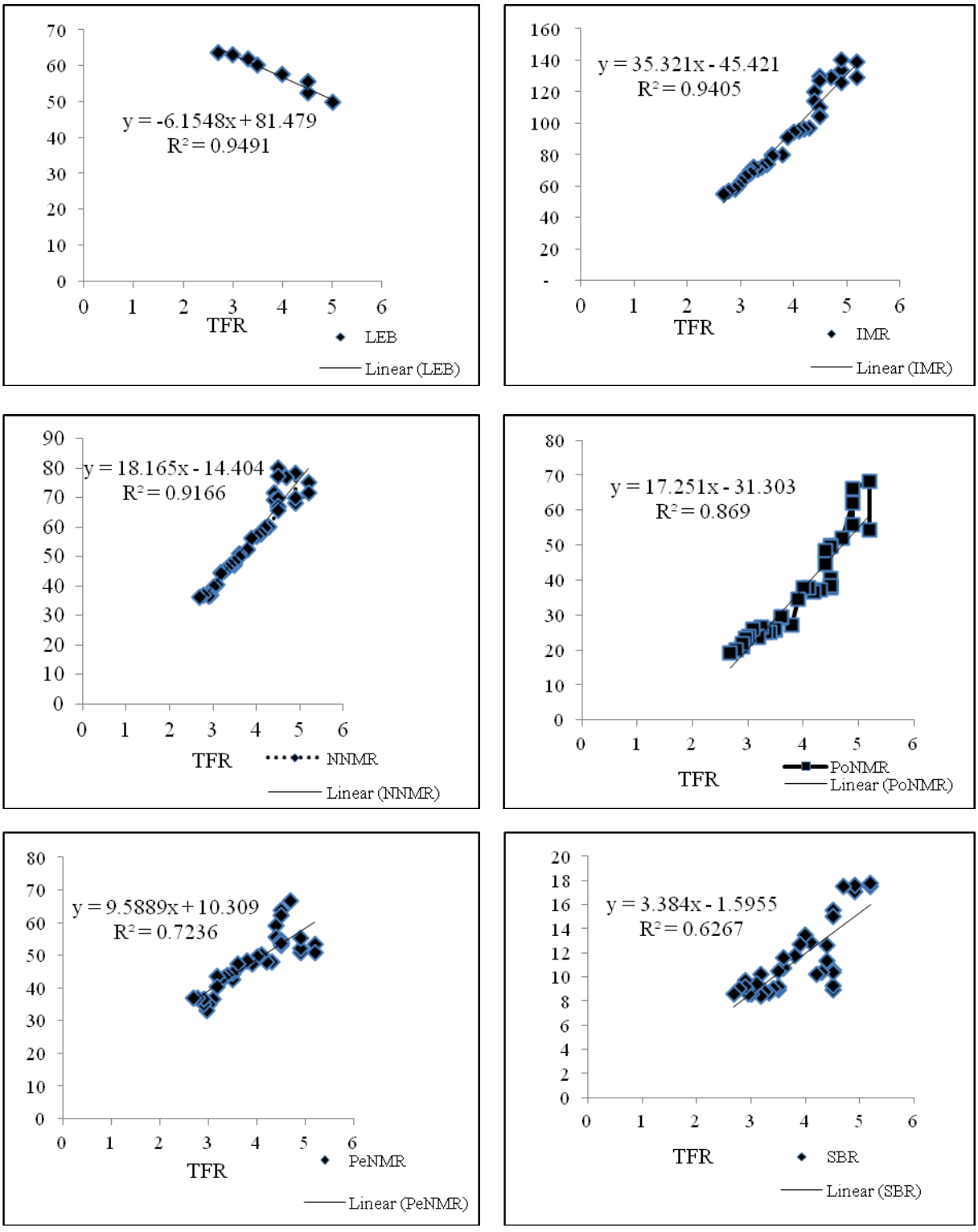


Table 1 compares the results of the regression models and the association between TFR and child mortality indicators. First, we observed that R^2 value is largest for TFR-LEB regression model and lowest for TFR-Still birth rate regression model. Second, it was more likely that the decline in TFR during 1971 to 2007 led to the largest decline in the IMR compared with other child mortality indicators. During this period, unit decline in TFR resulted in a decline of almost 35 infant deaths per thousand live births. The results suggest that the decline in TFR possibly had much greater impact on the decline of NNMR than of PoNMR. During this period, unit decline in TFR resulted in a decline of about 18 neonatal deaths and 17 postnoenatal deaths per thousand live births. The effect of the TFR decline on the decline of SBR was not much during the same period, as the unit decline in TFR resulted in a decline of just about 3 still births per thousand live births.

Table 1: OLS Time Series Regression Results of the Effect of Total Fertility Rate (TFR) on LEB and Disaggregated Child Mortality Indicators during 1971-2007

Y	X	Regression coefficient	Standard Error	p value	R^2	N (Time Points)
LEB	Constant	81.48	2.26	0.000	0.949	8
	TFR	-6.15	0.58	0.000		
IMR	Constant	-45.42	5.96	0.000	0.941	37
	TFR	35.32	1.50	0.000		
NNMR	Constant	-14.40	3.67	0.000	0.917	37
	TFR	18.16	0.93	0.000		
PoNMR	Constant	-31.30	4.49	0.000	0.869	37
	TFR	17.25	1.13	0.000		
PeNMR	Constant	10.31	3.97	0.014	0.724	37
	TFR	9.59	1.00	0.000		
SBR	Constant	-1.60	1.75	0.369	0.627	37
	TFR	3.38	0.44	0.000		

Note: 1. LEB-Life Expectancy at birth, IMR-infant mortality rate, NNMR-Neonatal mortality Rate, PoNMR-Postnatal Mortality Rate, PeNMR-Perinatal Mortality Rate, SBR-Still Birth Rate
2. Y is dependent and X is independent variables.

