

## Infant and Child Mortality in Andhra Pradesh: Analysing changes over time and between states

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Analysing changes over time and between states

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## **Abstract**

Most countries of the world are reducing infant and child mortality too slowly to meet the Millennium Development Goal of a two-thirds reduction by 2015. Yet, some countries and regions have achieved impressive reductions, Kerala in India being one example. This paper examines the determinants of infant and child mortality in Andhra Pradesh, where the Young Lives project is taking place, and Kerala and the factors explaining their differential performance. The determinants of mortality are estimated using a Cox proportional hazards model. Infant mortality is found to depend on biological factors, including mother's age and birth order, and also factors related to health service provision such as tetanus injection and use of antenatal services. Economic well being is not significant once these other factors are taken into account. By contrast, economic well-being is a significant determinant of child mortality, but substantially outweighed in importance by other factors such as maternal education and knowledge of health practices (ORS) and access to safe water. The data also show gender discrimination in Andhra Pradesh, notably toward girls with only female siblings, which is absent from Kerala. We conclude that raising service levels across India toward the levels found in Kerala is a necessary step toward meeting the MDGs, and that the success of these efforts is reinforced by female empowerment.

## I. Introduction

Every year about 11 million children die, of which 10 million are in the developing world and nearly 2 million in India alone<sup>1</sup> (UNICEF, 1999). Table 1 shows the infant mortality rates for three broad economic regions of the world and India. India's experience is fairly typical of low-income countries. There has been a steady reduction in mortality resulting in a halving of the rate in just over thirty years. So, whilst there has been a reduction, the level of mortality remains high and the rate of reduction is insufficient to achieve the Millennium Development Goal of reducing infant and under five mortality in the 25 years between 1990 and 2015.

	High incor	me countries	Middle income countries	Low income countries °	India
960	_	35	120	167	165
970		26	94	140	139
980		13	69	117	119
990		8	46	97	86
995		7	39	89	69
999		6	31	77	71
annual rate o	f				
hange over the	period <sup>2</sup> -	-3.7	-2.8	-1.6	-1.7

However, there are considerable variations in mortality within countries. Table 2 shows state-level infant and children mortality rates for 1992-93 and 1998-1999 obtained from Demographic Health Survey (DHS) data for all Indian states, and the percentage changes in the mortality rates between the two surveys.<sup>3</sup> Andhra Pradesh (AP) is a 'typical' Indian state in that its mortality rates are very similar to the Indian average. In contrast, Kerala has the lowest rates of both infant and child mortality. This paper examines two issues: what factors explain the decline in mortality in AP, and what accounts for the substantial differences in mortality between AP and Kerala?

Part 2 outlines data sources and method. Existing studies are reviewed in Part 3, identifying the variables to be included in our model. Results for AP are presented in Part 4, Part 5 considers evidence of gender bias, Part 6 explores the differences in mortality between AP and Kerala and Part 7 concludes.

Number of dead children refers to year 1996.

<sup>2</sup> The percentage annual rate of change over the period is the discrete rate of change derived from the trend coefficients of regressions of the log of mortality on time.

<sup>3</sup> Mortality rates in Table 2 are taken from IIPS (1995, 2000) and were calculated by applying the synthetic cohort probability method (see Appendix).

## 2. Data and methods

The data analysed in this paper are from the Indian National Family Health Surveys (NFHS) of 1992-93 and 1998-99. The Indian NFHS are a variant of the Demographic Health Surveys (DHS) carried out in many countries with the financial support of the US Agency for International Development (USAID). These surveys provide data for all 25 Indian States on fertility, family planning, maternal and child health, nutrition and infant and child mortality.

	1992-93	Infants 1998-99	% change	1992-93	Children 1998-99	% change
All India	79	68	-14	34	29	-13
North						
Delhi	66	47	-29	19	9	-53
Haryana	73	57	-23	27	21	-23
Himachal Pradesh	56	34	-38	14	8	-42
Jammu & Kashmir	46	65	43	14	16	12
Punjab	54	57	6	15	16	5
Rajastan	73	80	II	32	38	16
Central						
Madhya Pradesh	85	86	1	49	56	14
Uttar Pradesh	100	87	-13	46	39	-15
East						
Bihar	89	73	-18	42	35	-18
Orissa	112	81	-28	21	26	20
West Bengal	75	49	-35	26	20	-24
Northeast						
Arunachal Pradesh	40	63	57	33	37	12
Assam	89	70	-22	59	21	-64
Manipur	43	37	-13	20	20	-2
Meghalaya	64	89	39	24	36	49
Mizoram	15	37	152	15	18	23
Nagaland	17	42	143	4	23	531
Sikkim	76	44	-42	31	28	-9
West						
Goa	32	37	15	7	11	46
Gujarat	69	63	-9	38	24	-37
Maharashtra	51	44	-14	21	15	-28
South						
Andhra Pradesh	71	66	-7	22	21	-6
Karnataka	66	52	-21	24	19	-18
Kerala	24	16	-32	8	3	-69
Tamil Nadu	68	48	-29	20	16	-21

We analyse the mortality of three distinct age groups: infants (from 0 to 11 months), children (from 1 to five years) and 'children from five to ten' (between five and ten years old); see Table 3 for a summary.<sup>4</sup> Accordingly, the mortality rate is the number of children dying in an age range per 1,000 children who enter that age range.

TABLE 3. CHI	LDREN AGE GROUPS AN	ND MORTALITY RATES
Age group	Life span	Mortality rate
Infants	From birth to 11th month	Infant mortality rate (IMR)
Children	From 12th to 59th month	Child mortality rate (CMR)
Children from five to ten	From 60th to 119th month	Mortality rate of children from five to te

This paper examines the determinants of mortality. Using data on changes in those determinants, we examine the factors behind falling infant and child mortality in Andhra Pradesh and the mortality differential between AP and Kerala. Model estimation is carried out using a hazard model. This model is preferred to a standard regression model on account of its treatment of censored observations. When child mortality is analysed using a logistic regression model, as is common in the literature, only those children who were born five years before the survey can be included in the study. Children born more recently have not been fully exposed to the risk of dying and mortality measurements would be underestimated if they were included in the study. However, discarding these observations has two major disadvantages. The first is the loss of information from dropping observations. The second is an increase in measurement bias. Mortality determinants, like poverty or access to safe water, are measured at the time of the survey, but they are used as if they applied to the whole life of the child. This bias is reduced if the vital data refer to a time closer to the collection of the data.

The hazard model used is the Cox proportional hazard model (Cox and Oakes 1984, and Collet 1994). In order to simplify the exposition, suppose there are two groups of children: the mothers of the children of the first group (y) have been immunised against tetanus during pregnancy while mothers of the children in the other group (n) have not. The Cox model assumes that the hazard of death at time t for the children with non-immunised mothers is proportional to the hazard of the children with immunised mothers by the same factor  $\psi$  at every time t:

The categories of infant and child mortality are standard. The two are combined to make under-five mortality, which we do not use here because mortality determinants vary by age group. Deaths of children aged 5 to 10 are less commonly analysed, but included here as the Young Lives project is concerned with child welfare until adulthood.

$$h_n(t) = \psi h_y(t) \tag{1}$$

where  $h_n$  and  $h_y$  are the hazards (probabilities of dying) for the two groups and  $\psi$  is the hazard ratio. If  $\psi > 1$ , the hazard of death is larger for children whose mothers are not immunised relative to those whose mothers have been immunised, so that maternal tetanus immunisation reduces infant mortality. If  $\psi < 1$  or  $\psi = 1$ , the death hazard of the children with immunised mothers would be higher or equal to the hazard of those whose mothers are not immunised, in which case tetanus immunisation would be of no use  $(\psi = 1)$ , or even harmful  $(\psi < 1)$ .

