The Irreversible Health Effects of Economic Fluctuations

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First draft

Abstract

This paper investigates the impact of macroeconomic shocks on childhood mortality in India, allowing for endogenous changes in the timing of fertility. It also investigates the extent to which this impact is delivered *via* changes in public health expenditures, private expenditure, and changes in the income distribution. I merge demographic data at the individual-level with a panel of state-level data on GDP and other macroeconomic and political variables by cohort. The data contain almost 200,000 children born across 15 states and 30 years. Microeconomic evidence from rich and poor countries suggests a positive association of income shocks and health. Evidence of a favourable impact of aggregate income shocks on health is more limited. The results suggest that (temporary) downturns in the economy raise mortality, the elasticity for infants being about -0.3. In this way, business cycles have real and irreversible effects on welfare.

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1. Introduction

In the last thirty years, aggregate income growth in developing countries has been volatile, subject to both cyclical fluctuations and episodic crises (e.g. Pritchett 2000). By investigating their impact on childhood mortality, this paper investigates the extent to which macroeconomic shocks have irreversible real effects (see Lucas 1977). Given evidence of a lasting impact of poor living conditions in childhood, the effects of income shocks on mortality may be regarded as a lower bound on effects of income shocks on lifetime health.

In poor countries, 30% of deaths are amongst children, compared with less than 1% in rich countries, and most of these deaths are avoidable (Cutler et al 2005, p.15). While there is a vast demographic literature on the proximate determinants of childhood mortality, there is limited evidence on the question investigated here, which is of the extent to which stabilising aggregate economic fluctuations or else protecting the vulnerable from their effects would avert childhood death.

The analysis in this paper is conducted on data that I create by merging individual-level demographic information from a large household survey in India with state-level data on macroeconomic and political variables, matching the birth year of the child with calendar year in the state panel. The estimation sample contains 163907 children of 50379 mothers born during 1970-1998 across the fifteen major states of India. India offers an appropriate setting for the analysis as it has one in six of the world's people, one in four of under-5 deaths, and one in three of the world's poor. India has not had an aggregate crisis that compares in severity with the crises recently experienced by Indonesia, Peru, Argentina or Mexico. Although crises situations are more amenable to an experimental analysis, the results of such analyses are more difficult to generalize. In India, aggregate growth was steady and slow until the mid-80s, after which it accelerated to a less steady but consistently higher level. Moreover, there were considerable differences in the growth experiences of the Indian states. Less known, there was also considerable variation in childhood mortality rates around

a trend decline. The relatively long time dimension of the data makes it more likely that there are independent macroeconomic fluctuations across the states, upon which identification relies. Like the United States, India has a federal structure. Health is a "state subject", which means that the level and the allocation of health expenditure are decided at the state level. The states differ remarkably in other relevant dimensions, including political institutions, ethnic composition and climate.

There is an underlying downward trend in mortality and, with the exception of a set of countries in sub-Saharan Africa, a secular upward trend in aggregate income. In the long run, there is a fairly strong correlation between income and mortality that shows in cross-country data and in historical time series (e.g. Pritchett and Summers 1996, Lorentzen et al 2005). This probably reflects causation in both directions, from income to mortality and from mortality to income (e.g. Shastry and Weil 2000, Lorentzen et al 2005). A particular difficulty with identifying from such data a causal effect of income on mortality is that the effects of health technology will tend to load on to the income effect. Although panel data is useful in this regard, cross-country panels are only as good as is the assumption that health technology trends across diverse countries can be captured by common time effects (e.g. Deaton and Paxson (2001, 2004), Deaton 2003). The impact identified in a panel data model is, of course, of deviations of income from (a flexible) trend. As these are typically unanticipated and relatively high frequency changes, they may have different effects than similar changes in trend income. For instance, fluctuations in income, such as associated with economic cycles, may induce people in tight labour market conditions to make a temporary substitution of their time away from health-producing activities like sport, but this is consistent with sustained increases in economic activity being beneficial to health (e.g. Ruhm 2000).

