

# **Household Wealth and Child Health in India**

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### **Abstract**

This paper uses data from the Indian National Family Health Surveys (1992-93, 1998-99, 2005-06) to examine how the relationship between household wealth and child health evolved during a time of significant economic change in India. The main predictor is an innovative measure of household wealth that captures changes in wealth over time. Discrete time logistic models with community fixed effects are employed to examine mortality and malnutrition outcomes - neonatal, postneonatal, child, and under-five mortality; stunting, wasting, and underweight. Analysis is conducted at the national, urban/rural, and regional levels. Results indicate that the relationship between household wealth and mortality weakened while the relationship between household wealth and malnutrition became stronger over time. Findings suggest that the burden of improving child nutrition – but not child survival – is increasingly being borne by Indian households, possibly due to a weakening of complementary public inputs into child health.

## Introduction

Improving child health remains an important challenge in much of the developing world including India. India is home to almost three-quarters of South Asia's population under the age of five, and home to 20% of the world's under-five population (UNICEF, 2008 a). In the year 2000, India had more under-five deaths than any other country—over 2.4 million (Black, Morris *et al.*, 2003). More than 1 Indian child in every 18 dies within the first year of life, and more than 1 in every 13 dies before reaching age five. The under-five mortality rate (U5MR) was significantly higher in rural areas in 2006, at 82 deaths per 1,000 live births, with an urban U5MR of 52 deaths per 1,000. There are also considerable regional disparities within the country. Southern states, such as Kerala, have relatively low under-five mortality (16 deaths per 1,000 live births), while states in the Central region, such as Uttar Pradesh (96 deaths per 1,000), have the highest under-five mortality rates. Malnutrition is also extremely high. 45% of children are below the recommended height for their age, and 40% are below the recommended weight for their age (IIPS and Macro International, 2007).

These high mortality rates and malnutrition levels must not however deflect attention from the fact that India has made great progress in recent decades. The U5MR, which is generally considered an important development indicator, has been declining steadily. In the five-year period preceding India's 2005-06 National Family Health Survey (NFHS), the U5MR was 74 deaths per 1,000 live births, down from the 1998-99 estimate of 95 per 1,000, and the 1992-93 estimate of 109 per 1,000. Malnutrition also declined although declines were much smaller (IIPS and Macro International, 2007). These improvements occurred during a time of immense economic growth in India. But there is considerable debate on whether this economic growth benefited everyone, and whether it benefited everyone equally.

At the heart of this debate is whether the structural adjustment program of the early 1990s had desirable or undesirable consequences. The economic reforms initiated as part of this program emphasized globalization, liberalization, and privatization with its implicit retreat of the state. Some have argued that these new policies had an adverse effect on poverty reduction (Sen and Himanshu, 2004). Most scholars agree that there were increases in inequality at the national level, within and between states, and within and between rural and urban areas (Himanshu and Sen, 2005; Deaton and Dreze, 2002).

Given that child health is closely tied to economic welfare, these macro-level trends in child health and poverty prompt the question of what happened at the micro- or household-level. If the role of the state indeed diminished, were household factors increasingly responsible for producing improvements in child survival and nutritional status? Suppose that public spending directly or indirectly subsidizes the cost of a household procuring better health outcomes for its children. If it is true that India's recent economic growth was not pro-poor and involved a reduction in these subsidies to poorer people, then household income should have become more important over the years as a factor in improving child health. We thus take a look at how the role of household wealth as a determinant of child health has evolved between the mid-1980s and mid-2000s. The next section discusses the determinants of child survival, paying special attention to material determinants of survival.

### **Determinants of Child Health**

Historically, mortality reductions in various societies have been achieved through a combination of improved nutrition and economic growth, public health measures such as sanitation, clean water, and promotion of personal health practices, and medical innovations including vaccination, antibiotics, and intensive personalized interventions. Under optimal conditions, over 97% of newborns can be expected

to survive until at least age five. Decreases in this survival probability are generally due to social, economic, biological, and environmental forces (Mosley and Chen, 1984).

In their influential essay, Mosley and Chen (1984) proposed a comprehensive analytical framework for studying the determinants of child survival in low-income settings. Their emphasis was on integrating the usually distinct approaches taken by social scientists and medical scientists to the study of child health. The framework itself is based on the idea that all social and economic determinants of child morbidity and mortality necessarily operate through a set of proximate determinants, which in turn influence the risk of disease and the outcome of disease processes. Mosley and Chen also stress the importance of growth faltering measures. Body dimensions are not just reflective of the current health of individuals and populations but they can also be highly predictive of survival and future health status. Anthropometry is now frequently used in large demographic surveys thanks in part to its relatively inexpensive, non-invasive nature (Cogill 2003). In my paper, to the extent that the data permit, we include a number of proximate and socioeconomic determinants in my model of mortality and malnutrition among children, with the primary focus on household income, which is an important socioeconomic determinant in Mosley and Chen's model.

Child survival is more sensitive than most other health outcomes to the effects of material deprivation (Marmot, 2005) as is nutritional status. The leading causes of under-five deaths in poor countries include neonatal disorders (preterm births characterized by low birthweight, asphyxia, and tetanus), pneumonia, diarrhea, malaria, and measles. Malnutrition is an underlying cause of nearly half of these child deaths (Black, Morris *et al.*, 2003). The solutions to these causes of death are known. Adequate maternal nutrition and treatment of infections during pregnancy improves birthweight and reduce chances of

asphyxia (Tucker and McGuire, 2004). Skilled attendance at delivery decreases risks of asphyxia and also of infection during the birthing process. Administering the tetanus toxoid vaccine to pregnant women prevents tetanus infections. Vaccines and antibiotics in combination with better nutrition can eliminate most cases of pneumonia (UNICEF, 2008 b; WHO/UNICEF, 2009). Safe water and sanitation and simple rehydration therapy can prevent diarrheal deaths (PATH, 2009). Insecticide-treated bed nets and malaria prophylaxis can significantly reduce child deaths from malaria (UNICEF, 2004). Measles deaths can easily be prevented with a routine measles vaccine (WHO/UNICEF, 2009). And finally, adequate dietary intake can reduce malnutrition which in turn can reduce mortality (UNICEF, 1998).

A major constraint on implementing these solutions is the shortage of resources at multiple levels. Mosley and Chen (1984) identify a macro-level variable, the political economy, as an important determinant of child health. Political economy refers to the mode of production and distribution of benefits, the physical infrastructure, and the political institutions all of which exert an autonomous influence on child survival through proximate determinants. But these factors also affect the micro-level socioeconomic determinants of child survival. India has experienced dramatic macroeconomic changes in recent decades, accompanied by microeconomic changes in the welfare of households, all of which serve as the backdrop for the analyses in this paper. These economic changes are discussed in the next section.

### **Economic Change in India**

India's impressive economic growth over the past three decades has inspired dramatic pronouncements such as "The Indian giant is rising like Gulliver after being released from the web of threads with which he had been pinned down" (Rothermund, 2008). A series of post-independence economic policies and

developments preceded this impressive growth. These developments have implications for our understanding of trends in the determinants of child survival over the past two to three decades. When India became independent in 1947, the socialist vision of the leadership meant that the state would play a significant role in the economy. The 1950s consequently were marked by an emphasis on state investment in heavy industry such as iron and steel manufacturing, and large infrastructural projects such as dams. The Green Revolution in agriculture in the 1960s jumpstarted a decline in poverty that continued into the 1970s. As an agricultural revolution, it obviously benefited rural areas but its benefits also extended to urban areas through its effects on food prices and wages (Banerjee, Benabou *et al.*, 2006).

The period that our analysis covers starts in the mid-1980s saw the beginnings of deregulation and increased domestic demand, accompanied by the expansionary fiscal stance of the government. Together these led to a breakthrough in GDP growth and were paralleled by continued declines in poverty. While some have claimed that India's economic growth in the 1980s left the poor behind and also brought an increase in overall inequality, stronger evidence suggests that the macroeconomic growth did benefit people far below and even near the poverty line, and that growth was not linked to any noticeable increases in inequality (Datt and Ravallion, 2002).

Growth in GDP continued into the 1990s but the decade started with a fiscal crisis that necessitated a series of reforms. These included fiscal consolidation and stabilization, tax reforms, agricultural sector reforms, and policy changes related to industry, foreign investment, trade and exchange rates, and the public and financial sectors (Jha, 2004). What happened to poverty and inequality in the 1990s following these economic reforms is less clear than what happened in the 1980s. Some have argued that

poverty declined much more rapidly in the 1990s than before (Bhalla, 2000). Others argue that poverty levels continued to decline at the previous decade's pace (Datt and Ravallion, 2002). Their calculations suggest that the number of poor decreased by almost 30 million between 1993-94 and 1999-2000 (Deaton and Dreze, 2002). Still others have argued that poverty decline actually stalled and that the absolute number of poor remained steady, or even increased slightly in the 1990s (Himanshu and Sen, 2004). While the question of poverty decline has been contentious, there is more consensus on inequality in the 1990s (for a detailed discussion, see Pal and Ghosh, 2007). Several scholars agree that inequality increased along several dimensions—at the national level, between states, within states, within rural areas, within urban areas, between rural and urban areas, and so forth (Himanshu and Sen, 2005; Deaton and Dreze, 2002).

### **The Role of the State**

The fact that the increases in economic inequality followed the introduction of economic reforms that emphasized privatization, liberalization, and globalization directs our attention to the Coburn thesis (Coburn, 2000). Coburn urges scholars to look beyond the relationship between socioeconomic status (SES) and health, and beyond health inequalities, to examine the causes of SES inequalities themselves. His explanation for SES inequalities centers on the rise of neo-liberalism and its undermining of the welfare state. The basic assumptions of neo-liberalism are that markets are the best and most efficient allocators of resources, that societies are composed of autonomous individuals motivated chiefly by economic considerations, and that competition is the major market vehicle for innovation. Arguably, India adopted this philosophy (at least partially) starting in the mid-1980s.