The model can be extended and generalised to more than one explanatory variable. It is practically useful to model the hazard ratio in logarithmic form as a function of a set of x variables, whose values are observed for t individuals:

$$\log\left(\frac{h_j(t)}{h_0(t)}\right) = \sum_{ij} \beta_j x_i \tag{2}$$

The *j* coefficients  $\beta$ ' represent the change in the logarithm of the hazard ratio, for a unit change in the explanatory variable *x*. The advantage of writing  $\beta$  as the logarithm of the hazard ratio is that the relative hazard ratio will always be positive: larger than 1 for  $\beta$  positive and less then one for  $\beta$  negative. The estimated hazard function for each individual becomes:

$$h_i(t) = e^{\left(\sum \beta_j x_i\right)} h_o(t) \tag{3}$$

where, for example,  $x_i$  is one if the mother of the ith child has been immunised and zero otherwise. The values of  $h_o(t)$  constitute the 'baseline hazard function' which is the hazard function of an individual for whom all the variables included in the model are zero. Estimating the coefficients  $\beta$  requires first constructing a data set in which the time of analysis, the censored observations and the recorded deaths are defined. The time of analysis is the number of t time intervals considered, for example from 0 to 11 months in the case of infant mortality. Censored observations are those children who at the time of the interview had not completed one year of age and have been exposed to mortality risk in less than 12 t time- intervals. Deaths

<sup>5</sup> The statistical package Stata contains a series of practical commands by which these operations can be carried out and the model can be estimated. See Stata (2001).

are recorded at the specific time interval, t, in which they occurred. In order to estimate changes in hazard of dying over time and between regions, we derive the value of the cumulative hazard function H(t) for 'typical' average infants, children and for children 'from five to ten'. Cumulative hazard functions are obtained by integrating both sides of Equation (3) from 0 to 11:

$$H_i(t) = e^{\left(\sum \beta_j x_i\right)} H_o(t) \tag{4}$$

In order to find the value of the cumulative hazard function for average infants, we calculate the values of the cumulative baseline hazard function  $H_0(t)$  from the estimated model, which represents the cumulative hazard (or probability of dying) of an individual for whom all the variables included in the model are zero for each of the three periods of interest (the twelfth month in the case of infant mortality, the 48th month for child mortality and 60th month for the mortality of children from five to ten). This value is then multiplied by the exponential of the linear prediction of the model  $e^{\sum \beta_j x_i}$  calculated at the mean values of the variables included in the model, which gives the expected value of the dependent variable (the mortality rate).

We examine the impact of the different determinants as follows. First, the procedure just described is used to calculate the expected mortality for the reference group (say, infants in AP), and the value called Y. Then, for the variable of interest (say  $x_i$ ) the value is changed from the mean for the reference group (AP) to the mean for the comparison group (Kerala, K), whilst retaining the AP means for all the other explanatory variables. The resulting mortality rate estimate is called Y. The percentage change in the dependent variable (mortality) resulting from the change in  $x_i$  is given by:

$$Y'/Y - 1 = e^{b_1(x_{1,AP} - x_{1,K})} - 1 (5)$$

This procedure produces probabilities of dying (mortality rates) that differ from those shown in Table 2. Details on the procedure used to derive mortality rates from the Cox model, and an explanation for the difference in estimates respect to Table 2 can be found in the appendix.

# 3. A basic model for infant and child mortality

The selection of determinants of infant and child mortality was based on the review of previous studies. This literature has found four main groups of determinants of infant and child mortality: biological (z), behavioural (b), environmental (e) and socio-economic (s). The probability of dying (hj) for a child i in the age group j, can be expressed as a function of these four sets of variables:

$$h_{ij} = f(z_{ij}, b_{ij}, e_{ij}, s_{ij})$$

$$(6)$$

The z set includes mother's age at birth, sex of the child, previous birth interval, multiple births and birth order. These variables are often defined in the demographic literature as the endogenous causes of death to be distinguished from exogenous causes of death (Pressat, 1972, and Preston, 2001). Endogenous causes of death are those factors that determine the congenital and constitutional characteristics of the child at birth and exert their influence on the infant during the first year of life and particularly during the first month. After the first month of life, exogenous factors, like accidents and infections, begin to influence infant survival, and they determine child survival after the first 12 months of life. Exogenous causes of death are modelled through sets b, e and s. The elements of s represent the level of exposure to the risk of infections or accidents. The elements of b represent the ability (or willingness) of the households to prevent these infections from occurring or to mitigate/eliminate their effects once they have occurred. The environmental variables included in the model are the access to safe water and sanitation and the exposure to clean fuel in the house. The behavioural variables included are prenatal care (tetanus immunisation and antenatal medical visits), breastfeeding. Socio-economic factors include wealth and maternal education.

#### **Biological factors**

The biological factors included in the model for infant mortality are: multiple birth, mother's age, length of preceding birth interval and sex of the child. The higher mortality of children from multiple births, especially during the neonatal period (first month of life), is common in areas of the developing world. Complications at birth and low birth weight are considered among the most important determinants of higher risk of death of twins (Sullivan, 1994). There is a U-shaped relationship between mortality and mother's age at birth. Infant mortality tends to be high for children born to young mothers and again, but to a lower extent, those at older ages. The higher mortality of children born to old mothers may be the result of a deterioration of the reproductive system with age, while the higher child mortality at very young maternal ages may occur because mothers have not fully developed to manage a birth (Pebley and Strupp, 1987). This effect may be found to be large in societies, like India, where women are giving birth to

children at extremely young ages. Children born after a short interval to the previous birth, generally present higher mortality rates. The key factor determining this relationship is the physical and nutritional depletion of mothers (Boerma and Bicego, 1993). The complex relationship between birth order and mortality is not well understood. In general, mortality is higher among first birth, which is usually explained by the observation that many mothers have their first child before having reached physical and reproductive maturity (Sullivan, 1994). For children, rather than infants, there is often a higher risk associated with being a higher birth order child. In general, males have higher mortality rates at all ages of childhood. Exceptions to this apparent genetically determined phenomenon have to be taken as the result of behavioural factors (Sullivan, 1994). Exceptions are indeed commonly observed in Asia, including India, as a result of a preference for male children (Croll, 2001).

#### **Environmental factors**

Acute respiratory infections and diarrhoea are the most important killers of children under the age of five in India and worldwide (IIPS, 2000). Air, water, food and fingers are the principal ways through which respiratory and intestinal diseases are transmitted. As suggested by the Mosley -Chen framework for the analysis of child survival in developing countries (Mosley and Chen, 1984), the physical environment to which children are exposed is likely to have an influence on mortality. Data are most commonly included on the type of water supply. For example, Merick (1985) who found a significant effect of the access to piped water on differences in child mortality in urban Brazil, and Brockerhoff and Derose (1996) report the same result from analysis of five East African countries. In the case of India, Kishor and Parasuraman (1998) use a combined water and toilet facility index, which they find to have a significant impact on both infant and child mortality.

In our model, we use the absence of a toilet as a possible vector of faecal contamination. The absence of an accessible source of water, and use of surface or rainwater, is used as a general sign of water contamination through which various diseases can be transmitted. Finally, we consider the use of unsafe fuel like dung, wood or coal for cooking as a possible concurring cause in the production of respiratory diseases. Although such a variable has not commonly been used, indoor air quality is particularly relevant in households with small houses and room for cooking.