Panel Data Regression Analysis

Though the results from time-series data regression model point to TFR decline vis-à-vis high risk birth as the most plausible determinant of decline in infant mortality indicators and overall improvement in life expectancy, a two-way causal argument is plausible. Therefore, when assessing macro level data about the impact of fertility decline on health and development trends, the literature points to possible constraints of a perfect model that allows for simultaneous assessment of direct causation of socioeconomic determinants of fertility and reverse causational effect of fertility on health and development. Therefore, adopting an ideal method, we estimated panel data regression model, an econometric procedure, on pooled observations of state-wise cross-sectional data of India over three different time periods of 1981, 1991 and 2001 to examine the impact of fertility on infant mortality.

In the methodological literature, the Panel data regression model is argued to have the advantage of reduced levels of collinearity among the variables and more efficiency with degrees of freedom for measuring the effects that are easily not detectable in pure cross-section or time-series data (Sanderson 2001). Panel data sets are considered more dynamic as they encompass time-trend data over cross-sections. Time series variables depend on their time and estimating these underlying processes precedes estimation of the relationship of interest. The best predictor of y_{t+1} is its own past values that include the influence of all related variables in the true model.

Two methods of estimation are used in panel data regression model– fixed and random effects. In the fixed effects model, the effects of the predictor variables are estimated using ordinary least squares. The fixed effect model suffers from a greater loss of degrees of freedom and time invariant variables are not allowed whereas in the random effects model, the effects of the predictor variables are estimated using generalized least square method for which Hausman's test[4] is used to test the statistical significance. The random effects model is more helpful to capture the temporal effects of predictors including possible lagged effects during specific time periods. Both fixed and random effects models are estimated with IMR as a dependent variable.

We included six predictors: total fertility rate (TFR), female literacy rate, sex ratio, the percentage of males working in the non-agricultural sector, the percentage of urban population, and the period dummy variables for 1981-91 and 1991-2001 as predictors to estimate their effects on infant mortality rate (Table 2). In accordance with the reverse causation theoretical framework, TFR represents the principal predictor variable for each of the models predicting infant mortality. The other predictors represent socioeconomic conditions and gender norms. The period dummy variable is crucial to explain the temporal effects of the changes in the predictors during the period including possibly the lagged effects.

The estimates of fixed and random effects of predictors from panel regression models for infant mortality are presented in Table 2. The results of both fixed and random effects models together establish credible evidence of fertility impact on infant mortality. Controlling for other relevant socioeconomic covariates, the random effects model shows that fertility decline during the last three decades has had a notable infant mortality reducing effects. Aside from fertility impact, the negative effect of variables female literacy, per capita SDP and the percentage of males employed in the non-agricultural sector on infant mortality (fixed effects model) indicate that they continue to mediate as important socioeconomic macro determinants of child health improvement. The lack of significance of the effect of varying level of urbanization on infant mortality points to the possibility that trends in rural areas predominantly determine the net association between the dependent factor and the explanatory variables. The period dummy variables for 1981-91 and 1991-2001 reveal stable negative effects on infant mortality in both fixed and random effects models. In particular, the period effects for 1991-2001 suggest a modest reduction in infant mortality rate that might represent the temporal effects of the combined improvements in health care, the socioeconomic predictor variables, besides fertility decline.

[4] The Hausman test or Hausman specification test is a statistical test in econometrics named after Jerry A. Hausman. The test evaluates the significance of an estimator versus an alternative estimator. It helps one evaluate if a statistical model corresponds to the data; see also reference number (Bartels 2008).

Table 2: Results of Panel Data Regression Analysis with Lagged Effects of TFR (1971, 1981, 1991) on Infant Mortality Rate (2001)

Predictors	Fixed effects		Random effects	
	Model I	Model II	Model I	Model II
Total Fertility Rate (Time lagged)	0.4380**	0.4230*	0.3408**	0.3063**
Sex ratio of the population	-0.0001	-0.0031	-0.0013	-0.0018
Per capita State Domestic Product (SDP)	-0.1041	-0.4577*	-0.0663	0.2627
Female literacy rate	-0.0123*	-0.0267**	0.0027	0.0047
Female work participation rate	0.0184*	0.0201*	0.0162*	0.0211*
%of male in the nonagricultural sector	-0.0100	-0.0065	-0.0103*	-0.0131*
Percent of urban population	-0.0054	-0.0262	0.0054	-0.0083
Year 1981_91 dummy variable		-0.7196**		-0.4267*
Year 1991_01 dummy variable		-1.6015**		-0.7706*
Constant	3.322	1.9079	4.644*	3.156
R^2 (within)	0.86	0.90	0.83	0.88
Wald Chi^2			124.84**	143.53**
F-Value	19.53***	21.03***		
No. of observations	42	42	42	42

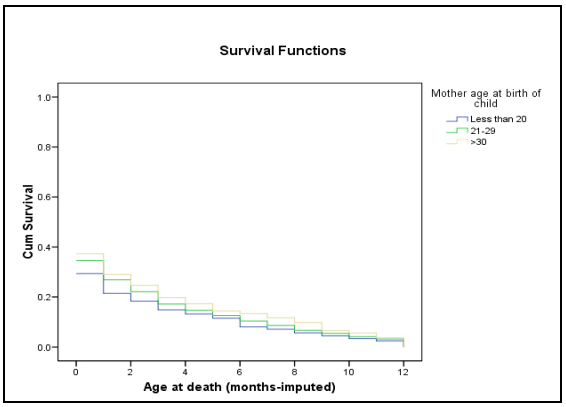
Level of Significance: *p<0.10; **p<0.05;***p<.001