The rise of neo-liberalism is also historically tied to the decline of the welfare state, which has the ability to correct inequalities generated by the market, through labor market policies, social welfare measures, and the decommodification of education and health, for example (Coburn, 2000). In India, the role of the state did in fact diminish following the reforms. Public investment as a percentage of GDP was much lower in the 1990s compared with the 1980s, as public investment did not keep pace with GDP growth (D'Souza, 2007). Also, the annual growth rate of total government spending was lower in the 1990s compared to the 1980s (Murty and Soumya, 2009). Public spending on health as a percentage of GDP also declined – from being 1.05% in 1985-86 to just .90% by 2000-01. Transfers from the Central government to states as a proportion of the total budget of the Ministry of Health and Family Welfare declined sharply, from about 57% to 44%. Now health is constitutionally a state subject in India and is thus financed primarily by the state governments. By 1999-2000, budgetary allocations by states to the health sector were 20% lower than in 1985-86 (National Commission on Macroeconomics and Health, 2005).

Such decline in spending can have serious consequences as the state has an important role to play in improving child health in many ways—for example, by ensuring food security, providing physical infrastructure, implementing disease-control measures, and offering subsidies (Mosley and Chen, 1984). Specifically, public programs can affect health outcomes in three ways (Schultz, 1984):

1. They can reduce the price of health inputs, either directly by subsidizing goods and services, or indirectly by increasing access to them, thereby reducing the costs and time involved in using the services.
2. They can provide information on how to produce health more efficiently. This might include information on new inputs or best practices with traditional inputs—for example, how long to

breastfeed or how to sterilize baby formula—that yield better health outcomes for a given expenditure.

3. They can alter the health environment, without directly affecting other opportunities available to people. Malaria control and smallpox eradication are often cited as examples. Such exogenously introduced changes in health conditions or technology affect everyone living in the area, regardless of their economic status or educational attainment.

Public goods thus complement household investments in child health. As an example, consider health expenditures. One important element of child health is health care, both preventive and curative. Generally, governments and households jointly bear the costs of health care. In 2001-02, the Government of India commissioned a systematic study of health spending, producing the first national health accounts for India. The results indicated that 65-75% of total health spending (including both adults and children) came from households, almost three times the amount spent at all levels of government (Economic Research Foundation 2006). This shows an inordinately high burden on households. In developed countries government health expenditures usually far exceed private spending, while in developing countries the ratio of public to private health spending is about 2:1. India's health care spending pattern, therefore, is truly an exceptional case among low-income countries as well as developed countries.

So far we discussed the determinants of child health including the political economy, recent economic changes in India, accompanying changes in the role of the Indian state, and the importance of the state to child health. This discussion leads us to the key hypothesis of this paper.

## Research Question

We know that child survival and nutrition in India on average have improved over time. If these improvements occurred despite the apparent retreat of the Indian welfare state, it is possible that there were (more than) compensatory changes in the role of household-level factors. We thus examine changes over time in the role that household wealth<sup>1</sup>, an important household-level factor, has played in influencing child survival and nutrition. The primary hypothesis is that household wealth became more important as a determinant of child health from one survey to the next. Urban/rural and regional disparities in levels and trends in economic development as well as in child health lead us to investigate this hypothesis at these various geographic levels as well. If household wealth has indeed become a more important determinant, this would imply an increase in inequalities in child health - suggesting missed opportunities for public intervention that could have improved equity.

## Past Work

There is an abundance of studies documenting the positive relationship between wealth and child health *net* of other direct determinants of health. Many of these are ecological studies that examine the association between average wealth and health. In one of the most widely-cited developing country studies on this topic, Pritchett and Summers (1996) conclude that wealthier is in fact healthier. They estimate the effect of income per capita on the infant mortality rate (IMR) and under-5 mortality rate (U5MR) using cross-national, time-series data. Using determinants of income growth exogenous to health as instrumental variables, they isolate the effect of income on health, net of education and free from reverse causation or unobserved heterogeneity. They find strong evidence to indicate that there exists a causal, structural relationship going from income to mortality in developing countries (Pritchett

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<sup>1</sup> While ideally we would use household income as a predictor, it is difficult to measure accurately and consequently not measured in most demographic surveys in developing countries. See Rutstein and Johnson (2004) for an excellent discussion.

and Summer, 1996). In a more recent cross-national study, using DHS data from over 60 countries including India, Wang et al (2003) investigate the determinants of child mortality both at the national level, and separately for urban and rural areas. At the national level, the regression results identify GDP per capita as an important determinant of the IMR as well as the U5MR. They also use information on assets (which is what we use) to predict their outcomes. They find that only in urban areas, a country's score on an index for possession of durable goods (for all households) is a statistically significant predictor of IMR (Wang et al 2003).

Simultaneously, there is an interest in documenting the association of wealth with health in low-income countries at the individual level. Casterline et al (1989) provide an excellent review of the classic studies on this topic (Casterline, Cooksey et al. 1989). In a more recent study, authors use DHS cross-sections collected in 42 developing countries including India to conduct a multilevel, multivariate analysis of child health. Their results show that household wealth is positively associated with height-for-age and weight-for-age (Boyle, Racine et al. 2006).

My analysis contributes to this literature on the relationship between household-level economic factors and child health in several ways. Due in part to a lack of data, there are relatively few developing country studies examining the impacts of household income/wealth net of other determinants. Even fewer of these studies are set in India. The handful of studies that do exist are dated and cannot tell us about the wealth-health relationship in recent years of rapid transition (see (Claeson, Bos et al. 2000) for a review of the older Indian studies). Further, the innovative wealth measure created here enables us to examine changes in the wealth-child health relationship over time. The 'absolute' measure of wealth moves us beyond studying disparities within a given year based on relative ranking of households.

Finally, examining both mortality (at different ages) and malnutrition outcomes leads to some key insights.

## **Data**

Data for this analysis come from the Demographic and Health Survey (DHS) series known as the National Family Health Surveys (NFHS) in India. The NFH surveys have become an important source of data on India's population, health, and nutrition at the national and state levels (IIPS and Macro International, 2007). To obtain reliable estimates at the state level, a stratified random sampling design was used within each state. In rural areas the sample was selected in two stages, where primary sampling units (PSU) or villages were selected first with probability proportional to population size (PPS), followed by selection with equal probability of households within PSU. In urban areas the sample was selected in three stages. First, wards were selected with PPS, followed by selection of one census enumeration block (CEB) within each ward, again with PPS. Finally, households were randomly selected within each selected CEB<sup>2</sup>.

One of the fundamental aims of these surveys was to obtain reliable estimates of the parameters of interest at various geographic levels (states, urban/rural, metropolitan cities), so target sample sizes were determined based on the lowest level of aggregation at which estimates were needed. This meant that ultimately the national sample size was unusually large by survey standards. In 1992-93, interviews were conducted with a 99% nationally representative sample of 89,777 ever-married women age 13-49 living in 88,562 households, in 24 states and Delhi (then a union territory). In 1998-99, the survey covered a nationally representative sample of 89,199 ever-married women age 15-49 living in 91,196

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<sup>2</sup> For more details on sample selection and information on the different levels of stratification, see the final reports for each of the surveys, available at [www.measuredhs.com](http://www.measuredhs.com).

households, in 26 states. In 2005-06, interviews were conducted with 124,385 women age 15-49 living in 109,041 households, in the 29 states of India (<http://www.nfhsindia.org>).

Four survey instruments were used: a woman's questionnaire, a men's questionnaire, a household questionnaire, and in rural areas a village questionnaire. Relevant to this study are the data tabulated from responses to the women's and household questionnaires. Women who were usual residents of the selected household or visitors who stayed in the selected household the night before the survey are interviewed using the woman's questionnaire. Information on children comes from the complete birth history recorded for all interviewed women. Information on assets and housing characteristics that form the basis of the wealth measure comes from the household questionnaire, which can be answered by any adult resident of the household.

All births to women that occurred in the 5 years preceding the survey (except births in the month of the interview) are eligible for the mortality analysis. For the malnutrition analysis, we have to consider that in NFHS-1, only children (of interviewed women) under four years of age were measured, and in NFHS-2, only children (of interviewed women) born in the three years preceding the survey were measured. In contrast, in NFHS-3 all children listed in the household roster that were under five years of age were eligible for measurement. To minimize bias from changes in sample selection, for analyses of anthropometric outcomes, we retain children who meet the most restrictive criterion i.e. children of interviewed women who are under three years of age at the time of survey (International Institute for Population Sciences and Macro International 2007).

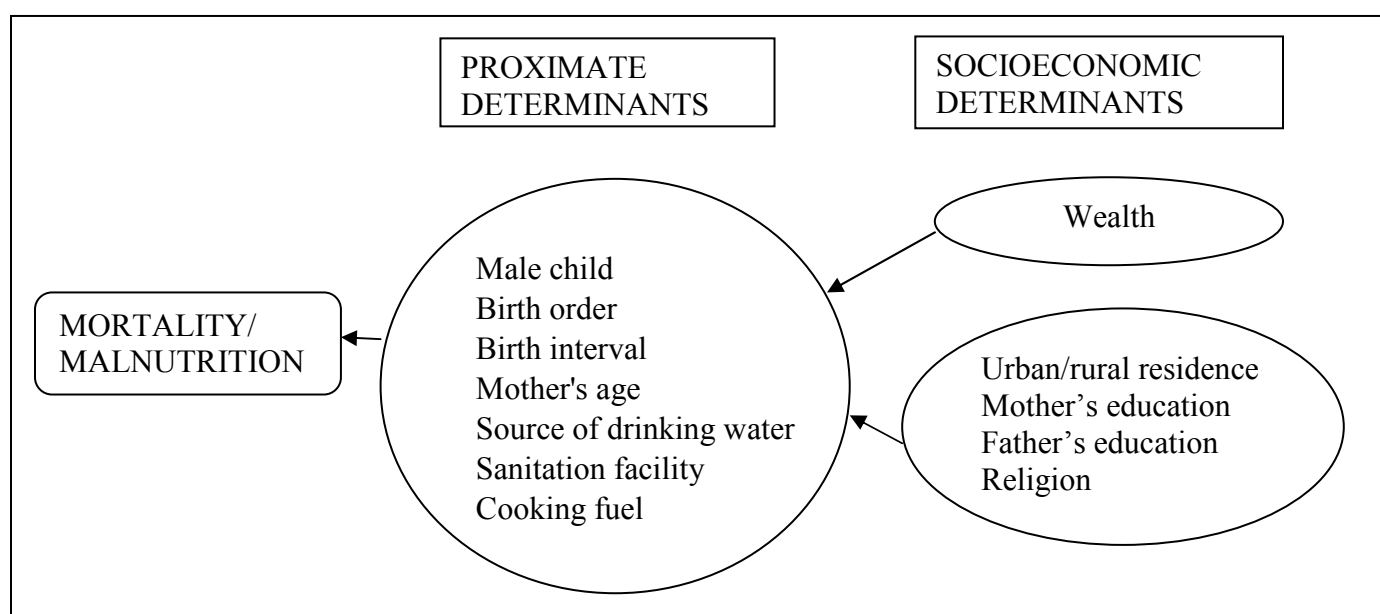
From all three surveys combined, 1.1% of births were dropped due to missing values on at least one of the covariates (1,871 out of 167,458 total births in the past 5 years) for a final unweighted sample of 165,587 births for the mortality analysis and 91,650 children for the malnutrition analysis.