#### **Behavioural factors**

According to UNICEF (1999), 73 per cent of deaths of children under five are a consequence of low-cost treatable diseases. Deaths caused by respiratory diseases, diarrhoea, measles, tetanus and pertussis, which account for more than 60 per cent of all deaths of children in developing

countries, could be easily avoided by the use of antibiotics, immunisation and oral rehydration methods. The knowledge and use of pre- and post-natal care seems to be the most important determinant of infant and child mortality. Das Gupta (1990) found a highly significant correlation between immunisation, knowledge of rehydration methods and lower mortality risk in rural Punjab. Both Murthi et al. (1995) and Gokhale et al. (2002) report that the availability and use of medical facilities affects child mortality in India. Beenstock and Sturdy (1990) also found that vaccination and the usage of medical facilities are important determinants of infant mortality in rural India. These findings are in line with those of Bidani and Ravallion (1997), who analyse poverty and health outcomes in 35 developing countries, and find that public spending on health has important effects in reducing infant mortality, in particular among poor households. Similarly, Hanmer et al. (2003) find immunisation to be a robust determinant of infant and child mortality in cross-country regressions - in contrast to Filmer and Pritchett (1999), who conclude that differences between countries are better explained by differences in income, mothers' education, ethnicity and religion. In this study, we use tetanus immunisation and the number of antenatal visits to assess the impact of prenatal care on the reduction of infant mortality. Unfortunately, data did not include information on postnatal care for the analysis of child mortality. However, as a proxy we included mother's knowledge of oral rehydration salts (ORS). Studies have found mother's knowledge of health care to have a significant impact on nutrition (Glewwe, 1999, and Christiaensen and Alderman, 2001), and can eliminate the significance of a more general mother's education term in such regressions. Knowledge of ORS might thus be similarly expected to affect mortality.

There are good reasons to believe that mother's milk, which is highly nutritious and rich in illness-preventive substances, has important effects in reducing infant mortality. For example, Palloni and Tienda (1986) report empirical evidence of an effect of breastfeeding on mortality risk, using data on 5000 Peruvian women. In our model, we included a variable for those children who were never breastfed, after excluding children who died in the first month of life.

As already noted, the relation between birth order and child mortality is not well understood. While higher parity reduces the mortality risk during infancy, it has been observed that outside sub-Saharan Africa high birth order increases child mortality in the age period 1-4 years (Sullivan, 1994). There may be medical factors behind this higher risk of high-birth order children, but higher birth order children also, by definition, come from larger families. Also, in larger families they may suffer from competing over limited resources and the higher probability

in spreading infectious diseases (Pebley and Strupp, 1987). Moreover, children in large families are likely to have a lower birth interval, which is a well-established risk factor; see, for example, the results of Kishor and Parasuraman (1998) for infant mortality.<sup>7</sup>

The existence of a large gender differential in child mortality in India is widely recognised and the mechanisms identified as responsible for excess female mortality rates are the underallocation of food and medical resources to female compared to males (Kishor, 1995). The reasons behind the operation of these mechanisms are still debated (e.g. Murthi *et al.*, 1995). However, in this study we will not consider the reasons for excess female mortality, but limit ourselves to identify the presence of higher mortality risk among female children and to test the hypothesis that this risk is higher for girls born in households without male children, as has been argued by Croll (2001). Das Gupta (1987), amongst others, argued that female disadvantage was particularly pronounced among children of higher birth order in Punjab. Murthi *et al.* (1995) mention Khan *et al.* (1989), who obtained similar findings in a study on rural Uttar Pradesh.

#### Socio-economic factors

The socio-economic factors included here are mother's education and economic well being. The correlation between mother's education and child mortality is well documented in a large number of studies and for various countries. Children of illiterate women have a much higher probability of dying at all ages and the older is the child, the greater is the difference in mortality between mothers of different educational levels (Sullivan, 1994). Education can be highly correlated with income, but as an explanatory variable should be able to capture the knowledge and the usage of health care methods and facilities. Mother's education is usually found significant when included as explanatory variable in regression models (Retheford, 1989, Palloni and Tienda, 1986, Pebley and Stupp, 1987) or is generally correlated with low levels of children mortality (Shiva Kumar, 1995, Boerma and Bicego, 1993, Bourne and Walker, 1991). However, this result can weaken or disappear when education is used together with indicators of health care (Das Gupta, 1990 and 1997).

Income levels may have an influence even on the circumstances at the birth of the child, for example on biological factors like mother's age or birth interval (Sullivan, 1994). However, once the child is born, the biological characteristics of the mother and of the child are given. Therefore, income may have an effect on mortality probabilities only if not all income-correlated

<sup>7</sup> Models should include both birth order and birth interval to separate out these effects, though there may be a problem of multicollinearity.

behaviours and risks are included in the model. Hence, it can be argued that income as explanatory variable can be interpreted as a test for omitted variables (Wolpin, 1997). However, most authors include the variable, implying that it has some sort of direct effect.

Several authors have considered per-capita income an important determinant of the reduction in child mortality. Fogel (1997), in his historical study of European countries, maintains that the secular decline in mortality rates has been a result of the increase in living standards via the improvement in nutritional status. Cross-country regressions (e.g. Filmer and Pritchett, 1999; and Hanmer et al., 2003) tend to find an elasticity of child mortality with respect to income per capita of between –0.6 and –0.8; although White (2003) shows that the elasticity varies according to the level of immunisation. However, in India, the explanatory power of per capita income as a determinant of cross-state differences in child mortality is generally weak because there are other factors, not necessarily correlated with income, that have important effects on child mortality (Murthi et al., 1995). Indian studies using household data find a small but significant effect (Kishor and Parasurman, 1998).

The Indian NFHS does not contain data on income or expenditure, but there is extensive information on a variety of assets owned by each household, which we use to construct a wealth index. We used these data to build a wealth index based on possession of durable goods.<sup>8</sup> Whilst income is more commonly used when available, all the variables included in the index are correlated to income.<sup>9</sup>

The wealth indicator used here is a simple score index based on the possession of durable goods. The index is obtained by summing dummy variables representing the ownership of a given good, scaled to one dividing by the number of goods used in the index. The durable goods used are: radio, TV set, refrigerator, bicycle, motorcycle, car, land, livestock, clock and sewing machine. Access to services in the form of type of toilet, water and electricity was not included in the index in order to avoid correlation with the variables representing the environmental factors.

<sup>9</sup> It may be argued that assets are, in fact, more appropriate than income, since the former can be drawn upon in times of hardship (e.g. illness).

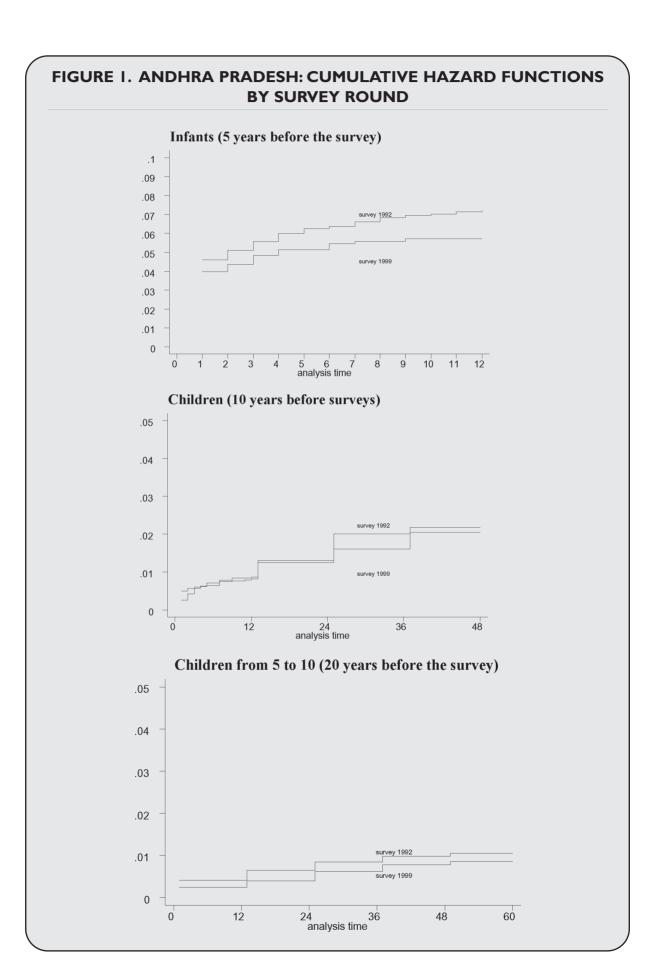
# 4. Determinants of infant and child mortality in Andhra Pradesh

#### The empirical hazard functions

Figure 1 shows the empirical cumulative hazard functions for infants, children and children from 5 to 10 of Andhra Pradesh. The horizontal axis represents the study time, which is 12 months for infants, 48 months for children and 60 months for children from 5 to 10. In each graph, the vertical axis shows the values of the cumulative hazard functions obtained using the data of the surveys of 1992 and 1999 respectively. These values are calculated using the Nelson-Alen method<sup>10</sup> and can be interpreted as the probability of dying at different time intervals. The hazard functions for infants indicate a slight reduction in mortality risk between 1992 and 1999. The functions for children show no substantial change and their values are hardly distinguishable. The functions for children from 5 to 10 also show relatively little change in mortality between the two periods. These visual impressions are confirmed by a test (Table 4) on the difference of the survivor functions from which the hazard functions are derived. The hypothesis of equality of the survivor functions from the two surveys is not rejected for any of the three age groups. For the age group 5-10, the number of deaths is too small to conduct further statistical analysis.