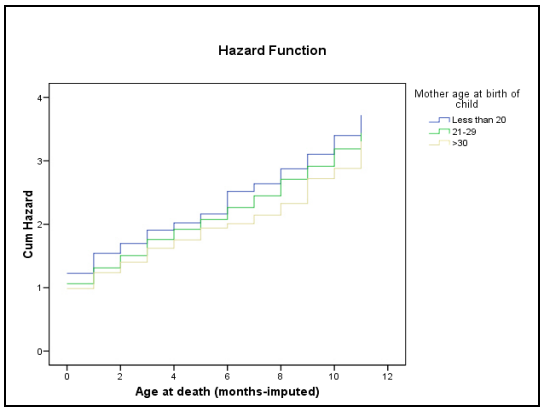
Kaplan Meier Estimates of Survival and Hazard Plots for Infant and Child

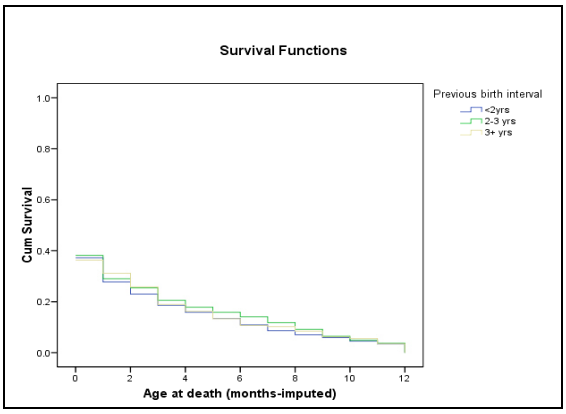
In this section, we examine micro data to understand the reverse causation effect of fertility on infant and childhood mortality. Adopting Kaplan Meier estimates of survival functions and hazard functions, we generated the survival times and risk of child death at time *t* of the data that may be incomplete. An important advantage of the Kaplan–Meier estimates of survival and hazard plots is that the method can effectively deal with censored data. The goal was to plot and estimate infant and child survival and hazard function curves by their key predictors. However, for effective comparison of survival curves of two or more groups of independent variables, we used log rank test (Altman, 1991). This test is often used because it does not assume any particular distribution of the survivor function but provides with confirmation of statistical evidence for two or more survival curves being different.

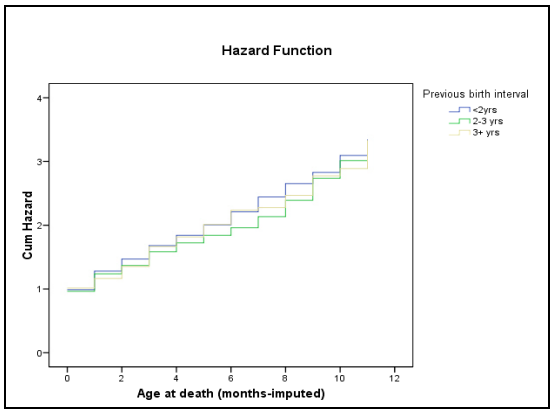
The Kaplan plots for birth survival function (probability that the time of child death is later than some specified time *t*) and hazard function (risk of dying at time t) have been estimated on the birth history data of National Family Health Survey (2005-06). The Kaplan plots indicate steady decline in survival times with time and the probability of child deaths was much more pronounced during the infant stages than later childhood stages. Results indicate considerable differences in survival and hazard rates by key fertility indicators (Figure 8,9,10 and 11). The results of the log rank test reveal that the effect of mother's high risk fertility characteristics: age of the mother at child birth and parity at sterilization on mortality indicators is much more pronounced than the other covariate like previous birth interval. The mortality reducing effects of fertility related determinants (Age at child birth, parity, birth order) are more prominent for an infant than child mortality.

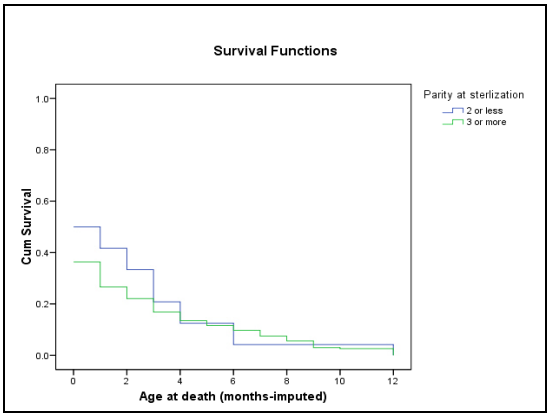
Figure 8. Plots of Kaplan-Meier Estimates of Survival and Hazard Functions for Neonates in India, 2005-06