## Measures

The various dependent and independent measures used in this analysis are described in this section.

Figure 1 lays out the conceptual model.

*Figure 1: Conceptual Model of Determinants of Child Health*



The *dependent variables* in the model start with a set of mortality outcomes -

- (i) Neonatal mortality – Deaths at age 0-30 days (from birth till before completed age 1 month)
- (ii) Postneonatal mortality – Deaths at age 1-11 months (from completed age 1 month till before completed age 12 months)

- (iii) Child mortality – Deaths at age 1–4 years (from completed age 1 year till before completed age 60 months).
- (iv) Under-five mortality – Deaths under age 5 years (from birth till completed age 60 months)

A second set of outcomes involves anthropometric measures -

- (i) Stunting – Defined as low height-for-age, stunting reflects shortfalls in achieved linear growth, and indicates long-term, cumulative inadequacies in health or nutrition.
- (ii) Underweight – Defined as low weight-for-age, underweight reflects low body mass relative to chronological age, and usually indicates long-term health and nutritional experience of an individual or population, similar to stunting
- (iii) Wasting – Defined as low weight-for-height, wasting reflects low body weight relative to height, and indicates a recent and severe process that that has led to significant weight loss, usually as a consequence of acute starvation and/or severe disease. (World Health Organization 1995).

Each child's anthropometric indicators are expressed in standard deviation units from the median of the reference population. The reference population used in this current analysis is the one adopted in the latest NFHS, newly prescribed by the WHO (World Health Organization Multicentre Growth Reference Study Group 2006). The standard is based on measurements of children around the world that are raised in healthy environments, with non-smoking mothers, are exclusively breastfed for the first 6 months, and given appropriate complementary foods from 6 to 23 months (International Institute for Population Sciences and Macro International 2007). Generally, children more than two standard deviations below the respective reference medians are considered stunted, wasted, or underweight. In this paper, we



present results for *severe* stunting, wasting, and underweight i.e. children that are more than three standard deviations below the reference median.

The *socioeconomic determinants* included in the model are:

1. Household wealth, as measured by a principal components approach to data on multiple household assets and characteristics – the dwelling’s construction material, i.e. a *kachha* house made of mud, thatch, or other low-quality material, a semi-*pucca* house made of partly low-quality and partly high-quality materials, or a *pucca* house made of high-quality materials throughout, including the floor, roof, and exterior walls (IIPS and Macro International, 2007); source of drinking water; sanitation facility; cooking fuel; and whether the household owns a fan, radio, television, sewing machine, refrigerator, clock, bicycle, motorcycle, and car.
2. Whether the household is in an urban or rural area; level of mother’s and father’s schooling (education in single years classified as no schooling if 0 years, primary if 1 to 6 years, secondary if 7 to 12 years, post-secondary if greater than 12 years); and religion (whether the household head is Hindu, Muslim, or other).

The *proximate determinants* included in the model are:

1. Sex of the child; birth order of the child; the interval between the previous birth and the index child’s birth in months (marriage to birth interval in the case of first births); and mother’s age in years at the time the index child was born.
2. Whether the household uses an improved source of drinking water - where an improved source is defined as private or public piped water, tube well, or borehole (WHO/UNICEF, 2010); whether the

household uses improved sanitation - for comparability across surveys, an improved sanitary facility is limited to being defined as one with a flush toilet; and whether a solid cooking fuel is used - where solid fuels are defined as wood, coal, dung, agricultural residues, or shrubs/straw.

Results are presented at the national, urban/rural, and regional levels. The regional classification of states is as follows:

1. North – Delhi, Haryana, Himachal Pradesh, Jammu & Kashmir, Punjab, Rajasthan, Uttarakhand
2. Central – Chhattisgarh, Madhya Pradesh, Uttar Pradesh
3. East – Bihar, Jharkhand, Orissa, West Bengal
4. Northeast – Arunachal Pradesh, Assam, Manipur, Meghalaya, Mizoram, Nagaland, Sikkim, Tripura
5. West – Goa, Gujarat, Maharashtra
6. South – Andhra Pradesh, Karnataka, Kerala, Tamil Nadu

## **Methods**

### *Wealth scores*

First, to generate the principal socioeconomic measure of interest, household wealth, we use data on household assets and housing characteristics to conduct principal components analysis (PCA). PCA is a multivariable statistical technique to transform information from several variables into a smaller number of ‘dimensions’. The idea is that an underlying variable, in our case wealth, can be predicted by the observed data on assets and housing. PCA gives us a way to generate weights or coefficients on those observed data. From an initial set of  $n$  correlated variables, PCA creates  $m$  uncorrelated components  $PC$ ,

where each component is a linear weighted combination of the initial variables  $X$  (Rutstein and Johnson, 2004; Vyas and Kumaranayake, 2006).

$$\begin{aligned} PC_1 &= b_{11}X_1 + b_{12}X_2 + \cdots + b_{1n}X_n \\ &\dots \\ PC_m &= b_{m1}X_1 + b_{m2}X_2 + \cdots + b_{mn}X_n \end{aligned}$$

An eigenvector is a vector such that, when a matrix is multiplied by that vector, the result can be written as a vector multiplied by a scalar. That scalar is the eigenvalue of the eigenvector. The eigenvectors of each of the  $m$  correlation matrices above give us the weights or factor score ( $b$ ) for each principal component. The eigenvalue of each vector gives us the amount of the total variance in our variables that each principal component explains. The first component has the largest eigenvalue and explains the most variation in the data. It is unrelated to the second component, which explains additional variation, and so on until the  $m$ th component. The more highly correlated the assets, the fewer components required to explain variation in the latent wealth variable. Using just the first component is usually adequate to capture economic status as represented by asset information. The marginal gain from including higher-order components is small and in addition presents difficulties of interpretation (Houweling *et al.*, 2003; McKenzie, 2003; Filmer and Pritchett, 2001).

Since we are interested in changes in the effect of household socioeconomic status on child health over time, we need a metric to value the wealth of households over time, without the content of that metric changing. The usual wealth quintiles employed in studies using DHS data only tell us about the gap between the richer and poorer households. In my trend analysis, however, we need to take into account the possibility that the poorest households in one year may be better or worse off than the poorest households in the next survey. If we used the standard relative wealth index, we would only know the

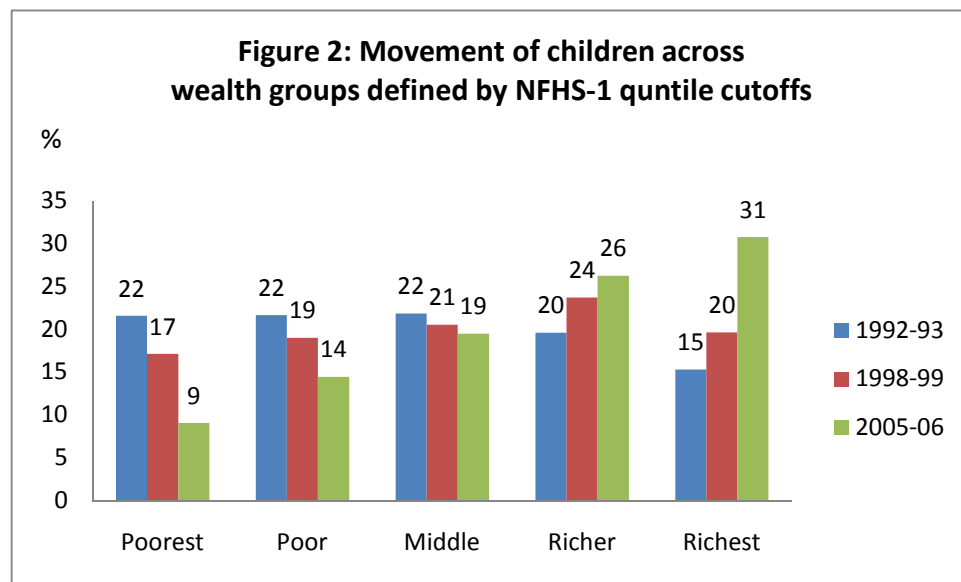
effect of being wealthier with reference to the poorest household in a single survey year, rather than relative to the poorest household in all three survey years (1992-93, 1998-99, and 2005-06). The procedure we employ to produce an absolute measure of wealth (as opposed to a relative measure) is as follows:

- (i) we start with the matrix of information on ownership of 13 assets/housing characteristics in the 1992-93 data (the 13 that are common to all three surveys – electricity, drinking water source, sanitation facility, home’s building material, type of cooking fuel, fan, radio, television, sewing machine, refrigerator, clock, bicycle, motorcycle, car).
- (ii) we then find the eigenvector of factor scores associated with the first principal component of wealth in the 1992-93 data.
- (iii) Next, we multiply the factor score ( $b$ ) for each asset by the indicator variable indicating whether or not each household in the 1992-93 data owns that asset.
- (iv) we sum the factor scores to yield a wealth score for every household in the 1992-93 survey.
- (v) we rank the households on this score and then divide them into five quintiles at 20% cutoffs<sup>3</sup>.
- (vi) we then use the same eigenvector of factor scores from the 1992-93 survey to generate a continuous wealth score for households in the next two surveys.
- (vii) The same values of the wealth score that form the quintile cutoffs in the 1992-93 survey are used to divide the households in these other surveys into five groups each.