## TABLE 4. WILCOXON TEST FOR THE EQUALITY OF SURVIVOR FUNCTIONS OF 1992 AND 1998

	Chi square	P-value
Infants	1.85	0.17
Children	0.24	0.62
Children from 5 to 10	1.81	0.18



### **Determinants of infant mortality**

Table 5 presents the result of the regression analysis of infant mortality using a Cox proportional hazard model. This model includes a selection of biological, behavioural, socio-economic and environmental factors. The results are not expressed as coefficient estimates, but in the form of derived hazard ratios, which are shown in the first column together with an indication of their significance.

Variable e	Model with	Survey 1992 les	Survey 1999 A	ttributable change in mortality	Share of the
	Hazard ratio	Mean value	Mean value	% change	difference
Estimated infant mortality		61.4	45.5		
Biological factors					
Multiple birth	5.45**	0.02	0.01	-1.8%	6.5%
Mother's age	0.89	21.84	21.91	0.0%	0.0%
Mother's age square	1.01*				
Birth interval (24 – 47 months)	0.57***	0.37	0.34	1.5%	-5.6%
Birth interval (48+ months)	0.55**	0.15	0.14	0.7%	-2.6%
Birth order	0.91	2.60	2.24	1.6%	-5.9%
Birth order square	1.01				
Female infant	0.88	0.49	0.51	-0.2%	0.7%
Behavioural factors					
Two or more tetanus injections	s 0.60***	0.74	0.83	-4.5%	16.6%
Antenatal visits	0.90***	2.76	5.19	-22.3%	82.9%
Never breastfed	1.68*	0.03	0.02	-0.5%	1.7%
Environmental factors					
Unsafe water	0.61	0.02	0.03	-0.4%	1.5%
Unsafe sanitation	0.97	0.78	0.72	0.2%	-0.8%
Unsafe fuel	1.13	0.82	0.74	-1.0%	3.6%
Socio-economic factors					
Household wealth index	1.07	0.22	0.29	0.5%	-1.7%
Wald chi square	144.25		n = 3253		
P-value	0.000		(208 dead infants)		
Log likelihood	-1624.8				

In the case of dummy variables, the hazard ratio is the ratio of the mortality risk of an infant with given characteristics respect to an infant without that characteristics. Thus, for example, an infant born from a multiple birth turns out to have a probability of dying which is more than five times the probability of dying for an infant born in ordinary conditions. In the case of continuous variables, the interpretation of hazard ratios is more complicated because these are calculated respect to the baseline hazard function of an infant for whom all variables are zero, but it remains the case that a ratio of less than one indicates a reduced risk associated with that factor.

The third and fourth columns report the mean values of the variables at the time of the first and of the second survey respectively. Using the method described above, the fourth column calculates the percentage change in infant mortality between the two periods resulting from the mean change in that variable. The last column expresses these changes as a percentage of the total change.

#### **Biological factors**

All biological factors have a significant influence on infant mortality with the exception of birth order (which has the expected value of less than one) and sex of the infant. The highest hazard ratio is found for infants from multiple births. Understandably, there is little difference in the proportion of multiple births between the two surveys and, therefore, the effect on the overall change in mortality is minimal. Mortality has the expected inverted-U relationship with mother's age at birth with a hazard ratio of less than one on age but more than one for age squared, with a turning point of 21.4 years.<sup>11</sup> However, the ratios are both close to one and mean's mother age is just above this turning point, so that there is a negligible impact in mortality from the slight increase in mother's age between the two surveys.<sup>12</sup>

The hazard ratio for infants born after large birth intervals is particularly low, but the percentage of infants of large birth intervals has decreased between the surveys thus producing a slight increase in mortality rate. Birth order of the child is not significant and the hazard ratios are closed to one. However, there has been a decline in the average birth order between the two surveys as a result of the reduction in fertility rates. As a result, a lower average birth order has contributed to the reduction of mortality rate. Female infants appear to have a lower probability

<sup>11</sup> The turning point is given by age=-b1/(2b2) where b1 and b2 are the estimated coefficients on age and age squared respectively. Note it is the regression coefficient, not the hazard ratio which is used in this calculation.

<sup>12</sup> It would not make sense to analyse mother's age and mother's age squared separately, so the mother's age row shows the result of varying both these variables simultaneously.

of dying, but the coefficient is not significant and the relationship between mortality and sex of the child will be discussed in section 5.

#### Behavioural factors

Three behavioural factors were included in the model: tetanus immunization, prenatal check-ups and breastfeeding. The first two factors have proved to be particularly important in the reduction of infant mortality rates in Andhra Pradesh. Tetanus is one of the most important causes of neonatal death in India (IIPS, 2000a). Infants can be infected at the moment of delivery when unsterilised instruments are used to cut the umbilical cord. The infection is particularly common among infants delivered in unhygienic environments. When the mother is vaccinated, the immunity is transferred to the foetus trough the placenta. In 1975-76, the Government of India launched a program of tetanus immunisation for expectant women, yet full coverage had still not been reached by 1998. According to the National Immunisation Schedule, a pregnant woman should receive two doses of tetanus toxoid injection and a third reinoculation is recommended every three years. In our model, infants born to mothers who received two or more tetanus vaccinations have a hazard ratio of 0.6. The percentage of mothers vaccinated has increased between the two surveys, and this increase has made a substantial contribution to the reduction in mortality rates.

The Reproductive and Child Health Program of the Government of India recommends that as part of antenatal care, pregnant women receive at least three antenatal check-ups (IIPS, 2000a). Antenatal check-ups consist of a series of procedures directed to detect pregnancy complications. The regression results show that the number of antenatal visits is highly significant in reducing infant mortality risk. More importantly, the average number of such visits among expectant mothers has almost doubled between the two surveys from a low 2.7 in 1992-93. The decrease in mortality rate produced by this change is by far the largest factor driving the mortality reduction in AP in the 1990s. The combined effect of increased antenatal visits and tetanus injections accounts for over 90 per cent of the estimated reduction.

Whilst not breastfeeding increased the risk of mortality, the percentage of mothers not doing so was small and changed little between the two years. Hence, this variable does not explain much change in the infant mortality.

#### **Environmental factors**

None of the environmental factors included in the model proved to be significant, although the first two have the expected 'sign'.

#### Income effects<sup>13</sup>

The inclusion of the wealth indicator among the regressors did not increase the explanatory power of the model. The corresponding hazard ratio is not significantly different from one. Once other factors are taken into account, the infant mortality rate in Andhra Pradesh appears to be independent from households' wealth. Bivariate analysis does show a relationship between income and mortality. Our results thus show that the channels for higher income to affect mortality are mainly the greater propensity of higher income women to use antenatal services and receive tetanus immunisation. Once near universal coverage of these services is achieved, then this channel cannot operate, which will weaken the link between income and infant mortality.