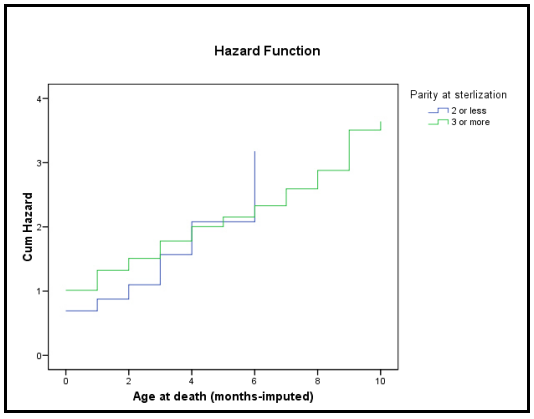






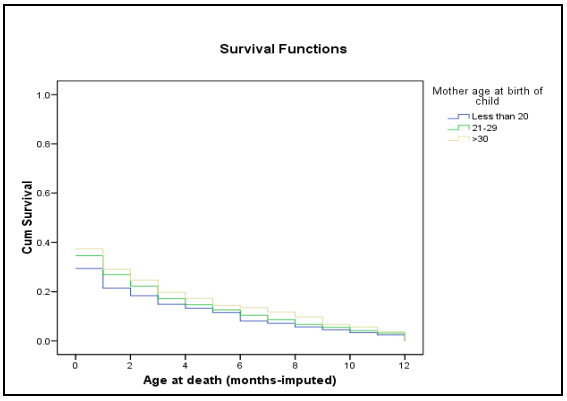


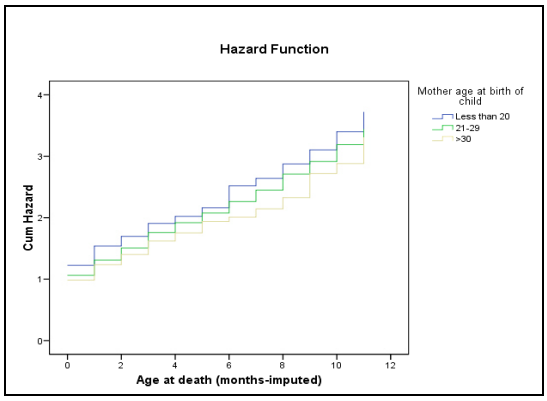


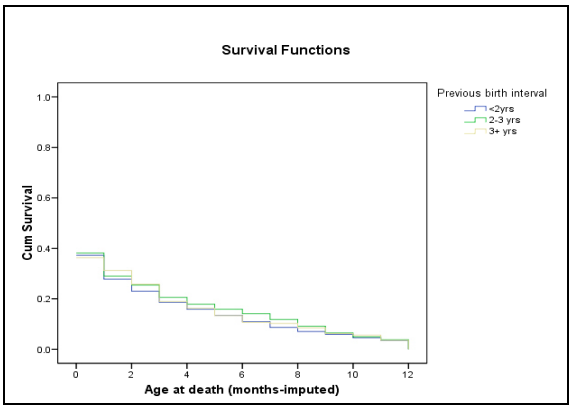


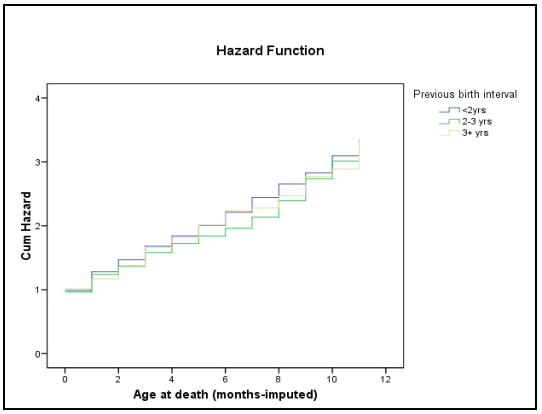
Independent and dependent variables	Log Rank (Mantel-Cox)	
	Chi-Square	Sig.
Neonatal mortality by age of the mother at birth	5.61	0.06
Neonatal mortality by Birth Interval in successive births	0.68	0.71
Neonatal mortality by parity at sterilization	0.94	0.33

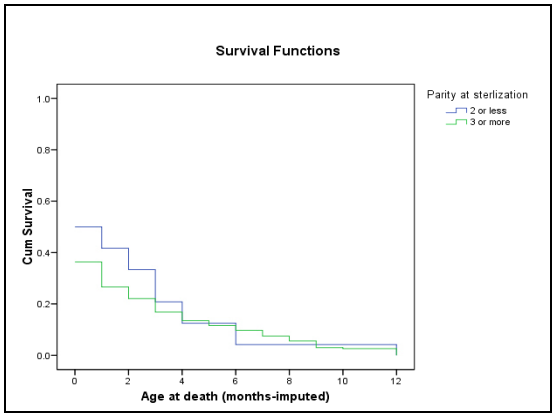
Figure 9. Plots of Kaplan-Meier Estimates of Survival and Hazard Functions for Infants in India, 2005-06