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<sup>3</sup> The official quintiles included in the publicly available data are based on household *population*, not households, because most analyses are concerned with poor people, not poor households. We think our analysis is more related to households, and thus we create quintiles of households. Although our quintiles do not align perfectly with the official ones, our wealth score for each household is highly correlated with the official score - close to a 99% correspondence.

These groups in later surveys would no longer necessarily contain 20% of the households if there were improvements or declines in levels of household wealth. For example, improvements in wealth would lead to movement of households from lower groups to higher ones, thus diminishing the size of lower wealth groups and increasing the size of higher wealth groups. Indeed, this turns out to be true. Figure 2 shows that, over the course of the three surveys, the bottom three groups shrunk and the top two increased in size. There is a distinct pattern in changes in the size of groups. The biggest decline is in the poorest group, and the decline becomes progressively smaller over the next two groups. The groups increase in size starting with the fourth quintile, but the biggest gain is in the top quintile. Given that the wealth score is based on a limited set of fairly basic items, in one sense this tells us that many more households in India now have access to basic necessities.



#### *Discrete time logistic model*

A simple logistic regression is use for the malnutrition outcomes. The mortality analyses however are performed in an event-history framework that extends the proportional hazards model to discrete time.

While the survival process is a continuous one and death can occur at any time in a child's life, our data are discrete in nature. For example, in the NFHS questionnaire information on age at death for deaths at and after age 2 is recorded in years. This results in deaths grouped at discrete intervals, thus calling for a model more suited to discrete-time processes. In such a model, the entire length of time that each child is observed is divided into smaller intervals, and the dependent variable is recorded as a series of binary outcomes denoting whether or not the death occurred in that time interval.

Consider first  $T$ , a discrete random variable indicating the time of death. The probability mass function for  $T$  can be written as

$$f(t) = \Pr(T = t_i)$$

and represents the probability of a death occurring at time  $t_i$ . The survivor function of  $T$  can be written as

$$S(t) = \Pr(T \geq t_i)$$

denoting the probability that survival time  $T$  is equal to or greater than some time  $t_i$ . Relating the concepts of failure and survival is the hazard rate or the risk of death

$$h(t) = \frac{f(t)}{S(t)}$$

which is a ratio of the probability of failure to the probability of survival. Thus in the discrete time method, the hazard is not a rate but a conditional probability. In other words, the hazard function gives us the probability of death *conditional* on survival up to the start of a time interval.

$$h(t) = \Pr(T = t_i | T \geq t_i)$$

Now the probability of death can be made conditional on a set of covariates, just as it is conditional on survival.

$$h(t|\mathbf{x}) = \Pr(T = t_i | T \geq t_i, \mathbf{x})$$

Since the dependent variable takes a binary form, one could use the logistic distribution to relate the dependent variable to the covariates (Box-Steffensmeier and Jones, 2004). The logit function can then be written as

$$\log \left( \frac{h(t | \mathbf{x})}{1 - h(t | \mathbf{x})} \right) = \beta_0 + \beta_1 x_{1i} + \dots + \beta_k x_{ki}$$

which is the ratio of the probability of occurrence of death to the probability of nonoccurrence. The logit coefficients  $\beta_k$  represent the relationship of each covariate to the log-odds of death. Exponentiating these coefficients will thus yield the increase or decrease in the odds of death associated with a change in the respective covariate.

The logit discrete-time model makes the assumption that the baseline hazard is flat, whereas in all likelihood the hazard probability changes with age. In our data where each child contributes multiple records (because we split their lives into episodes), there generally is some type of temporal dependence between the repeated measures on the  $i$ th child. To account for this duration dependence, we include temporal dummy variables in the regression. Following a visual analysis of the pattern of the baseline hazard, we also included a second-order duration term to better model its shape.

Survival analysis presents the distinct advantage of allowing censored observations to contribute to analysis time, thus maximizing the use of available data. Children that die contribute information regarding the probability of failure,  $f(t)$ , and those that survive through the entire relevant age range or are censored by the survey date contribute information regarding the probability of survival,  $S(t)$  (Box-Steffensmeier and Jones, 2004). Observation time is divided into seven-day intervals for the neonatal period (0-30 days), one-month intervals for the postneonatal period (1-11 months), and three-month intervals for child (1-4 years) and under-five mortality. Consequently, a child who exits at age 3 years

and 2 months contributes 38 months of observation time to the analysis and is considered to have existed in the last three-month period, thus excluding the last two months from survival time.

### *Fixed effects logistic model*

The regression model as specified in *Figure 1* does not include any information about the larger contexts that the children live in, beyond the family and the household. For example, we know that features of communities can have an impact on child outcomes (Desai and Alva, 1998; Kravdal, 2004).

Characteristics of these communities that are related to household wealth levels but are also related to child health will be captured in the error term, thus violating the fundamental regression assumption that the independent variables in the model and the error term are uncorrelated (Greene 2003). In order to address this problem of unobserved group-level heterogeneity, we use a fixed effects model. we include fixed effects at the community or primary sampling unit (PSU) level. The fixed effects models essentially contain a dummy variable for each community, yielding a vector of community-specific parameters. These parameters capture omitted variables that are common to the group, and give a concrete representation to community background. In contrast to the group-specific ‘nuisance’ parameters, the effects of interest here, the effect of household wealth on child health (and the effects of other determinants) are represented by a parameter vector common to all communities. A regression function that conditions on group membership will thus estimate the latter parameter vector more accurately by reducing omitted variable bias (Chamberlain 1980).



Since we do not have longitudinal data but repeated cross-sections, this is not a panel fixed effects where there are individuals on whom we have repeated observations over time<sup>4</sup>. Instead, there are groups – communities – in which we have several observations. The analogy to a panel of individuals is as follows. The  $k = 1, \dots, K$  communities are the ‘individuals’ and the  $i = 1, \dots, N$  births within each community are the ‘time periods. When working with a limited dependent variable model (child died or not, child stunted or not), we use a fixed effects binary logit model which can be written as

$$\text{Prob}(y_{ik} = 1 | x_{ik}) = \frac{e^{\alpha_k + x'_{ik}\beta}}{1 + e^{\alpha_k + x'_{ik}\beta}}$$

Traditional likelihood maximization breaks down in a fixed effects model with a qualitative dependent variable and the intercept for some groups is not estimable under certain conditions (Greene 2003). The inconsistency of the maximum likelihood estimate of the intercept is transmitted into inconsistency of the estimates of the parameters of interest. One workaround to this is to maximize the conditional likelihood function as demonstrated by Chamberlain (1980). This requires us to select the maximum possible number of pairs of births within a community and then discard all pairs where both births have had the same outcome. What remains are pairs of births where one child survived and one died, or one child is stunted and the other is not. The conditional probability for any remaining pair of births  $i = 1$  and  $i = 2$  in group  $k$  is

$$\frac{\frac{e^{\alpha_k + x'_{1k}\beta}}{1 + e^{\alpha_k + x'_{1k}\beta}} \frac{1}{1 + e^{\alpha_k + x'_{2k}\beta}}}{\frac{e^{\alpha_k + x'_{1k}\beta}}{1 + e^{\alpha_k + x'_{1k}\beta}} \frac{1}{1 + e^{\alpha_k + x'_{2k}\beta}} + \frac{1}{1 + e^{\alpha_k + x'_{1k}\beta}} \frac{e^{\alpha_k + x'_{2k}\beta}}{1 + e^{\alpha_k + x'_{2k}\beta}}} = \frac{e^{x'_{1k}\beta}}{e^{x'_{1k}\beta} + e^{x'_{2k}\beta}}$$

And similarly,

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<sup>4</sup> There is data on the same states in the three surveys but since we are not analyzing aggregated state-level data, we do not use state panel fixed effects model. As for clusters or PSU's, they change from survey to survey so aggregation and a panel at that level would not be possible.

$$\frac{e^{x'_{2k}\beta}}{e^{x'_{1k}\beta} + e^{x'_{2k}\beta}}$$

Note that conditioning the child's outcome on the sum of the two children in the pair removes the unobserved group-level heterogeneity captured by the  $\alpha$ 's. The  $e^{\alpha_k}$  term in the numerator and denominator cancel each other out and the group intercept drops out of the equation.

### *Clustering*

we use an estimator of the variance that is robust to the fact that cluster samples, unlike simple random samples, violate the assumption of independence of observations. Households are not drawn randomly from a listing of all households in the country. Rather, households are chosen randomly *within* a cluster, thus violating the assumption of independence of observations and leading to incorrect estimates of the standard errors (Rogers, 1993). This in turn leads to incorrect inferences about the significance of the coefficients attached to the independent variables in the regression model. Parameter estimates are not affected by this violation. The robust variance estimator is specified using the `vce (cluster)` option in Stata 11. Cluster membership is specified using the cluster ID as the group-membership variable. Note that there are additional layers of clustering—of children within women, and of women within households. For example, in the pooled mortality sample we have a total of 165,587 births from 116,034 women residing in 107,329 households. The average number of children per woman is 1.43, and per household is 1.37. Including all children of all interviewed women from all households in our sample poses the same problem as clustering of households within primary sampling units. However,

accounting for clustering of children and women shows results not much different from results of analyses accounting for clustering of households<sup>5</sup>.

### *Weighting*

The principal component analyses are conducted using household data and are consequently weighted using household weights. The woman weights provided in the NFHS are generally normalized weights, i.e. the sampling weight is multiplied by the sampling fraction. When data are pooled, these weights are no longer correct and must be de-normalized. Since the woman files from all three NFH surveys are pooled for the multivariable analysis, weights need to be adjusted. The factor by which weights are multiplied is a product of (i) the proportion of women in the pooled sample that are from that survey<sup>6</sup>, and (ii) the ratio of the total number of women in the pooled sample to the sum of the mid-year populations of women age 15-49 in 1992, 1998, and 2005 (population data accessed from the International Data Base of the U.S. Census Bureau).