#### **Determinants of child mortality**

Table 6 presents the results for child mortality.<sup>14</sup> Biological factors have been excluded from the analysis since child mortality should be entirely explained by behavioural and environmental factors. The exception is birth order, but the higher risk for birth order children, rather than infants, can be characterised as behavioural rather than biological.

#### Behavioural factors

Female children have a higher probability of dying than male children, clearly indicating the presence of behaviours discriminating against young girls. Naturally, there is no change in the percentage of female children between the two surveys and the overall mortality rate remains unchanged. Here, mother's age is associated with a higher risk over the whole range, but since the average change in average mother's age between the surveys is minimal, the final effect on mortality rate change is negligible. As expected, the mortality risk of children increases with birth order and is highly significant. The reduction in average birth order between the surveys is a product of fertility decline.

Finally, we include mother's knowledge of ORS, which has a very pronounced effect on child survival. Diarrhoea is the second most important killer of under-five children worldwide, and is responsible for a significant proportion of deaths of Indian children. Most deaths from diarrhoea are caused by dehydration, which can be easily prevented through the administration of

<sup>13</sup> The maternal education variables were dropped from the regression. The estimated coefficients were perverse, though insignificant.

Our analysis explored differences in determinants at the sub-state level. In AP, three regions are identified. The data did not support the idea of a different data generation process for the different regions (partly as the small number of deaths at that level makes the standard errors large). The region intercept dummies were significant in the child mortality regression, but not in that for infant mortality. They are reported in Table 6, but not subject to further discussion.

<sup>15</sup> The coefficients give an inverted U rather than the expected U, but the hazard ratio on age squared is practically unity.

rehydration solutions. In the early nineties, the Indian Government launched a program directed to instruct mothers on how to manage diarrhoea by using ORS packets (IIPS 1995 and IIPS 2000). The effects of this programme can be seen in the reduction of mothers unfamiliar with ORS, which has delivered the largest single impact on child deaths in AP over this period.

Variable	Model with environmenta variab	Survey 1992 les	Survey 1999 A	attributable change in mortality	Share of the
Overall mortality change	Hazard ratio	Mean value  8.	Mean value  3.7	% change	difference
Behavioural factors					
Female child	1.64***	0.50	0.48	-0.8%	2.9%
Mother's age	1.25*	21.73	21.77	0.2%	-0.4%
Mother's age square	0.99**				
Birth order	1.24***	2.66	2.48	-3.9%	14.5%
Lack of knowledge of ORS	1.54**	0.47	0.14	-13.1%	49.3%
Risk factors					
Unsafe water	3.09***	0.01	0.03	1.9%	-7.0%
Unsafe sanitation	0.99	0.77	0.73	0.1%	-0.2%
Unsafe fuel	1.76	0.81	0.74	-3.7%	13.9%
Regions					
Coastal	0.69*	0.42	0.38	1.6%	-5.9%
Telangana	0.63**	0.39	0.45	-2.4%	9.0%
Socio-economic factors					
Wealth index	0.37*	0.23	0.28	-5.2%	19.6%
Mother illiterate	1.11	0.73	0.63	-1.1%	4.0%
Scheduled caste	1.05	0.15	0.20	0.3%	-1.0%
Scheduled tribe	1.70*	0.07	0.06	-0.1%	0.4%
Hindu	1.79*	0.86	0.85	-0.9%	3.5%
LR chi-square	97.26		n = 7787		
P-value	0.000		(144 deaths)		
Log likelihood	-1229.75				

#### **Environmental factors**

Environmental factors are important determinants of child mortality, probably, as sources of various infections. Unsafe water is associated with very high hazard ratios, although these children represent an extremely small percentage of total population. The number of these children has increased over time, thus, producing a certain increase in mortality rate. The lack of a proper system of sanitation shows no significant effect on hazard ratio and the final effect on mortality is close to zero since the percentage of children without safe sanitation has remained unchanged. The use of contaminating fuel inside the house increases the probability of dying as

expected, although the effect appears insignificant in this regression. The use of unclean fuel has decreased over time generating a reduction in mortality rate.

#### Socio-economic factors

Children from wealthier households have a much lower hazard ratio and the improvement in living standards has brought about a significant reduction in child mortality rate. It is difficult to know which behaviours are facilitated by higher income and so responsible for this change. Reasonable candidates are better nutritional levels and larger expenditure on health. The increase in the wealth index over the period accounts for 20 per cent of the decline in child mortality.

Children of uneducated mothers have a higher probability of dying, but the coefficient is not statistically significant. The insignificance of this variable may be in part explained by its high correlation with the wealth index (a simple correlation coefficient of –0.55), and partly as education is mediated through the ORS variable (lack of knowledge of ORS is correlated to mothers' illiteracy with a coefficient of 0.35). Children of scheduled caste and, in particular, children of scheduled tribes are at a higher mortality risk. Scheduled castes and scheduled tribes are castes and tribes that the government of India considers as socially and economically backward and in need of special protection from injustice and exploitation (NIIP 2000a). The number of children from these castes has not changed over the period considered, thus leaving the mortality rate unchanged. Children of Hindu families also show higher mortality risk.

## 5. Gender discrimination in Andhra Pradesh

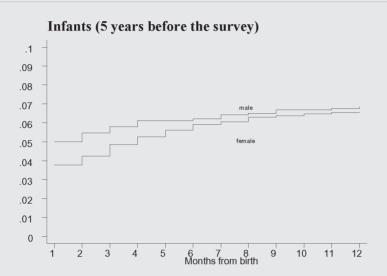
As mentioned in section3, the presence of a 'son preference' in India is well documented, and several empirical studies have found evidence of gender discrimination against girls. In what follows, we will measure the impact of gender discrimination on mortality rates, without attempting to identify the reasons behind it. Figure 2 presents the cumulative hazard functions disaggregated by gender for infants, children and children from 5 to 10 of Andhra Pradesh. In the case of infants, the mortality risk is slightly higher for males, but not significantly, as shown by the Wilcoxon test on the equality of the underlying survival functions in Table 7. The female hazard function seems to be steeper, reflecting a slower decrease in the mortality risk of female infants over the first year of life. In the case of children, the graph clearly shows a larger mortality risk for females, which is confirmed by the test on the equality of the survival functions. Finally, the graph for children from 5 to 10 shows no significant difference between males and females. The slopes are almost identical indicating the same decreasing rate of mortality risk over time.

## TABLE 7. WILCOXON TEST FOR THE EQUALITY OF SURVIVOR FUNCTIONS OF MALE AND FEMALE CHILDREN

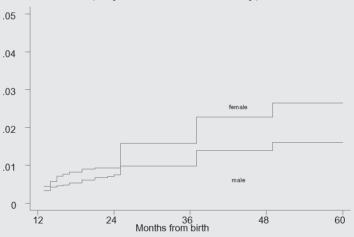
Chi square	P-value
0.36	0.54
8.15	0.00
0.22	0.64
	0.36 8.15

Similar results were obtained from the regression results. The coefficient representing a female infant is not significant in the regression of infant mortality (Table 5) and the hazard ratio for female infants respect to male infants is 0.88, indicating a lower probability of dying among females. The opposite is true for children, however: the coefficient is highly significant in the regression applied to children from one to five years (Table 6), which shows that girls have a substantially higher probability of dying in this age group.