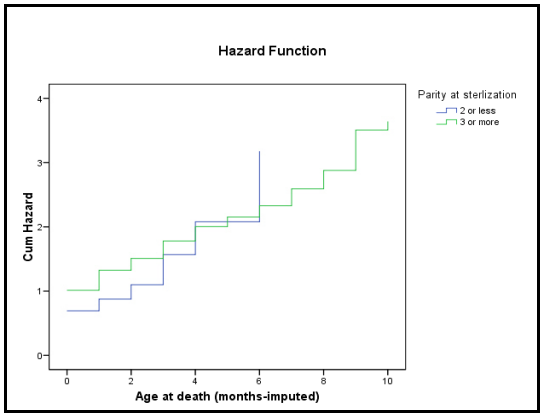






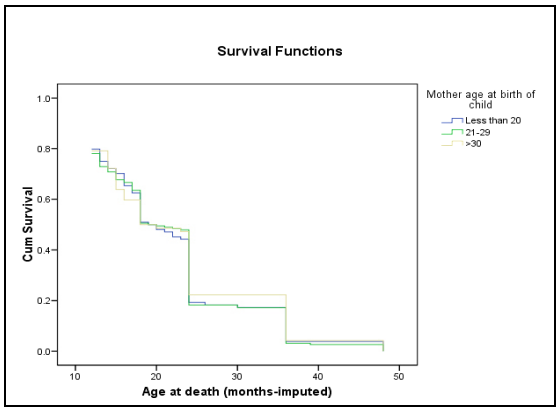


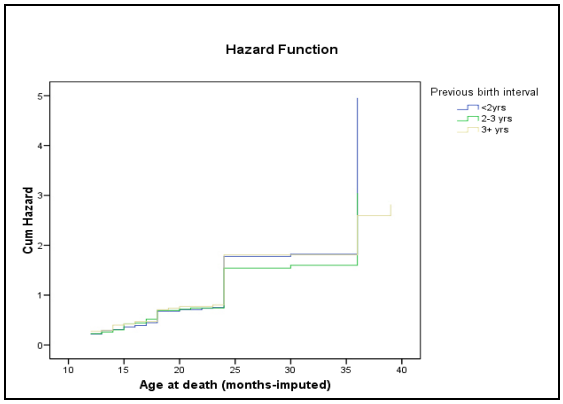


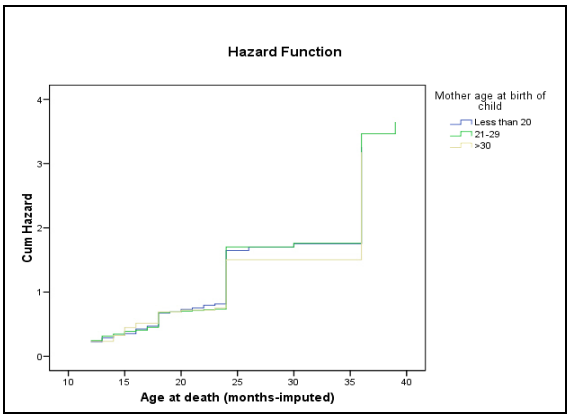


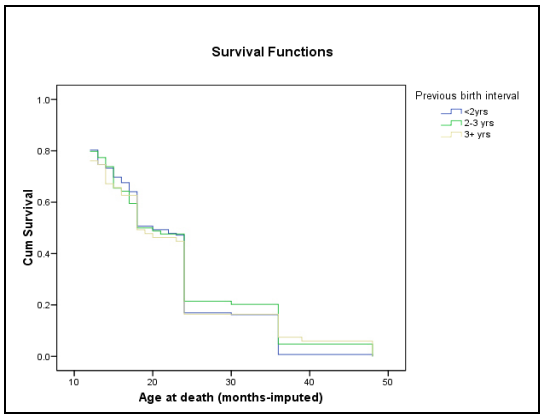
Independent and dependent variables	Log Rank (Mantel-Cox)	
	Chi-Square	Sig.
Infant mortality by age of the mother at birth	8.10	0.02
Infant mortality by Birth Interval in successive births	0.90	0.64
Infant mortality by parity at sterilization	0.32	0.57

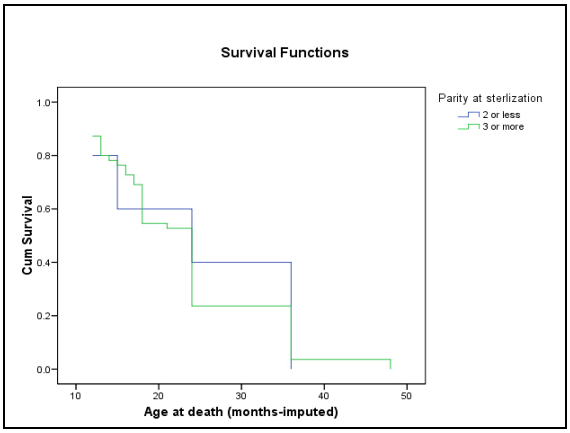
Figure 10. Plots of Kaplan-Meier Estimates of Survival and Hazard Functions for Children (1-5) in India, 2005-06