## **Results**

### *Sample description*

Table 1A shows at the national level, mortality rates, malnutrition levels, and means of the various determinants of child health that are included as control variables in our model. Table 1B shows these numbers at the regional level. The means are based on the most inclusive sample – the neonatal mortality sample. As expected, we see that rural areas show higher mortality and malnutrition than urban areas. Rural households are far less likely than urban households to have an improved sanitation facility and far more likely to use a solid fuel for cooking. Rural women and men are also much more

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<sup>5</sup> It may be that accounting for clustering at higher levels automatically considers clustering at lower levels, at least partially.

<sup>6</sup> This is calculated separately for each survey and applies only to women from that particular survey.

likely than urban women and men to have had no education. Both rural and urban areas however, had increases over time in the proportion of households with improved drinking water and sanitation, as well as in education levels of both fathers and mothers. The Central, Eastern, and Northeastern regions are dominantly rural (as seen by the proportion of households urban) and have higher mortality and malnutrition than other regions. These regions also have lower proportions of households with improved sanitation, higher proportions using a solid fuel, and higher proportions of women and men with no education.

TABLES 1A and 1B HERE

#### *Bivariate analysis*

Table 2 shows changes in mortality rates and malnutrition levels for the five wealth groups in each year only at the national level. This table tells us several things. First, within year, across wealth groups, there is a clear wealth gradient in health – poorer groups always have higher mortality and malnutrition than wealthier groups. Second, across year, within wealth groups, the bigger absolute declines in mortality from 1992 to 2005 occurred in the poorer groups. Of course, these groups did have higher mortality to begin with and thus had more potential for improvement. However, this pattern does not hold for malnutrition except at the very extremes, i.e. the wealthiest group did have bigger absolute declines than the poorest group. Looking at percentage changes, we see a clearer pattern for malnutrition than for mortality. Richer groups consistently had larger declines in stunting and wasting than poorer groups. The biggest relative declines in neonatal and under-5 mortality – with trends in the latter being strongly influenced by the former – also occurred in the poorer groups. However, when it came to mortality at older ages - postneonatal and child mortality - both the upper and lower quintiles had large declines

TABLE 2 HERE

To understand these patterns better, and understand changes at the urban/rural and regional levels, we move to a regression framework. The odds ratios in Table 4 can be interpreted as the effect of a standard deviation change in the wealth score. Remember that our wealth predictor is a continuous wealth score. Since a unit increase in this wealth score is somewhat difficult to interpret, a standardized version is used in the regressions (subtract mean and divide by standard deviation). Table 3 shows summary statistics (mean, standard deviation, minimum and maximum) for the unstandardized wealth scores at each level of geographic aggregation. At the national level, it ranges from -2.34 to 7.54 with a mean of .08 in 1992, .48 in 1998, and 1.34 in 2005.

TABLE 3 HERE

### *Multivariate analysis*

Table 4 shows results from logistic regressions at the national and regional levels. The odds ratios associated with wealth for all outcomes are generally below 1.0 corroborating what we know about children from wealthier households having lower odds of dying or being malnourished than children from poorer households. We start by examining results from the ‘simple’ regressions or the regressions without community fixed effects. Focusing first on *under-5 mortality*, we see that a one standard deviation increase in the wealth score results in a 23% decline in the odds of mortality in 1992, at the national level. By 2005, a one standard deviation increase led to only a 14% decline in the odds. Note that one standard deviation in the national wealth score is 2.58 which implies that around four standard

deviations would take us from the minimum to the maximum value of the national wealth score distribution. Comparing urban and rural areas, wealth effects on under-5 mortality are stronger in rural areas. At the regional level, coefficients are significant in all regions but strongest in the Western region. Wald tests of coefficients from later surveys against coefficients from earlier ones yield evidence of a trend of *weakening* wealth effects over time on under-5 mortality at most geographic levels, particularly in rural areas and the Eastern region (except in the North the effect stayed strong). Even in urban areas, the fact that the coefficient was no longer significant in the third survey does suggest a weakening trend in wealth effects.

We then unpack under-5 mortality to look at mortality in smaller age intervals. Wealth effects are much weaker for *neonatal mortality* than mortality at older ages. For neonatal mortality, although coefficients in the first two surveys were significant in rural areas, there are no significant wealth effects remaining by the third survey. Interestingly, in all three surveys, wealth does not seem to have mattered for neonatal mortality in urban areas. No regional pattern emerges. On the whole, we are left with a *weakening* trend in wealth effects on neonatal mortality at the national level. Of all the mortality outcomes, wealth effects are strongest for *child mortality*. A one standard deviation increase in wealth is associated with a 45% decline in the odds of child death. Wealth effects on child mortality are larger in urban areas than in rural areas (unlike in neonatal mortality). However, while the effect is no longer significant by the third survey in urban areas, it actually remains strong in rural areas, leaving us with a national pattern of *consistently strong* wealth effects. At the regional level, we see that the largest effects of wealth on child mortality are in the South and the West but, there is no pattern of strengthening wealth effects. However, there is no real weakening of effects either with coefficients remaining very large in size – for example, in the North, the coefficient goes from being .55 in the first survey to being

.57 in the last survey. Finally, wealth effects on *postneonatal mortality* tend to lie in between neonatal and child mortality in terms of size and significance of coefficients. Wealth only affects postneonatal mortality in rural areas (like in the case of neonatal mortality). Regional results are mixed, with no clear pattern.

Now turning our attention to *malnutrition*, we see that of the three outcomes, wealth is most strongly correlated with underweight followed by stunting. While these two outcomes show similar results, wealth effects on wasting are the weakest, and are only evident at the national level (including urban and rural groupings) with no consistent effects at the regional level. Wealth is more strongly associated with malnutrition in rural areas than in urban areas. All regions show consistently strong wealth effects both for stunting and underweight.

Looking for trends in the wealth effect on stunting and underweight we see that at the national level there is a *strengthening* of effects in later surveys (odds ratios moving further below 1.0). This is especially true of underweight. For example at the national level in 1992, a one standard deviation increase in wealth would have resulted in a 26% decline in the odds of a child being underweight. By 2005, a one standard deviation increase results in a 39% decline in the odds of being underweight. As stated earlier, India being dominantly rural, the national trend is generally dominated by the rural trend. However, both urban and rural areas show a strengthening (or emergence) of wealth effects from the first to the last survey. The Northern, Central, and Eastern regions, which showed strong wealth effects, were the three regions to have a strengthening of wealth effects, whereas in the Western and Southern regions while the wealth effects remain strong, they do not strengthen over time.

### *Community fixed effects models*

Parallel to the columns showing results from the “simple” logistic regressions are columns showing results from logistic regressions that include fixed effects terms for the community (or primary sampling unit) that the household belongs to. We again start with *mortality*, specifically under-5 mortality. Going from the simple model to the one with fixed effects, while the coefficient sizes are comparable, wealth is no longer significant in any of the surveys in urban areas or in the third survey in rural areas. At the regional level, adding fixed effects to the model renders almost all wealth effects for all mortality outcomes insignificant. In urban areas and at the regional level, there is thus no trend in wealth effects. In rural areas although Wald tests do not show a trend, significant coefficients in the first two surveys followed by a insignificant coefficient in the third survey result in a *weakening* trend in wealth effects on under-5 mortality at the national level. For neonatal and postneonatal mortality, when fixed effects are included, wealth does not seem to matter in any of the survey years at the national or regional level. However in the case of child mortality, in rural areas and thus at the national level, wealth effects stay strong even after the inclusion of fixed effects. Overall, as a quick glance at the mortality results at the national level will indicate, the results from the fixed effects model mirror the trends from the simple model. In other words, even after accounting for cluster-level unobserved heterogeneity, there was no evidence of a strengthening of wealth effects. In fact, notwithstanding the continuing importance of household wealth to child mortality, the importance of household wealth to overall mortality actually declined, driven largely by the declining trend in wealth effects on neonatal mortality.

The fixed effects regression results for *malnutrition* similarly mirror results from the simple regressions, at least for stunting and underweight. At the national level, wealth effects from both models are similar in significance. In urban areas, even though the Wald tests do not show that later effects are statistically



different from previous effects, later effects do emerge as significant (and further below 1.0) where they were not before. In rural areas, Wald tests show that later coefficients are statistically different from (and further below 1.0 than) previous coefficients. This amounts to a finding of *strengthening* wealth effects at the national level. Similarly, at the regional level, in every region except the Northeast, either the Wald test or emerging significance establish the pattern of strengthening wealth effects for at least stunting or underweight if not both.

TABLE 4 HERE

## **Discussion**

### *Effects on mortality*

Household wealth is an important predictor of mortality at all ages in all three surveys, but the associations are weakest for mortality at younger ages. This finding is consistent with the general literature. Variations in the causes of death at younger and older ages may explain the differences in the strength of wealth as a predictor of mortality. The weaker association of wealth with neonatal mortality than with child mortality can largely be ascribed to the effects of genetics, birth accidents, and other endogenous causes that are quite rare for mortality beyond the neonatal period. Older children on the other hand have greater exposure time such that socioeconomic factors like household wealth can affect child survival through a multitude of pathways. The fact that the wealth coefficients for postneonatal mortality lie in between the coefficients for neonatal and child mortality further validates this argument. Children in the postneonatal period (from 1 to 11 months) are young but no longer newborn. Thus both biological and socioeconomic factors strongly exert an influence on their survival.

The addition of community fixed effects to the models results in rural (and national) wealth coefficients for neonatal and postneonatal mortality no longer being significant. This suggests that there are important community-level factors excluded from the model that are correlated with household wealth and also affect mortality at younger ages. These unmeasured factors could be determinants like the availability of health services in the community that particularly impact younger infants, for example antenatal care and skilled delivery assistance. If the availability of these services is negatively correlated with mortality but positively correlated with household wealth, that would lead to what we saw in the simple model i.e. the household wealth effect was possibly standing in for a services effect.