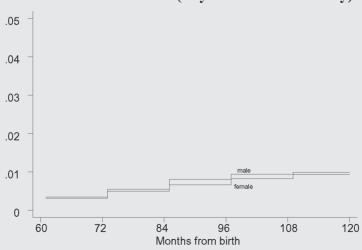




#### Children (10 years before the survey)



Children from 5 to 10 (20 years before the survey)



However, the lower mortality rate of female infants should not be taken as evidence of the absence of gender discrimination in this age group for two reasons. In Table 8 we show the result of a regression containing all the variables already used for the regression in Table 5, but with the inclusion of three dummy variables representing female infants having no male siblings at different birth orders. The hazard ratios of the other variables are not reported because they are almost identical to those of Table 5. Females born without having male siblings have a much higher probability of dying during the first year of life, and this probability increases with the birth order of the female infant. By contrast, hazard ratios of females with male siblings are not significantly different from one. These results confirm similar findings observed in other Indian states and are a reflection of a strong 'son preference' (Das Gupta, 1990 and Croll, 2001).

## TABLE 8. HAZARD RATIOS OF FEMALE INFANTS WITH AND WITHOUT MALE SIBLINGS COMPARED BY BIRTH ORDER

	Girl with all female siblings	Girl with male siblings
2nd birth order	2.16*	1.13
3rd birth order	3.06**	1.02
4th and more birth order	4.65***	1.03
Wald chi square	170.0	141.6
P-value	0.000	0.000
Log likelihood	-1618.0	-1623.4

The second reason for not considering the female infant hazard ratio of Table 5 as a sign of no gender discrimination is that higher male mortality is generally concentrated in the neonatal period (Figure 2) for genetically determined reasons. Male infants have a higher probability of dying in the first month of life and the majority of infant deaths occur during this first month of life. But what happens after the first month of life, when mortality is mainly determined by behavioural rather than biological reasons? In order to answer this question, we used a method developed by Bourgeois-Pichat and illustrated in Pressat (1972). This method consists of the estimation of the number of dead infants for behavioural reason only, by abstracting from the biological deaths. We applied this methodology to males and females separately, we calculated the ratio of mortality risk for behavioural reasons of female over male infants and we obtained a value of 1.63, which is much in line with the hazard ratio of 1.68 for female children shown in Table 6.

<sup>16</sup> The estimated model was that shown in Table 5, plus an intercept dummy for children with all male siblings, and the interactive term of sex of child, dummy for all male siblings and birth order. It is the coefficients of these three variable interactive terms, which are reported in Table 8.

## 6. Kerala and Andhra Pradesh compared

Kerala's exceptional performance with respect to social indicators is well known. As Table 2 showed, not only are Kerala's mortality rates for infants (16 per 1000) and children (2.6 per 1000) the lowest in India, but they are also still decreasing rapidly. The decline of Kerala mortality rates is not a new phenomenon, dating back to the 50s and 60s. This decline has been explained in very different way by the demographic and sociological literature (Robin, 1992). According to some authors, the Keralan achievements in terms of low mortality and fertility rates are the result of educational and family planning programs promoted by the government. Others maintain that a more equitable distribution of resources relative to other Indian states has made health services and information accessible to a larger part of the population. According to some observers a higher autonomy of Keralan women and a greater political consciousness among the poorer sectors of the population has played a large part in this process. The majority of observers believes, however, that the provision of curative medical services, in particular to mothers and babies, and the adoption of public health actions against preventable diseases are responsible for the impressive reduction in mortality rates.

The difference in mortality rates between Kerala and Andhra Pradesh (which is around the Indian average) is striking. Figure 3 compares the cumulative hazard functions of infants and children of Andhra Pradesh. Here, data from the two surveys have been considered together in order to concentrate only on regional differences. The difference in the prospects of survival for the infants and children from the two states is apparent. There is not only a difference in the distance between the hazard functions, but their slopes are also different. This means, firstly, that infants and children of Andhra Pradesh have a higher probability of dying at all time intervals and, secondly, that the probability of dying over the lifetime is decreasing more slowly for the infants and children of Andhra Pradesh compared to those in Kerala. Table 9 shows the results of the test on the equality of the hazard functions, which confirms the large differences observed.

## TABLE 9.ANDHRA PRADESH AND KERALA: WILCOXON TEST FOR THE EQUALITY OF SURVIVOR FUNCTIONS OF INFANTS AND CHILDREN

 Chi square
 P-value

 Infants
 68.84
 0.00

 Children
 44.17
 0.00

Table 10 presents the results of a Cox regression for infant mortality. The hazard ratios are calculated from a pooled sample of infants from Andhra Pradesh and Kerala. The table also contains the mean values of the variables for each state, and, in the last column, and the difference in mortality rates between the states that can be attributed to each variable.

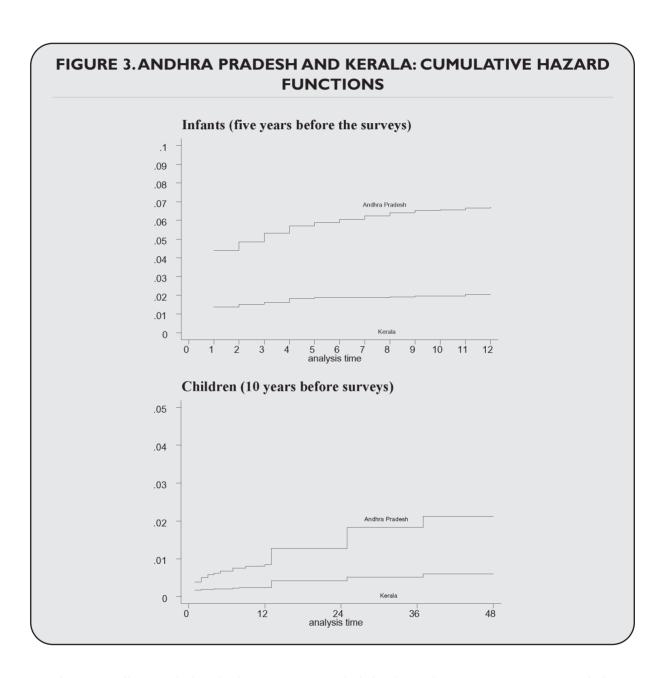
#### **Biological factors**

The hazard ratio for infants born from a multiple birth is extremely high, but, understandably, there is no difference in this respect between Andhra Pradesh and Kerala. The infants' chances of survival are increasing with the mother's age at birth. In spite of the Child Marriage Restraint Act (1978) that fixed the minimum age at marriage for women in India at 18, many Indian women are still marrying at very young ages. The number of these women is much higher in Andhra Pradesh than in Kerala, where in 1998-99 (NIIP, 2002) only 5% of all women were married by age 15. According to Mahadevan and Sumangalan (1987), three reasons can explain a late age at marriage in Kerala: the need for completing education, the social obligations (i.e. the need for looking after the parents, brothers etc.) and the financial cost of weddings (i.e. dowry, ornaments, house etc.). The Keralan average mother's age at birth is not exceptionally high, but higher than that in Andhra Pradesh and this has some effect on the difference of mortality rates between States. Birth intervals are on average larger in Kerala compared to those in Andhra Pradesh, thus leading to lower mortality rates.

Although classified as biological factors, the lower average birth order, longer birth interval and higher mother's age in Kerala are all functions of the lower fertility rate in that state, which is itself a function of women's status, in part associated with the importance attached to female education. Hence, biological factors may also be affected by indirectly by social policy, as well as directly through reproductive health services.

#### Behavioural factors

Two variables are particularly important in explaining the difference in mortality rates between Kerala and Andhra Pradesh: tetanus immunisation and the number of antenatal visits. The hazard ratio of infants born to mothers with two or more tetanus vaccinations is significantly smaller than one. There is a notable difference between states in the percentage of mothers immunised which results in a substantial difference in mortality rates. This is not surprising given that Kerala (along with Goa) ranks third after Tamil Nadu and Punjab in the coverage of two or more tetanus vaccination (NIIP 2002). The hazard ratio for a unit change in the number of antenatal check-ups is significantly smaller than one. The difference in this respect between the states is impressive. The average number of antenatal visits in Kerala is double the average



number in Andhra Pradesh, which in turn is just slightly above the minimum recommended. Just increasing AP's level of antenatal visits to that in Kerala would alone achieve a 40 per cent reduction in infant mortality.