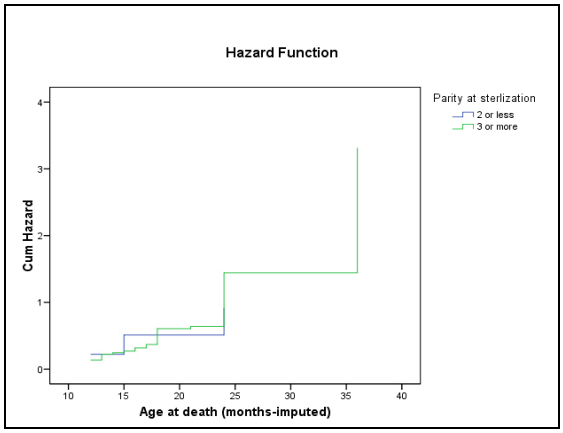






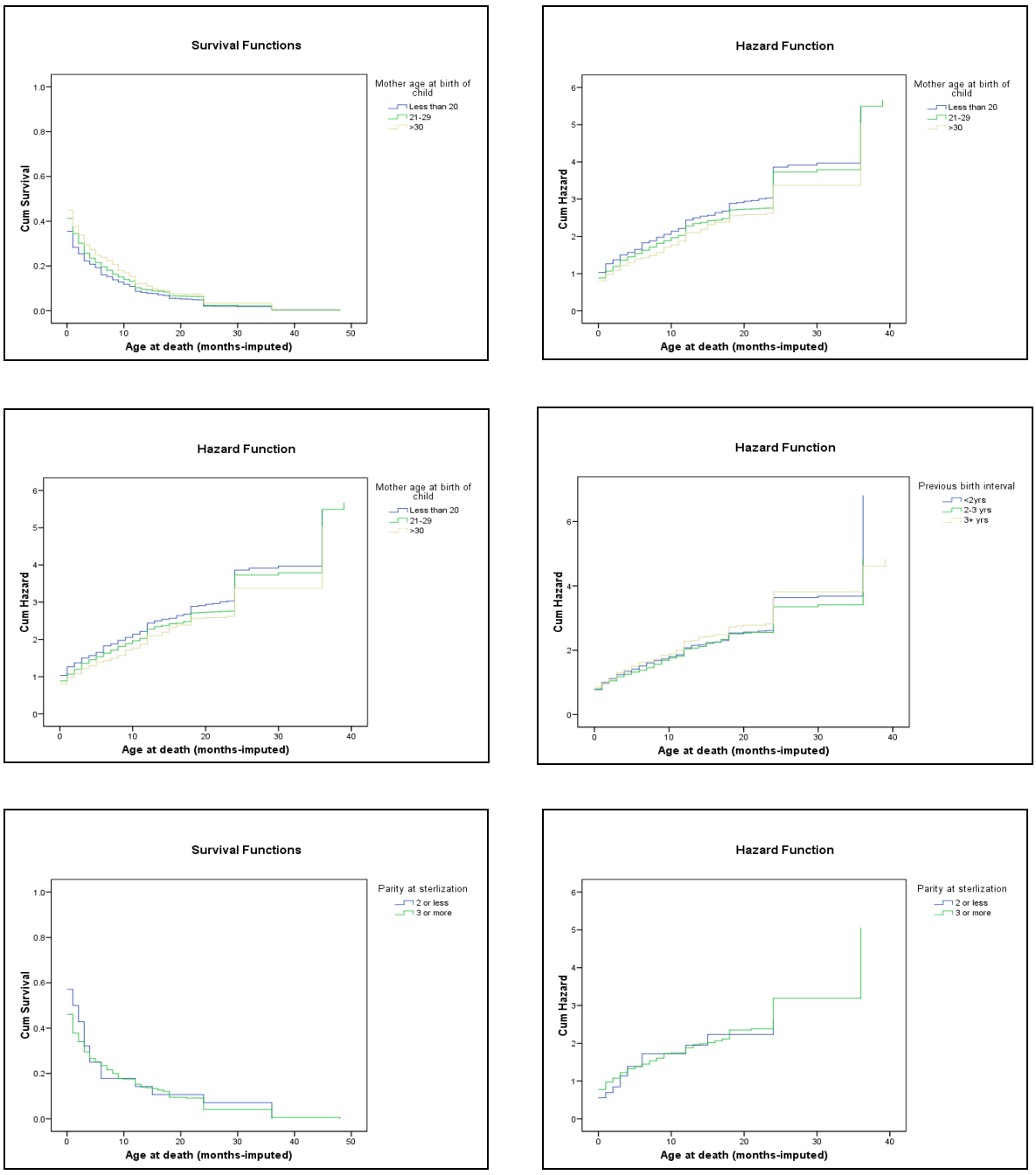






Independent and dependent variables	Log Rank (Mantel-Cox)	
	Chi-Square	Sig.
Child mortality by age of the mother at birth	0.24	0.89
Child mortality by Birth Interval in successive births	0.43	0.81
Child mortality by parity at sterilization	0.09	0.76

Figure 11. Plots of Kaplan-Meier Estimates of Survival and Hazard Functions for under Five Children in India, 2005-06



Independent and dependent variables	Log Rank (Mantel-Cox)	
	Chi-Square	Sig.
Under five mortality by age of the mother at birth	10.45	0.01
Under five mortality by Birth Interval in successive births	1.87	0.39
Under five mortality by parity at sterilization	0.15	0.7

Kaplan-Meier Mean Survival Time Estimates

Mean survival time is estimated as the area under the survival curve based on the entire range of data. Mean survival estimates are used for comparative assessment of the average time that the sample of children covered in the study remained alive by key fertility related characteristics of mothers. The results presented in table 3 display considerable differences in mean survival times among children born to women of either lower or higher age groups and high parities and, children of low birth order and short birth interval with their respective counter groups.

Also mean survival time increased with increase in age of mother at birth for neonatal, infant, childhood and under-five children. The mean survival time was higher for the births of higher order and longer birth interval than for births of lower order and shorter birth interval. By women's parity at sterilization, expected mean survival time for children under five is higher for lower parity births than higher parity births except for neonatal mortality. Overall, women's parity at sterilization emerges as a key predictor of infant and child mortality signifying infant and child mortality reducing effects of high parities.

Modelling Child Survival by Mother's Fertility Status: Cox Regression Model

The log rank test is not necessarily adequate to explore the adjusted effects of several independent variables which are likely to affect child survival. Adjustment for other socioeconomic predictors of infant and child survival may improve the precision with which the effects fertility characteristics are estimated. To determine the effect of mother's fertility related characteristics of infant and child mortality, we used the Cox proportional hazard model to estimate the hazard ratios (relative risk) of infant and child deaths by mother's fertility characteristics controlling for other socioeconomic covariates.

Estimated relative risks of neonatal, infant, child and under five mortality are substantially lower among mothers of medium ages than mothers of younger and older ages at birth of child. In first birth order, the relative risks of child mortality are lower for female than male neonatal, infant and under-five children. The relative risk of child mortality is lower among mothers with birthorder2-3 and longer birth interval between successive births compared with children of higher birth order and shorter birth interval. Births following a longer birth interval indicated a significant reduction in relative risk of infant and child mortality. Mothers of lower parities (2 and less) are strongly associated with lesser relative risks of infant and child mortality that fertility. On the other hand, the results indicate that the relative risks of neonatal, infant, child and under-five mortality is much greater for births among younger age women and with very short successive birth intervals and women of higher parities compared to their counter groups (table 4).

Table 3: Mean Survival Time by High Risk Related Predictors, India, 2005-06

Predictors		Mean survival time in months											
		Neonatal	95% CI		Infant	95% CI		Children (aged 0-4)	95% CI		Under five Children	95% CI	
Age of mother at birth of child	<20	0.13	0.13	0.14	1.21	1.10	1.31	12.15	11.39	12.91	4.69	0.19	4.32
	21-29	0.14	0.13	0.16	1.48	1.38	1.58	12.37	11.78	12.96	5.62	0.17	5.29
	>30	0.15	0.13	0.16	1.67	1.49	1.86	13.54	12.56	14.51	6.82	0.32	6.20
Composite variable of birth order, birth interval and sex of the child	First BO and male	0.13	0.12	0.14	1.01	0.87	1.15	10.42	9.29	11.55	3.34	2.89	3.80
	First BO and female	0.13	0.12	0.15	1.06	0.89	1.22	11.69	10.32	13.06	4.05	3.45	4.64
	BO 2-3 BI<23 months and male	0.13	0.11	0.15	1.37	1.16	1.59	11.21	9.84	12.57	4.46	3.78	5.13
	BO 2-3 BI<23 months and female	0.18	0.13	0.24	1.52	1.29	1.75	12.10	10.83	13.37	5.89	5.13	6.66
	BO 2-3 BI>24 months and male	0.12	0.10	0.13	1.39	1.18	1.60	12.34	10.93	13.75	5.35	4.59	6.10
	BO 2-3 BI>24 months and female	0.16	0.14	0.19	1.91	1.61	2.20	14.41	12.99	15.84	7.64	6.69	8.59
	BO 3+ BI<23 months and male	0.14	0.12	0.16	1.44	1.22	1.66	12.25	10.91	13.60	5.66	4.88	6.43
	BO 3+ BI<23 months and female	0.16	0.13	0.19	1.84	1.58	2.11	13.81	12.61	15.01	7.65	6.81	8.49
	BO 3+ BI>24 months and male	0.15	0.12	0.17	1.46	1.21	1.71	13.08	11.52	14.63	6.07	5.17	6.96
	BO 3+ BI>24 months and female	0.15	0.13	0.18	2.01	1.71	2.31	14.13	12.79	15.46	8.11	7.16	9.06
Women's parity at sterilization	2 or less than 2	0.13	0.07	0.18	2.64	1.71	3.56	15.70	11.12	20.29	9.60	6.29	12.91
	3 or more than 3	0.14	0.13	0.16	1.36	1.21	1.50	13.23	12.28	14.18	5.86	5.32	6.39