Wealth is related to child mortality in both urban and rural areas at least in the simple model underscoring the importance of household-level socioeconomic determinants. When community fixed effects are added to the model, the effects are no longer significant in urban areas but persist in rural areas. Again this suggests there are community-level factors in operation that mask the similarities/differences between children from poorer and richer households when not accounted for. When they are in fact accounted for, it turns out that the differences between children of different family wealth backgrounds are less important than suggested by the simple model in urban areas, and more important in rural areas. Stated differently, family and community-level factors being equal (including sanitation, healthcare, and related services), household wealth does not make as much of a difference to child mortality in urban areas than was first evident and even more of a difference in rural areas. Villages generally have low levels of public health and healthcare services, and thus household wealth can make it easier for wealthier rural families to seek out hard-to-access infrastructure or services. For example, leading causes of child deaths include acute respiratory infections, and diarrhea both of which are influenced by the child's physical environment. Public provision of proper sanitation and water

supply, which is considered a neighborhood or community-level factor, is in fact particularly poor in rural areas (only 16% of households had an improved sanitation facility in 2005-06). Also vaccination coverage for example, which can prevent ARIs, is worse in rural areas.

### *Effects on malnutrition*

Nutritional status is very much a function of constant household-level inputs such as food and healthcare. Poor nutritional intake and repeated illnesses both have an effect on the nutritional status of a child, and the risk of both increases with exposure time to these factors. In the simple model, wealth was associated with malnutrition in both urban and rural areas, underscoring the widespread nature of the malnutrition problem in India (of children under the age of 5, 48% were moderately or severely stunted, and 43% were moderately or severely underweight in NFHS-3). When community fixed effects were added, the effects persisted in both urban and rural areas, very plausibly suggesting that malnutrition outcomes are more heavily dependent on household-level factors than on community-level factors.

Underweight was the outcome most strongly associated with household wealth. Weight-for-age as a measure presents the advantage of reflecting both past and current malnutrition. This means that it is more responsive to short-term changes in inputs to health, and current wealth status of the household. The correlation of household wealth with stunting was slightly weaker. Stunting unlike underweight does not change in the short term. It is an indicator of past growth failure, and is considered irreversible beyond the age of 2. Stunting may thus not exhibit as close a correlation with a current measure of household wealth as underweight.

### *Trends*

Our hypothesis was that the relationship between household wealth and child health would strengthen over time. We find that this is true in the case of malnutrition but not in the case of mortality. Wealth effects for under-5 mortality declined in both urban and rural areas, and this pattern is dominated by the weakening wealth effects on mortality at younger ages (since neonatal mortality is the largest component of under-5 mortality). As neonatal mortality rates decline, infants that die may be a somewhat more selective group, increasingly comprising those who are inherently more vulnerable and thus less affected by external factors like household wealth. While one could argue that this is true in the case of child mortality as well, the potentially greater importance of biological factors in neonatal mortality makes the selectivity argument more appropriate for neonatal mortality. The weakening trend in wealth effects suggests that mortality is not as dependent on private wealth as it was before, possibly due to the universally subsidizing effect of public programs aimed at reducing mortality among children, specifically the Universal Immunization Programme, the Child Survival and Safe Motherhood Programme, and the Reproductive and Child Health Programme. Despite declines in public investment in general, the success of these programs could have offset the investments that household might have had to make.

For malnutrition, there was a pattern of strengthening wealth effects in urban as well as rural areas. A plausible programmatic explanation emerges in the story of the public distribution system (PDS), which provides wheat, rice, and sugar at affordable prices to families to enhance food security. Improved food security can reduce malnutrition among children, and thus also lower under-five deaths. The PDS is India's most far-reaching safety net in terms of coverage and also the most expensive in terms of public expenditure (Radhakrishna and Subbarao 1997). In 1997, the government introduced the Targeted Public Distribution System (TPDS) with the aim of redistributing income by providing more of the

distributed food to the poor and at cheaper prices than to the non-poor. The move from a universal system to targeted system was motivated both by concerns of program efficacy as well as the liberalization imperative to shrink the role of the government – in this case in the food grains sector. Five years after the introduction of the TPDS, a high level committee reported that the move from PDS to the TPDS “may have served to blunt the efficacy of the PDS in meeting its original goal of price stabilization, while not delivering fully in terms of the new concern to focus subsidies to the poor. By excluding a large number of families, the TPDS undermined the viability of Fair Price shops and increased scope for distortion and leakage. It penalized states with relatively low incidence of income poverty but relatively high incidence of calorie deficiency. At the same time, it did not reach the poor in states where the PDS was weak prior to its introduction. We feel that it is essential to go back to a universal PDS” (Ministry of Consumer Affairs and Food & Public Distribution 2002). This acknowledgment of even greater failure (during years relevant to our analysis) – of a system that had already been widely criticized for its shortcomings – is in line with our finding of the increasingly important role of private wealth in child malnutrition.

### *Other factors*

One important question to ask in the discussion of our results would be: to what extent did changes in other important factors known to impact child survival and nutrition affect the results? For example, antenatal care usage, and the proportion of women giving birth in a health care facility or with assistance from a skilled attendant have increased. While these factors were not included in the multivariable models, this is not necessarily a limitation of this study. These factors can be conceived as being on the pathway from wealth to mortality. The changing effects of wealth may well be capturing changing usage

of antenatal and delivery care. And in fact we argue that policy can have an impact on variables such as these and thus reduce the burden on households (and their wealth) of purchasing these services.

### *Limitations*

This analysis has some limitations. The wealth index is generally only recommended for use as a ranking mechanism, not as an absolute measure. It is limited in its ability to measure multiple dimensions of household economic well-being, and trends in those dimensions. Also, it is more a test of long-term than short-term economic welfare. This matters if child survival and nutrition are more responsive to short-term measures of welfare such as household income. Additionally, although my hypothesis stems from an observation of economic growth and general indicators of a change in the role of the state, we did not conduct a direct test of the role of government and cannot render a verdict on the government's performance in terms of service provision. Changes in the relationship between household wealth and child health could have occurred due to factors other than changes in the effectiveness of the public sector.

### **Conclusion**

The numbers presented in this paper tell a compelling story. While mortality and malnutrition among children have both declined, household wealth still plays a crucial role in ensuring desirable outcomes. The right programs can improve child health while simultaneously decreasing the burden on households. Malnutrition in particular continues to be very high in India even when compared to other developing regions. We show indirect evidence for the inadequacy of programs aimed at reducing child malnutrition in India.

Extensions to this research include additional tests of the central hypothesis about changes in the wealth-health link. One could test for changes in the effects of household wealth on outcomes of immunization, antenatal care, and other child health care. These are important inputs into infant and child health, and they have potentially different relationships to household wealth. Another important extension is a decomposition analysis to partition the sources of change in mortality and malnutrition into changes in the levels of the determinants versus changes in the effects, in order to identify the relative importance of the sources of change.

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## Tables

**Table 1A: Mortality rates, malnutrition levels, and means of determinants (proportions unless otherwise indicated)**

	National			Urban			Rural		
	1992-93	1998-99	2005-06	1992-93	1998-99	2005-06	1992-93	1998-99	2005-06
<b>MORTALITY (Deaths per 1000 live births)</b>									
Neonatal mortality	0.048	0.043	0.038	0.033	0.031	0.028	0.052	0.046	0.042
Postneonatal mortality	0.029	0.024	0.018	0.021	0.015	0.013	0.031	0.027	0.020
Child mortality	0.034	0.031	0.017	0.019	0.017	0.009	0.038	0.035	0.020
Under-five mortality	0.110	0.097	0.074	0.073	0.063	0.050	0.121	0.107	0.082
<b>MALNUTRITION</b>									
Stunting	30.9	28.0	22.2	24.3	19.9	16.5	32.9	30.5	24.0
Wasting	8.6	6.6	7.9	7.7	5.2	6.7	8.9	7.1	8.3
Underweight	21.9	17.8	15.9	16.6	11.4	10.6	23.5	19.8	17.6
<b>PROXIMATE DETERMINANTS</b>									
Male child	0.51	0.52	0.52	0.51	0.52	0.53	0.51	0.52	0.52
Birth order (number)	2.99	2.87	2.76	2.66	2.47	2.33	3.09	2.99	2.91
Birth interval (months)	34.06	33.04	33.76	33.19	33.06	33.67	34.32	33.04	33.79
Mother's age (years)	24.27	23.95	24.26	24.47	24.30	24.48	24.22	23.86	24.18
Improved source of drinking water	0.67	0.77	0.84	0.87	0.93	0.93	0.61	0.72	0.81
Improved sanitation facility	0.16	0.18	0.31	0.54	0.58	0.73	0.05	0.07	0.16
Solid fuel used for cooking	0.83	0.81	0.80	0.49	0.37	0.40	0.93	0.94	0.94
<b>SOCIOECONOMIC DETERMINANTS</b>									
Urban household	0.23	0.22	0.25	0.26	0.27	0.28	0.18	0.18	0.21
Mother's education									
No education	0.65	0.57	0.50	0.40	0.32	0.28	0.73	0.64	0.58
Primary	0.14	0.17	0.17	0.17	0.18	0.16	0.14	0.17	0.17
Secondary	0.17	0.22	0.28	0.34	0.38	0.42	0.13	0.18	0.23
Higher	0.03	0.03	0.05	0.09	0.12	0.13	0.01	0.01	0.02
Father's education									
No education	0.37	0.31	0.30	0.20	0.16	0.17	0.42	0.35	0.35
Primary	0.20	0.20	0.18	0.18	0.17	0.16	0.20	0.21	0.19
Secondary	0.36	0.40	0.42	0.45	0.48	0.48	0.34	0.38	0.40
Higher	0.07	0.08	0.10	0.17	0.19	0.19	0.05	0.06	0.07
Religion									
Hindu	0.79	0.79	0.78	0.70	0.71	0.73	0.82	0.82	0.80
Muslim	0.15	0.16	0.17	0.23	0.23	0.22	0.13	0.14	0.16
Other	0.05	0.05	0.05	0.07	0.06	0.06	0.05	0.04	0.04
<b>N</b>	<b>49396</b>	<b>54878</b>	<b>67041</b>	<b>11182</b>	<b>12059</b>	<b>17004</b>	<b>38214</b>	<b>42820</b>	<b>50038</b>