The practice of breastfeeding seems to have a significant effect on infant survival, but the number of infants not breastfed in both states is so low, that there is no effect on mortality differences.

TABLE 10. ANDHRA P.AND KERALA: INFANT MORTALITY,
MEAN VALUES OF VARIABLES AND ATTRIBUTABLE
DIFFERENCE IN MORTALITY

Variable	Hazard ratio	Kerala	Andhra Pradesh	Attributable difference in mortality	Share of the difference
Estimated infant mortality		15.1	51.9	,	
Biological factors					
Multiple birth	5.14***	0.02	0.02	0.0	0.0%
Mother's age	0.89*	24.52	21.87	38.2	21.0%
Mother's age square	1.00**				
Birth interval (<24 months)	1.19	0.16	0.15	0.4	0.2%
Birth interval (24 – 47 months)	0.59***	0.33	0.28	-4.9	-2.7%
Birth interval (48+ months)	0.51***	0.17	0.18	2.1	1.2%
Birth order	0.91	2.11	2.47	-3.3	1.8%
Birth order squared	1.00	6.67	8.66	-0.8	0.4%
Female child	0.84	0.50	0.50	0.1	0.1%
Behavioural factors					
2 or more tetanus injection	0.54***	0.94	0.77	7.9	4.3%
Number of antenatal visits	0.87***	7.5	3.6	74.3	40.9%
Never breastfed	1.70*	0.02	0.03	1.0	0.6%
Risk factors					
Unsafe water		0.00	0.02		
Unsafe sanitation		0.25	0.76		
Unsafe fuel		0.92	0.85		
Wealth indicators					
Wealth index	0.91	0.37	0.32	0.4	0.2%
Andhra Pradesh	1.97***	0.00	1.00	96.9	53.3%
Wald chi square	291.4		n = 5971		
P-value	0.000		Dead infants: 262		
Log likelihood	-2152.1				

#### **Environmental factors**

None of the environmental factors had a significant impact on infant mortality risk and, therefore, these factors were not considered when computing the contribution of each variable to the difference in mortality between Andhra Pradesh and Kerala.

#### Socio-economic factors

The effect of the wealth indicator is negligible, partly because its hazard ratio is not that different from one (and is not significant), and partly because there is very little difference in households' wealth between Kerala and Andhra Pradesh.

Even though education has been indicated by many authors as a key factor for the impressive Keralan performance in terms of low mortality rates, we could not directly explain any difference in mortality risk between Kerala and Andhra Pradesh by the use of educational variables. Finally, it should be noted that the dummy variable indicating that the infant is from Andhra Pradesh is very important in explaining the difference in mortality respect to Kerala, thus pointing to the omission of relevant explanatory variables.

#### Differences in child mortality

Table 11 illustrates the results of applying the same type of analysis used for the study on infant mortality to child mortality in Andhra Pradesh and Kerala. The observations made in Section 4 on the general interpretation of the behavioural factors included in the model for child mortality also apply here.

#### Behavioural factors

Female children have higher hazard ratios. This is particularly true for Andhra Pradesh (see section 5), while Kerala is usually seen as an exception to the widespread 'son preference' in India. This view is strongly supported by our analysis, which allows the hazard ratio for female children to vary between the states. There is an insignificant risk of being female in the 'base case' of Kerala (indeed the ratio is well below unity), but in AP a female child has a two and a half times greater mortality risk than a male child. This gender discrimination accounts for over 40 per cent of the difference in mortality between the two states.

Mortality risk increases with mother's age at birth, probably reflecting the effect of variables, which are positively correlated with time. Since mother's age at birth is on average smaller in Kerala, this factor has some effect on the difference in mortality rates. There is a substantial difference in knowledge of ORS between the states, it being much more widespread in Kerala. This difference plays some part in explaining child mortality differences.

#### **Environmental factors**

The effect of environmental factors was not found significant, in spite of a potential large attributable difference in mortality rates, given that basic services (sanitation in particular) are more largely available in Kerala than in Andhra Pradesh.

#### Socio-economic factors

The hazard ratio associated to one unit change of the wealth index is extremely low. The average difference in the wealth difference between the two states is not particularly high at 15 per cent. This seems a good approximation of income differences between Kerala and Andhra Pradesh. Using data on net domestic product between 1990 and 1999, we found an average difference of 15 per cent between Kerala and Andhra Pradesh. Since Keralan households are only slightly wealthier than those in AP, income differences turn out to be unimportant in explaining the difference in mortality rates.

Children of illiterate mothers have much higher probability of dying. The difference in the percentage of illiterate mothers in the two states is impressive. The Kerala illiteracy rate is 11 per cent with very little difference by gender and between rural areas (NIIP, 2002). As a result, education directly explains nearly one fifth of the difference in mortality rates between Kerala and Andhra Pradesh. As argued above, female education also has indirect effects which reduce both infant and child mortality.

Among the other socio-economic variables, only the hazard ratio associated to scheduled castes was found significant. However, the percentage of households of backward castes is only slightly larger in Andhra Pradesh, and the effect on the difference in mortality is very small.

## TABLE II.ANDHRA P.AND KERALA: CHILD MORTALITY, MEAN VALUES OF VARIABLES AND ATTRIBUTABLE DIFFERENCE IN MORTALITY

Variable	Hazard ratio	Kerala	Andhra Pradesh	Attributable difference in mortality	Share of the difference
Estimated mortality rates		5.4	15.3	·	
Behavioural factors					
Female child	0.63	0.48	0.49	-0.6%	-0.4%
Female child (Andhra Pradesh)	2.59**		0.49	59.9%	42.7%
Birth order	1.23***	2.21	2.58	7.8%	5.6%
Mother's age	0.96**	24.3	21.8	11.8%	8.4%
Lack of knowledge of ORS	1.47**	0.13	0.32	7.7%	5.5%
Risk factors					
Unsafe water	2.88***	0.01	0.02	1.6%	1.2%
Unsafe sanitation	1.27	0.25	0.75	12.7%	9.1%
Unsafe fuel	1.22	0.89	0.78	-2.2%	-1.5%
Socio-economic factors					
Wealth index	0.33**	0.26	0.28	2.3%	1.6%
Mother illiterate	1.51*	0.12	0.68	26.2%	18.6%
Scheduled caste	0.93	0.05	0.17	-0.9%	-0.6%
Scheduled tribe	1.57*	0.02	0.06	1.9%	1.4%
Hindu	1.35	0.47	0.86	12.3%	8.8%
Rayalseema (AP)	1.56***	0.18		-0.3%	-0.2%
LR chi2	142.0		n = 13671		
P-value	0.000		Dead children: 175		
Log likelihood	-1570.6				

Note: \* significant at 10%, \*\* significant at 5% and \*\*\* significant at 1%.

In summary, it is social factors that underpin differences in child mortality between AP and Kerala. These social factors are linked to female empowerment, which has been argued by Caldwell (1986) to be critical for reducing mortality. Most important is discrimination against female children, which appears to be absent in Kerala. Female education has a substantial direct effect on lower mortality, and an indirect one through knowledge of ORS and the link between education and lower fertility with associated higher mother's age and lower average birth order. The combined impact of these effects show that if AP could attain the level of female empowerment present in Kerala that alone would be sufficient to meet the MDG.

### 7. Conclusions

The change in infant mortality rates of Andhra Pradesh during the nineties can be largely explained by an expansion of preventive health care and, in particular, by a growing practice of antenatal check-ups among pregnant women. Several biological factors were found significant in determining infant mortality rates. In contrast with other similar studies, we could not find evidence of an impact of income, education or environmental factors on infant mortality. Child mortality rates in AP have decreased over the period partly as a result of a general improvement in living standards, but also because of increased knowledge of ORS. Evidence was found of gender discrimination, especially by the increased mortality risk of higher-birth order girls without male siblings.