Table 4. Adjusted Relative Risks of Neonatal, Infant, Child and Under-Five Mortality for Sterilized Women by High Risk Related Predictors, India 2005-06

Predictors		Neonatal	Infant	Child	Under five
Age of mother at first birth	<20	1	1	1	1
	21-29	0.77***	0.85**	0.81	0.85**
	>30	0.98	1.09	1.16	1.13
Composite variable of Birth order, Birth interval and sex of the child	First BO and male	1	1	1	1
	First BO and female	0.70**	0.74**	1.19*	0.84
	BO 2-3 BI<23 months and male	0.95	1.07	2.20*	1.11
	BO 2-3 BI<23 months and female	1.09	1.31**	5.17	1.53***
	BO 2-3 BI>24 months and male	0.44***	0.48***	1.65	0.57***
	BO 2-3 BI>24 months and female	0.39***	0.50***	0.66**	0.63***
	BO 3+ BI<23 months and male	1.56**	1.62***	4.21***	1.78***
	BO 3+ BI<23 months and female	1.38*	1.50***	1.11***	1.91***
	BO 3+ BI>24 months and male	0.69**	0.69**	2.03	0.79*
	BO 3+ BI>24 months and female	0.77	0.82	1.63***	0.98
Parity at sterilization	2 or less than 2	1	1	1	1
	3 or more than 3	9.35***	6.94***	2.25**	5.81***
-2 Log Likelihood		14892.7	21930.0	5363.2	27384.1
Chi-square statistic		506.0***	825.7***	401.7***	1104.9***

Level significance: *p<0.10; **p<0.05;***p<.001

Note: The control variables here including Place of residence, mother' education, caste of the women, wealth quintile of the household, and religion of women, ANC visits, and Medical assistance received.

CONCLUSION

With India's fertility rates continuing to fall far more quickly than socioeconomic structural conditions and infant mortality rates, we sought to explore the reverse causation hypothesis of 'effect of fertility decline on infant mortality decline' in India and its states. Our two stage macro and micro data analysis demonstrated complementing patterns of reverse causation effect of fertility decline on the infant mortality decline.

The first stage trend analysis of macro level data on fertility and infant mortality showed that fertility decline in the 1970s via proactive family planning was more likely to have negatively impacted infant mortality rates in the successive lag periods more than the impact of infant mortality on fertility decline. Trends in fertility and infant mortality indicators if looked at in conjunction with evidence of widespread fertility decline among large sections of the poor and uneducated population suggest that fertility decline most likely proceeded faster than mortality decline. Further, the OLS regression analysis of time trend data with reasonably good fit (R^2) showed visible effects of fertility decline on the infant mortality decline. Additional corroborative results from panel data regression model estimates of time-trend and cross-sectional data over three decades by states showed a net negative effect of fertility (TFR) decline as a credible determinant of infant mortality decline. Even in an

alternative likelihood of both fertility and infant mortality rates following that a somewhat parallel course, the more logical assumption of causal connection would be that both fertility and infant mortality may have a bi-directional impact. Therefore, collectively, these results provide distinctive indications that India's family planning policy action to reduce the fertility rate has considerably impinged on infant mortality rate during the last more than three decades and in the same way one would infer that the infant mortality rate has impinged on the fertility rate.

The second stage micro level analysis of NFHS-3 birth history data sheds more useful insights of mother's low fertility characteristics resulting in an effective reduction in infant mortality rates. Both Kaplan-Meier plots and Cox proportional hazard estimates of fertility related predictors on infant mortality reveal that higher parities, high risk births at both ends of the childbearing span and shorter birth interval continue to be closely associated with a high relative risk of infant mortality; vice-versa, a reduced share of such high risk births is seen closely tied with lower infant mortality. Particularly, the recent stagnations in India's infant mortality decline were more likely to be the result of continuance of early childbearing practices, lack of progress in lengthening birth intervals and stagnation in high parity births. Fertility decline in India across both high and low fertility states continues to be driven by the widespread use of terminal methods of contraception through which high risk births still remain a significant barrier to infant mortality reduction. While family building strategies have been changing steadily, increase in age at marriage and birth spacing, and reduction in higher order births, are necessary thrust areas of policy to contain the stability in infant and child mortality rates in India.

In Indian context, the results of the reverse causal relationship between fertility and mortality represent a contra force of previous theoretical assumption of change (infant mortality decline) and response (fertility decline). The issues surrounding this outcome do not merely support commensurate positive effects of fertility decline on the infant mortality decline but imply broad-based repercussions of fertility decline on potential health and development benefits in India. The findings thus have a strong bearing on previous studies that have tried to measure net positive health benefits to children and women, poverty reduction and progress in development indicators including education of children, women's employment etc. (Hobcarft, 1985; Woods et al., 1988, LeGrand and Philips, 1996; Sandersen, 2001; Pyne, 2004). From a policy point of view, in India and elsewhere in developing countries, where expenditure on health is proportionally and significantly low compared with developed countries, and there is an absence of an effective health care mechanism, controlling fertility is a parsimonious step for curtailing infant mortality, improving women and child health and uplifting overall living conditions.