The model includes year and state fixed effects and state-specific trends. These control for omitted variables, such as trends in health technology and state-specific initial conditions, which will tend to confound the relationship of interest if they are correlated with both income and mortality at the state level. The fact that the states are regions within a country, rather than a diverse set of countries, makes it more likely that there are common trends in technology and below, we will see that observed patterns in the Indian data are consistent with the diffusion of technology across the states over time. By virtue of regressing individual mortality risk on a regional income aggregate, I limit concerns about reverse causation. For comparison,

I also estimate panel data models using the same mortality data but aggregated to the state-year level. In these models, reverse causality is possible. Although child mortality may not directly impact upon income, indirect effects may arise via fertility (e.g. Kalemli-Ozcan 2002) or via the impact on adult productivity of caring for sick children and, indeed, of grieving their deaths. In the panel data version of the model, I use lags of GDP and rainfall shocks as instruments in a systems framework.

The survey data contain detailed information on maternal and household characteristics, which is used to control for heterogeneity in the composition of births. For example, if recessions induce a shift in the composition of mothers giving birth towards low-risk mothers then, other things equal, childhood mortality will be lower in recessions. However, if public and private expenditures decline in recessions, then we would expect that mortality rises. Thus, failing to control for heterogeneity in the children that enter the sample (live births of mother j in state s and year t) can bias conclusions regarding the cyclicality of mortality.

This paper also investigates possible mechanisms by which any composition-adjusted effects operate. In particular, it looks at the role of private consumption expenditure, public health expenditure, poverty and inequality. In work in progress, I investigate political economy variables that may explain inter-state or inter-temporal variation in the main effects.

The data contain longitudinal information on cohorts of children, which is used to identify when economic conditions have their greatest impact on survival chances- whether at the time the child is in the womb, at birth, or in early childhood. To investigate how the impact of income shocks varies with the age of the child, separate models are estimated for neonatal (first month of life), infant (first year of life) and under-5 (first five years of life) mortality. The alternative, of estimating the model for successive age-groups defined, for example, as 0-1 year, 1-2 years, 2-3 years, etc., is avoided because it involves conditioning upon survival until the entry age. Since the data record only live births, I am still conditioning upon survival until birth. To the extent that recessionary conditions that occur when the child is in the womb result in miscarriage or stillbirth, I will under-estimate their impact on survival risk. With this caveat in mind, I nevertheless estimate this impact as well, and this is done by using lagged GDP.

The main results are as follows. In the baseline model, I find no effect of (annual) economic fluctuations on the probability of neonatal death, but GDP attracts

a significant negative coefficient of about -0.3 in the equation for infant mortality, and about -0.6 in the equation for under-5 mortality. With contemporaneous GDP in the model, the effect of lagged GDP is insignificant. Consider the difference that the fixed effects and state-trends make for the case of under-5 mortality. The unconditional growth elasticity is about -0.7. Including state fixed effects pushes this up to -1.0. Once I also control for year effects, it falls to -0.6. This is consistent with the year effects capturing trend improvements in health technology or possibly health-related infrastructure and services.

There is some evidence that the composition of births changes across the cycle. In recessions, the proportion of births contributed by mothers with no education and mothers of a disadvantaged group referred to as "scheduled tribes" falls. Controlling for selection on education and ethnicity of the mother raises the absolute value of the elasticity, but the change is not significant.

Adding state-level mean consumption per capita to the model results in a small reduction in the size of the GDP coefficient, but it remains significant; and consumption has an additional mortality-reducing effect. Similar results obtain upon adding a measure of poverty as a regressor. Public health expenditure per capita is insignificant and has no impact on the GDP coefficient. Recall that these results obtain after de-trending (and de-meaning) the series. Straightforward correlations show the expected negative association of public health expenditure and mortality. What the regression results suggest is that fluctuations in health expenditure do not impact on fluctuations in mortality.