**Table 1B: Mortality rates, malnutrition levels, and means of determinants (proportions unless otherwise indicated)**

	North			Central			East		
	1992-93	1998-99	2005-06	1992-93	1998-99	2005-06	1992-93	1998-99	2005-06
<b>MORTALITY (Deaths per 1000 live births)</b>									
Neonatal mortality	0.035	0.041	0.035	0.057	0.053	0.047	0.054	0.042	0.041
Postneonatal mortality	0.032	0.026	0.018	0.035	0.034	0.024	0.032	0.024	0.018
Child mortality	0.027	0.026	0.016	0.051	0.044	0.026	0.030	0.033	0.015
Under-five mortality	0.094	0.093	0.069	0.142	0.131	0.097	0.116	0.098	0.074
<b>MALNUTRITION</b>									
Stunting	24.9	30.4	19.5	38.1	35.6	28.0	37.1	30.9	22.2
Wasting	7.3	4.2	6.2	8.3	6.8	8.8	11.5	8.9	9.2
Underweight	15.4	15.0	12.3	25.0	23.1	19.4	30.8	21.2	20.0
<b>PROXIMATE DETERMINANTS</b>									
Male child	0.53	0.53	0.54	0.51	0.51	0.51	0.52	0.52	0.51
Birth order (number)	2.84	2.88	2.72	3.40	3.41	3.27	3.07	2.94	2.89
Birth interval (months)	33.79	32.24	32.61	35.04	32.97	33.58	34.96	34.55	34.41
Mother's age (years)	24.53	24.61	24.61	25.25	24.50	24.89	24.26	24.05	24.02
Improved source of drinking water	0.70	0.77	0.82	0.67	0.78	0.86	0.68	0.79	0.86
Improved sanitation facility	0.18	0.26	0.36	0.10	0.10	0.22	0.12	0.15	0.22
Solid fuel used for cooking	0.79	0.75	0.75	0.92	0.89	0.87	0.82	0.93	0.93
<b>SOCIOECONOMIC DETERMINANTS</b>									
Urban household	0.26	0.27	0.28	0.18	0.18	0.21	0.17	0.12	0.15
Mother's education									
No education	0.66	0.61	0.54	0.78	0.70	0.63	0.69	0.65	0.59
Primary	0.13	0.14	0.14	0.09	0.14	0.14	0.15	0.15	0.18
Secondary	0.17	0.20	0.26	0.10	0.13	0.19	0.14	0.17	0.21
Higher	0.04	0.04	0.06	0.02	0.03	0.04	0.02	0.02	0.02
Father's education									
No education	0.35	0.29	0.27	0.38	0.32	0.33	0.43	0.39	0.39
Primary	0.15	0.17	0.15	0.18	0.19	0.16	0.19	0.20	0.20
Secondary	0.42	0.45	0.48	0.37	0.40	0.41	0.31	0.33	0.33
Higher	0.08	0.09	0.10	0.08	0.08	0.09	0.07	0.07	0.08
Religion									
Hindu	0.79	0.76	0.76	0.85	0.84	0.83	0.78	0.78	0.75
Muslim	0.06	0.13	0.14	0.14	0.15	0.16	0.20	0.20	0.22
Other	0.14	0.11	0.10	0.01	0.01	0.01	0.02	0.02	0.03
<b>N</b>	<b>5800</b>	<b>7044</b>	<b>8738</b>	<b>14643</b>	<b>16165</b>	<b>19906</b>	<b>11018</b>	<b>12067</b>	<b>16877</b>

Table 1B continued

	Northeast			West			South		
	1992-93	1998-99	2005-06	1992-93	1998-99	2005-06	1992-93	1998-99	2005-06
<b>MORTALITY (Deaths per 1000 live births)</b>									
Neonatal mortality	0.044	0.040	0.038	0.037	0.033	0.031	0.042	0.035	0.028
Postneonatal mortality	0.032	0.025	0.020	0.018	0.015	0.010	0.020	0.014	0.012
Child mortality	0.051	0.028	0.022	0.029	0.017	0.012	0.016	0.020	0.009
Under-five mortality	0.126	0.092	0.080	0.083	0.065	0.052	0.078	0.070	0.050
<b>MALNUTRITION</b>									
Stunting	29.3	31.3	18.5	25.4	22.5	20.5	19.1	18.3	15.6
Wasting	4.0	7.0	6.3	10.3	6.5	6.8	5.5	5.4	6.3
Underweight	15.5	11.4	11.5	19.4	17.2	12.6	14.3	11.2	8.3
<b>PROXIMATE DETERMINANTS</b>									
Male child	0.49	0.53	0.50	0.51	0.52	0.53	0.50	0.51	0.53
Birth order (number)	3.42	2.91	2.75	2.65	2.49	2.25	2.50	2.22	2.02
Birth interval (months)	31.29	30.97	35.79	31.63	31.79	33.36	33.96	33.21	33.77
Mother's age (years)	24.79	24.69	25.18	23.19	23.04	23.58	23.24	23.06	23.48
Improved source of drinking water	0.45	0.54	0.61	0.73	0.80	0.88	0.64	0.76	0.81
Improved sanitation facility	0.10	0.13	0.31	0.29	0.32	0.49	0.23	0.22	0.43
Solid fuel used for cooking	0.93	0.89	0.84	0.67	0.59	0.57	0.83	0.74	0.70
<b>SOCIOECONOMIC DETERMINANTS</b>									
Urban household	0.12	0.10	0.15	0.36	0.38	0.43	0.29	0.28	0.37
Mother's education									
No education	0.58	0.47	0.36	0.50	0.40	0.30	0.52	0.38	0.29
Primary	0.22	0.25	0.26	0.19	0.21	0.17	0.19	0.22	0.20
Secondary	0.18	0.26	0.34	0.27	0.34	0.46	0.26	0.35	0.42
Higher	0.02	0.02	0.04	0.03	0.05	0.07	0.04	0.05	0.09
Father's education									
No education	0.38	0.33	0.29	0.26	0.20	0.16	0.36	0.27	0.24
Primary	0.27	0.25	0.26	0.23	0.22	0.17	0.23	0.23	0.19
Secondary	0.31	0.35	0.38	0.44	0.48	0.55	0.34	0.40	0.45
Higher	0.05	0.06	0.07	0.07	0.11	0.12	0.08	0.10	0.12
Religion									
Hindu	0.54	0.50	0.51	0.78	0.81	0.80	0.80	0.80	0.81
Muslim	0.28	0.28	0.26	0.15	0.13	0.14	0.14	0.13	0.13
Other	0.18	0.22	0.23	0.07	0.06	0.06	0.06	0.06	0.05
<b>N</b>	<b>2145</b>	<b>2007</b>	<b>2519</b>	<b>6537</b>	<b>7221</b>	<b>8390</b>	<b>9253</b>	<b>10375</b>	<b>10611</b>

**Table 2: Mortality and malnutrition by wealth groups (based on NFHS1 20% cutoffs)**

	<b>National</b>			<b>Change from 1992 to 2005</b>	
	<b>Wealth groups based on NFHS-1 cutoffs</b>				
	<b>1992-93</b>	<b>1998-99</b>	<b>2005-06</b>	<b>Absolute</b>	<b>Percent</b>
<b>MORTALITY (deaths per 1000 live births)</b>					
<b>Neonatal</b>					
Lowest	0.058	0.054	0.048	-0.011	-18.2
Second	0.057	0.048	0.046	-0.012	-20.6
Middle	0.048	0.045	0.044	-0.004	-8.7
Fourth	0.033	0.038	0.035	0.002	6.7
Highest	0.026	0.026	0.026	0.000	-1.0
<b>Poorest/Richest ratio</b>	<b>2.3</b>	<b>2.1</b>	<b>1.9</b>	<b>-0.4</b>	<b>-17.4</b>
<b>Postneonatal</b>					
Lowest	0.038	0.035	0.026	-0.012	-32.5
Second	0.034	0.032	0.022	-0.012	-35.9
Middle	0.027	0.024	0.022	-0.005	-17.4
Fourth	0.021	0.017	0.015	-0.007	-31.3
Highest	0.013	0.011	0.009	-0.004	-29.8
<b>Poorest/Richest ratio</b>	<b>2.9</b>	<b>3.1</b>	<b>2.8</b>	<b>-0.1</b>	<b>-3.8</b>
<b>Child</b>					
Lowest	0.052	0.052	0.026	-0.026	-50.5
Second	0.043	0.041	0.029	-0.014	-32.7
Middle	0.030	0.031	0.024	-0.006	-19.5
Fourth	0.017	0.020	0.009	-0.007	-43.3
Highest	0.007	0.005	0.004	-0.003	-43.4
<b>Poorest/Richest ratio</b>	<b>7.6</b>	<b>10.9</b>	<b>6.6</b>	<b>-0.9</b>	<b>-12.4</b>
<b>Under-five</b>					
Lowest	0.148	0.140	0.099	-0.049	-33.2
Second	0.134	0.122	0.096	-0.038	-28.3
Middle	0.105	0.100	0.090	-0.015	-14.0
Fourth	0.071	0.075	0.060	-0.012	-16.4
Highest	0.046	0.042	0.038	-0.007	-15.6
<b>Poorest/Richest ratio</b>	<b>3.2</b>	<b>3.4</b>	<b>2.6</b>	<b>-0.7</b>	<b>-20.8</b>

Table 2 continued

**MALNUTRITION (percentage)**

## Stunting

Lowest	38.7	39.6	32.7	-6.0	-15.5
Second	36.4	35.8	30.6	-5.8	-16.0
Middle	32.4	30.4	25.3	-7.2	-22.1
Fourth	23.2	23.1	19.3	-3.9	-16.8
Highest	16.6	13.0	10.0	-6.6	-39.8
<b>Poorest/Richest ratio</b>	<b>2.3</b>	<b>3.0</b>	<b>3.3</b>	<b>0.9</b>	<b>40.3</b>

## Wasting

Lowest	9.8	10.0	11.0	1.3	12.9
Second	10.6	7.8	9.5	-1.1	-9.9
Middle	8.5	7.1	8.6	0.2	1.9
Fourth	6.9	4.7	7.0	0.1	1.9
Highest	6.3	4.3	5.1	-1.2	-19.6
<b>Poorest/Richest ratio</b>	<b>1.6</b>	<b>2.3</b>	<b>2.2</b>	<b>0.6</b>	<b>40.4</b>