REFERENCES

Altman D. G., 1991, "Practical statistics for medical research", London: Chapman & Hall/CRC: pp. 365-396.

Arokiasamy P., 2009, "Fertility Decline in India: Contributions by Uneducated Women Using Contraception", Economic and Political Weekily, no. 44 (30).

Arokiasamy P., 2007, "Sex Ratio at Birth and Excess Female Child Mortality in India: Trends Differentials and Regional Patterns" in I Attané and C Z Guilmoto (ed.), Watering the Neighbour's Garden: The Growing Demographic Female Defi cit in Asia, CICNRD, Singapore.

Baltagi H, B., 2005, "Econometric analysis of panel data" Third edition, Chichester: John Wiley & Sons Limited.

Bhat M P. N., 2002, "Returning a favour: reciprocity between female education and fertility", *World Development,* no. 30(10) pp. 1791-1803.

Bloom D., Jeffrey, G. W., 1997, "Demographic Transitions, Human Resource Development and Economic Miracles in Emerging Asia", in J. Sachs and D. Bloom (eds), *Emerging Asia. Manila:* Asian Development Bank.

Bongaarts J, 1987, "Does family planning reduce infant mortality rates?", *Population and Development Review* no. 13(2), pp. 323-334.

Bongaarts J, Potter R. G., 1983, "Fertility, biology and behaviour: An analysis of the proximate determinant". New York, Academic Press.

Bingley, P. J., Douai, I. F., Rogers, C. A., Gale, E. A. M., 2000, "Influence of maternal age at delivery and birth order on risk of type 1 diabetes in childhood: prospective population based family study", *BMJ* no. 321, pp. 420-4.

Cleland J., Sathar Z. A., 1984, "The effect of birth spacing on childhood mortality in Pakistan", *Population Studies,* no. 38, pp. 401-418.

CMIE, 1971-2001. "Economic data base, Centre for Monitoring Indian Economy Pvt. Ltd", Mumbai, India.

Coale A. J., Hoover E., 1958, "Population growth and economic development in low-income countries: A case study of India", Princeton, Princeton University Press.

Cox D R., Oakes D., 1984, "Analysis of survival data", London, Chapman and Hall.

Curtis S. L., Diamond I., Mcdonald J. W., 1993, "Birth interval and family effects on post neonatal mortality in Brazil", *Demography*, no. 33, pp. 33-43.

Davis K., 1945, "The world demographic transition", *Annals of the American Academy of Political and Social Science*, no. 237, pp. 1-11.

Davis K., 1963, "The theory of change and response in modern demographic history", *Population Index,* no.29 pp. 345-66.

HONGLADAROM C, PRASITH-RATHSINT S., ROBINSON W. C., 1987, "The economic and social impact of declining fertility: a case study of Thailand", *Asia-Pacific Population Journal,* no.1, pp.3-22.

Hobcraft J., 1987, "Does family planning save lives? Technical background papers prepared for the International Conference on better health for women and children through family planning", Nairobi, 5-9 Oct.

IIPS, Macro International, 1992-2006, "National Family Health Survey (1-3) data", Ministry of Health and Family Welfare, Government of India.

James K.S., 1999, "Fertility decline in Andhra Pradesh: an alternative hypothesis", *Economic and Political Weekly,* no. 34, pp.491-499.

Legrand T. K., Phillips J. F., 1996, "The effect of fertility reduction on infant and child mortality: evidences from Matlab in rural Bangladesh", *Population Studies*, no. 50, pp. 51-68.

Mcnay K., Arokiasamy P., Carson R., 2003, "Why are uneducated women in India using contraception? A multilevel analysis", *Population Studies*, no.57, pp. 21-40.

Office of Registrar General Of India, 1971-2007, "Sample Registration System Year Books", Government of India, New Delhi.

Payne J, 2004, "The impact of reduced fertility rate on women's health", *BMC Women's Health* 4(Suppl): S11.

Potter J. E., 1988, "Does family planning reduce infant mortality? An exchange", *Population and Development* no.14, pp. 179-187.

Preston S. H., 1985, "Mortality in childhood: Lessons from WFS", in CLELAND J. G., Hobcraft J., (Eds), *Reproductive change in developing countries.* Oxford, Oxford University Press.

Pritchett L., 1994, "Desired fertility and the impact of population policies", *Population and Development Review* no. 20, pp. 1-55.

Rutstein S. O., 2005, "Effects of preceding birth intervals on neonatal, infant and under-five years' mortality and nutritional status in developing countries: evidence from the demographic and health surveys", *International Journal of Gynecology & Obstetrics* no.89, pp. S7-S24.

Sanderson S. K., 2001, "An evolutionary interpretation of fertility decline: new evidences. Population and Environment", *A Journal of Interdisciplinary Studies*, no. 22, pp. 555-563.

Taylor, C. E., Newman J. S., Kelly N. U., 1976, "The child survival hypothesis", Population 30 (2), 263-278.

Temkin-Greener H., Swedlund, A. C., 1983, "A test of the child replacement hypothesis: nineteenth century Massachusetts", *Biogeography,* no.30, pp. 218-227.

Thorne, M. C., Green L. W., 1976, "The contribution of family planning programs to health: from correlation to causal inference", *Paper presented at the annual meeting of the Population Association of America, Montréal,* 29 April-1 May.

Russell E. J., 1988, "Does family planning reduce infant mortality? An exchange", *Population and Development Review,* no. 14, pp.171-178.

Woods, R. I., Watterson P. A., Woodward J. H., 1998, "The cause of rapid infant mortality decline in England and Wales, 1861-1921 part I", *Population Studies,* no. 42, pp. 343-366.