The comparison with the impressive performance of Kerala in achieving low infant and child mortality rates reinforces the importance of health interventions, but more especially of the status of women in society. The enormous difference in infant mortality rates between these two states is explained by both much larger coverage of preventive health care in Kerala and by a long term decline in fertility which has resulted in older mothers' age at birth and larger birth intervals. The difference in child mortality rates can be attributed only in a small part to higher income levels in Kerala and the majority of the difference can be attributed to a gender discrimination effect and the much higher educational levels of Keralan women. Education also underlies some of the social changes, such as later age of marriage and birth, which also directly affect mortality.

The evidence in this paper thus strongly supports the argument that social service provision can play a direct and important role in reducing mortality, even when comparing states at similar income levels. This is particularly true for infant deaths, which account for a growing share of under-five mortality as mortality rates decline (White, 2003). Currently the provision of services between states varies greatly. Use of antenatal care ranges from a high of 98 per cent in Kerala to a low of less than 40 per cent in conflict-affected Nagaland and just 44 per cent in neighbouring Assam (Gokhale et al., 2002: 140). There is an even wider range for some other indicators of the use of health services: close to ninety per cent of babies are delivered in hospital in Goa and Kerala, compared to just 6 per cent in Nagaland and 11 per cent in Assam, Uttar Pradesh and Rajasthan. Over 90 per cent of mothers in Kerala have received iron and folic acid during pregnancy compared to less than a quarter in Nagaland and Bihar (ibid: 140). Similarly, female illiteracy is 17 per cent in Kerala but over two-thirds in Madhya Pradesh and Uttar Pradesh and three-quarters in Rajasthan. Only by closing these gaps in the availability of social services can India expect to reach the Millennium Development Goal of a two-thirds reduction in infant and child mortality.

The position of women also appears key, especially for child mortality. Discrimination against girls accounts for their higher mortality in AP, whereas this discrimination is not present in Kerala. Aside from this direct effect, increasing the status of women improves their access to education, which directly reduces mortality and does so indirectly through lower fertility. Meeting the MDG of gender equality should form a key part of the strategy to achieve the child health goals.

## **Appendix**

The procedure used to calculate the NFHS mortality rates reported in Table 2 (synthetic cohort probabilities of death), was first developed by Somoza and then modified by Rutstein  $(1984)^{17}$ . Mortality rates in Table 2 are based on death probabilities ( $m_i$ ) estimated for the following age groups: 0 to 1 month, 1 to 2 months, 3 to 5, 6 to 11, 12 to 23, 24 to 35, 36 to 47 and 48 to 59. Probabilities of death, for each single specific age group, are derived by using:

$$m_{i} = \frac{D_{(t-a,t'-b)} + \frac{1}{2}D_{(t-b,t-a)} + \frac{1}{2}D_{(t'-b,t'-a)}}{S_{(t-a,t'-b)} + \frac{1}{2}S_{(t-b,t-a)} + \frac{1}{2}S_{(t'-b,t'-a)}}$$
(1)

Where, t and t are the lower and upper time limits for which mortality is estimated. a and b are the lower and upper limits of the age groups specified above. S is the number of children born (and survived to the preceding age group interval) during the time interval defined by the subscript. D is the number of children survived (S) who died.

Children born in the interval (t-a, t'-b) were exposed to the risk of dying during the whole period considered. Children born in other intervals were exposed to the same risk for a shorter time. Therefore, only half of these children are reported as survivors or dead in the calculation of m<sup>18</sup>.

The m probabilities of death of each specific group are used to build cumulative survivor and hazard functions. The values of the cumulative hazard function (H(t)) are obtained by subtracting from one the values of the cumulative survivor function:

$$H_{(t)} = 1 - \prod_{i}^{t} (1 - m_i)$$

The probabilities of dying at each time t are the values of the hazard function at the same time t.

The procedure used to retrieve mortality rates from the Cox model is the following. The Cox model estimates a set of parameters  $\beta_1$ ,  $\beta_2$ , ..., bn for a set of variables  $x_1$ ,  $x_2$ , ...,  $x_n$ , and its linear prediction is:

$$\hat{y} = \beta_1 x_1 + \beta_2 x_2 + \dots + \beta_n x_n \tag{2}$$

<sup>17</sup> A full description is in Sullivan et al. (1994).

When mortality rates are calculated for the period immediately before the survey, all deaths (instead of one half) are counted for the children born in the interval (t'-b, t'-a), even though they were exposed only for half of this time. Because the death recorded must have occurred before the date of the survey.

We calculate this linear prediction at the mean values of the variables included in the model:

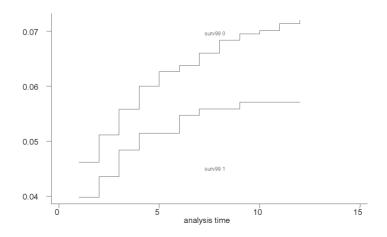
$$\hat{y} = \beta_1 \overline{x}_1 + \beta_2 \overline{x}_2 + \dots + \beta_n \overline{x}_n \tag{3}$$

We then multiply the exponentiated value of the linear prediction (at the mean values of the x's) by the value of the baseline cumulative hazard function at the time of interest, for instance the twelfth month in the case of infant mortality:

$$\hat{H}(12) = e^{\hat{y}} H_0(12) \tag{4}$$

The value obtained  $\hat{H}_{(12)}$ , is the value of the hazard function at the twelfth month for an 'average' infant. This value is different from the IMR of Table 2, because the samples of individuals used for the calculations are different. The IMR retrieved from the Cox model includes censored observations, which account only for a half in the other method. Moreover, the other method includes half of the observation for the interval preceding t-a. IMR of Table 2 therefore, are scaled back on time respect to those obtained through the Cox model. Since mortality rates are generally decreasing over time, Cox mortality estimates are lower than NFHS estimates.

The samples used in the Cox model are different from those used in table 2 in another sense. The reason is that NFHS of 1999 collected information on immunisation and prenatal care only for children born in the three years preceding the survey. The resulting hazard functions are in the graph below.



The smaller sample of children from the 1999 survey (and its vicinity to 1999 in a period of decreasing infant mortality rate), has the effect of producing from the model, estimates of mortality rates that are smaller than those obtained by comparing two periods of five years. In the same way changes in mortality rates obtained from the Cox model appear to be larger respect to those of Table 2.

The procedure adopted to derive the changes in mortality rates attributable to each single variable for different groups of children (of different regions or different cohorts) is the following. The differences in the hazard function at time *t* between different groups of children are obtained by calculating the effect that a change in the value of each explanatory valuable has on the hazard function of a specific group. For example, if a and b are the names of two different groups of infants (surveyed in 1992 and 1998 respectively) we calculate the linear prediction of the group a using the mean value of *x*'s for this group. We then use this linear prediction to calculate the cumulative hazard function relative to group *a*:

$$\hat{H}_{a}(12) = e^{\hat{y}_{a}} H_{0}(12) \tag{5}$$

where:

$$\hat{y}_a = \beta_1 \overline{x}_{1a} + \beta_2 \overline{x}_{2a} + \dots + \beta_n \overline{x}_{na} \tag{6}$$

We then substitute in (6) the mean value of  $x_{1b}$  of group b for  $x_{1a}$  of group a:

$$\hat{y}_{a1} = \beta_1 \bar{x}_{1b} + \beta_2 \bar{x}_{2a} + ... + \beta_n \bar{x}_{na} \tag{7}$$

We then calculate the cumulative hazard function corresponding to the linear prediction  $\hat{Y}_{a1}$ :

$$\hat{H}_{a1}(12) = e^{\hat{y}_{a1}} H_0(12) \tag{8}$$

The value:  $(\hat{H}_{al} - \hat{H}_{a})/\hat{H}_{a}$ , is called the change in mortality attributable to the change in variable  $x_{l}$ . This quantity can be calculated for all the explanatory variables included in the model.

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