## Underweight

Lowest	28.4	27.4	25.5	-2.9	-10.3
Second	26.8	25.2	23.4	-3.3	-12.4
Middle	22.7	19.3	18.5	-4.1	-18.2
Fourth	15.3	12.7	12.3	-3.0	-19.5
Highest	10.8	6.4	5.6	-5.1	-47.6
<b>Poorest/Richest ratio</b>	<b>2.6</b>	<b>4.3</b>	<b>4.5</b>	<b>1.9</b>	<b>71.3</b>



**Table 3: Summary statistics for the unstandardized wealth score**

	National	Urban	Rural	North	Central	East	Northeast	West	South
<b>Mean</b>									
1992-93	0.08	-0.12	0.24	0.28	-0.17	-0.17	0.25	-0.02	-0.08
1998-99	0.48	0.47	0.72	0.43	0.14	0.11	0.73	0.76	0.92
2005-06	1.34	0.76	1.50	1.02	1.38	1.06	2.04	1.68	2.13
<b>Median</b>									
1992-93	-0.70	0.06	-0.46	-0.06	-1.00	-0.90	-0.52	-0.59	-0.72
1998-99	-0.18	0.65	-0.09	0.17	-0.74	-0.76	-0.01	0.77	0.66
2005-06	0.90	1.06	0.71	0.90	0.39	-0.13	1.37	1.93	2.07
<b>Std. deviation</b>									
1992-93	2.27	2.24	2.09	2.28	2.12	2.03	2.21	2.30	2.10
1998-99	2.37	2.00	2.36	2.30	2.24	2.25	2.34	2.22	2.26
2005-06	2.58	1.95	2.74	2.29	2.82	2.69	2.72	2.35	2.39
<b>Minimum</b>									
1992-93	-2.34	-4.59	-1.72	-3.37	-2.08	-1.84	-1.75	-3.07	-2.38
1998-99	-2.34	-4.59	-1.72	-3.37	-2.08	-1.84	-1.75	-3.07	-2.38
2005-06	-2.34	-4.59	-1.72	-3.37	-2.08	-1.84	-1.75	-3.07	-2.38
<b>Maximum</b>									
1992-93	7.54	4.13	11.59	5.67	8.53	9.44	10.02	6.01	7.75
1998-99	7.54	4.13	11.59	5.67	8.53	9.44	10.02	6.01	7.75
2005-06	7.54	4.13	11.59	5.67	8.53	9.44	10.02	6.01	7.75
<b>Range</b>									
1992-93	9.87	8.72	13.32	9.04	10.61	11.28	11.77	9.09	10.13
1998-99	9.87	8.72	13.32	9.04	10.61	11.28	11.77	9.09	10.13
2005-06	9.87	8.72	13.32	9.04	10.61	11.28	11.77	9.09	10.13
<b>N</b>									
1992-93	59013	15939	43074	13277	14009	9807	6591	6263	9066
1998-99	55897	14307	41590	13155	12305	9680	7738	5533	7486
2005-06	50677	19160	31517	7972	12666	7969	9444	5500	7126

**Table 4: Relationship between wealth and mortality and malnutrition**  
(Odds ratios from logistic regressions (with and without fixed effects))

	National		Urban		Rural	
	Logit	Logit+FE	Logit	Logit+FE	Logit	Logit+FE
<b>MORTALITY</b>						
Neonatal						
Wealth - 1992	0.82 ***	0.86	0.88	0.91	0.84 ***	0.86
Wealth - 1998	0.89 **	0.92	0.93	0.97	0.90 **	0.92
Wealth - 2005	0.93 b	0.99	0.97	0.91	0.96 b	1.02
Postneonatal						
Wealth - 1992	0.81 ***	0.83	0.96	0.95	0.81 ***	0.84
Wealth - 1998	0.75 ***	0.76	0.92	0.97	0.77 ***	0.76 **
Wealth - 2005	0.82 **	0.78	0.94	0.97	0.85 *	0.81
Child						
Wealth - 1992	0.55 ***	0.51	0.57 ***	0.51	0.69 ***	0.64 *
Wealth - 1998	0.53 ***	0.50 *	0.58 ***	0.53	0.62 ***	0.60 **
Wealth - 2005	0.59 ***	0.55 *	0.73	0.78	0.64 ***	0.58 **
Under-5						
Wealth - 1992	0.77 ***	0.79 *	0.85 **	0.84	0.80 ***	0.82 **
Wealth - 1998	0.78 ***	0.79 **	0.86 **	0.88	0.81 ***	0.82 ***
Wealth - 2005	0.86 *** b,c	0.87	0.93	0.89	0.89 ** b,c	0.90
<b>MALNUTRITION</b>						
Stunting						
Wealth - 1992	0.78 ***	0.81 *	0.91	0.83	0.79 ***	0.85 *
Wealth - 1998	0.69 *** a	0.68 ***	0.80 *** a	0.74 *	0.76 ***	0.75 ***
Wealth - 2005	0.71 *** b	0.60 *** b	0.80 *** b	0.64 **	0.75 ***	0.68 *** b
Wasting						
Wealth - 1992	0.85 **	0.90	0.85 *	0.84	0.90	0.96
Wealth - 1998	0.75 *** a	0.88	0.92	1.09	0.75 *** a	0.85
Wealth - 2005	0.78 ***	0.89	0.83 *	0.96	0.84 ***	0.92
Underweight						
Wealth - 1992	0.74 ***	0.81	0.84 **	0.78	0.78 ***	0.87
Wealth - 1998	0.61 *** a	0.66 ***	0.72 *** a	0.76	0.68 *** a	0.72 *** a
Wealth - 2005	0.61 *** b	0.62 ***	0.73 *** b	0.67 *	0.67 *** b	0.70 *** b

\*\*\*p<.001, \*\*p<.01, \*p<.05

a: wealth 1998~=wealth 1992, b: wealth 2005~=wealth 1992, c: wealth 2005~=wealth 1998, at p<.05

Note 1: Control variables include sex of child, birth order, previous birth interval, mother's age at birth of index child, improved sanitation facility, improved source of drinking water, solid cooking fuel, urban residence, mother's and father's education, religion of household head.

Note 2: The coefficients on the wealth terms for 1998 and 2005 are calculated as a linear combination of the primary wealth effect and the survey year-wealth interaction effect.

Table 4 continued

	North		Central		East		Northeast		West		South	
	Logit	Logit+FE	Logit	Logit+FE	Logit	Logit+FE	Logit	Logit+FE	Logit	Logit+FE	Logit	Logit+FE
<b>MORTALITY</b>												
Neonatal												
Wealth - 1992	0.92	0.97	0.82 *	0.92	0.91	0.84	0.79	0.78	0.70 *	0.82	0.91	0.76
Wealth - 1998	0.74 *** a	0.74	0.92	0.95	0.82	0.94	1.05	1.12	0.94	0.89	0.98	0.95
Wealth - 2005	0.84	0.88	0.92	0.91	1.22	b,c	0.95	1.27	0.69 * c	0.60	1.05	1.15
Postneonatal												
Wealth - 1992	0.91	0.93	0.73 *	0.89	0.70 *	0.80	0.69	0.72	0.96	0.89	0.77	0.75
Wealth - 1998	0.81	0.92	0.67 **	0.71	0.84	0.82	0.86	0.98	0.74	0.47	0.66 *	0.86
Wealth - 2005	0.98	0.98	0.79	0.67	0.72 *	0.77	0.66	0.81	1.02	1.59	0.58 *	0.53
Child												
Wealth - 1992	0.55 ***	0.51	0.48 ***	0.49	0.76	0.58	0.40	0.60	0.47 **	0.46	0.33 ***	0.36
Wealth - 1998	0.54 ***	0.52	0.52 ***	0.54	0.50 *	0.48	0.60	0.51	0.49 **	0.50	0.39 ***	0.32
Wealth - 2005	0.57 *	0.53	0.56 **	0.54	0.58	0.68	1.13	b	0.48 *	0.73	0.39 **	0.32
Under-5												
Wealth - 1992	0.84 **	0.87	0.74 ***	0.82	0.82 *	0.80	0.67	0.70	0.69 **	0.75	0.79 **	0.68
Wealth - 1998	0.72 *** a	0.75 *	0.76 ***	0.80	0.78 **	0.82	0.94	a	0.82	0.70	0.80 **	0.83
Wealth - 2005	0.84 * c	0.85	0.82 **	0.77 *	1.02	b,c	0.89	1.22	0.71 **	0.73	0.83	0.81
<b>MALNUTRITION</b>												
Stunting												
Wealth - 1992	0.78 ***	0.79	0.94	1.02	0.84 *	0.83	0.58 ***	0.49	0.66 ***	0.71	0.68 ***	0.61
Wealth - 1998	0.68 *** a	0.72 **	0.64 *** a	0.64 *** a	0.71 ***	0.78	0.88	a	0.67 ***	0.61	0.72 ***	0.66 **
Wealth - 2005	0.70 ***	0.57 *	0.70 *** b	0.63 *** b	0.59 *** b,c	0.56 **	0.64 *** c	0.69	0.68 ***	0.62	0.66 ***	0.67 *
Wasting												
Wealth - 1992	0.75 **	1.13	1.05	1.21	1.03	1.08	1.03	1.10	0.81	0.61	0.68 *	0.61
Wealth - 1998	0.79 *	0.92	1.00	1.01	0.87	0.94	0.65	0.58	0.78	0.92	0.68 ***	0.70
Wealth - 2005	0.79 *	0.91	0.85	0.95	1.01	1.02	0.90	0.79	0.73 *	0.86	0.79	0.71
Underweight												
Wealth - 1992	0.77 ***	0.86	0.95	1.06	0.89	0.87	0.62 *	0.67	0.55 ***	0.54	0.69 **	0.57
Wealth - 1998	0.57 *** a	0.61 **	0.65 *** a	0.63 ** a	0.72 *** a	0.84	0.99	a	0.50 ***	0.63	0.66 ***	0.64 *
Wealth - 2005	0.64 *** b	0.70	0.68 *** b	0.67 ** b	0.65 *** b	0.63 *	0.73 *	0.61	0.58 ***	0.51 *	0.65 ***	0.60 *