There are significant effects on survival chances of mother's and father's education, ethnicity, religion, maternal age at birth and birth-order. The sectoral composition of growth matters: mortality is decreasing in the ratio of agricultural to non-agricultural output, total output constant. The consumer price index in rural relative to urban areas is insignificant, agricultural price inflation is insignificant but industrial price inflation is mortality-increasing. Estimates of state-specific elasticities show that the estimated impact of cyclical income on mortality is significant in only 8 of the 15 states. The estimates are robust to a range of specification tests, including allowance for dynamics, endogeneity and measurement error.

The rest of the paper is organized as follows. Section 2 reviews the relevant literature and outlines the contributions of this paper. The data and descriptive statistics are described in sections 3 and 4 respectively. Section 5 describes the

research strategy, discussing the choice of estimator and specification issues. Results are presented in section 7, and conclusions in section 8.

2. Related Research and Contributions

In this section, I survey the related literature, explain some of the hypotheses to be investigated, and highlight some contributions of the proposed work. Methodological contributions are detailed in the section on research strategy below.

There is an impressively wide scale of health differences both within and across countries. The evidence at the microeconomic level is fairly robust (e.g. Case *et al* 2005, Wagstaff 2000). Although cross-country data also show an income gradient in health, the relation is, at this level, confounded by the enormous heterogeneity across countries in institutions, governance, diet and climate, all of which are expected to impact mortality at any given level of aggregate income. Using cohort-data from the UK and the US, Deaton and Paxson (2004) find no effect of income on mortality in the UK and a small effect in the US. In both cases, the effect is considerably diminished by addition of time dummies and of education to the model. That the protective effect of income is smaller in richer countries is consistent with their having crossed a threshold standard of living associated with the epidemiological transition.

Income shocks may be expected to impact on health in households that are poor and liquidity constrained; and children along with elderly people are especially vulnerable. The evidence from micro-data is consistent with this. Looking at the health of American children, Case *et al.* (2001) find a large and robust effect of permanent income but little impact of the timing of income. In contrast, Hoddinott and Kinsey (2000), Rose (2000) and Duflo (2000) show that income shocks affect child health in Zimbabwe, India and South Africa respectively.

The impact of macroeconomic shocks is harder to predict, as several possible mechanisms are at play, some of which have opposing effects. Aggregate income fluctuations can impact individual health by changing mean incomes and also the income distribution. They can further have an impact by changing the supply or the demand for health services. On the supply side, recessions are often associated with cuts in social spending, that result in reduced availability or higher prices for health services (e.g. Lustig 1999, Stiglitz 1999) and this, on its own, may be expected to

damage health. On the demand side, however, there is some evidence that people are more likely to adopt healthy behaviours, including utilization of available health services, in recessions because the opportunity cost of their time is lower (e.g. Ruhm (2003, 2005) and Dehejia and Lleras-Muney (2004), who relate annual US data state unemployment rates to indicators of gym attendance and utilization of antenatal care respectively). They also show that alcohol and tobacco consumption are lower in recessions, possibly because of lower stress levels amongst those who remain employed but work fewer hours or, in the case of pregnant mothers, because partners will also tend to work fewer hours. In the case of babies and young children, any adverse income effects of recession on health may be mitigated by the (endogenous) timing of birth, with poorer women tending to avoid giving birth in a recession (e.g. Dehejia and Lleras-Muney 2004, Paxson and Schady 2005). Further channels through which fluctuations in aggregate economic activity may impact upon health arise in the level of industrial pollution (Chay and Greenstone 2003), or the extent of local population congestion (e.g. Haines 1979). In both of these cases, a slackening of economic activity is associated with a healthier environment. Over longer time periods, compensating adjustments are made. For example, pollution abatement technologies improve, city infrastructure develops to sustain an inflow of migrants, and individuals revert to their trend behaviours.

In their analysis of US panel data, Ruhm (2000) and Dehejia and Lleras-Muney (2004) find relative gains in health during recessions, recessions being indicated as deviations of the state-level unemployment rate from aggregate or within-state trends. Using German micro-data, Dustmann and Windmeijer (2004) show that while higher wealth is associated with better health, temporary rises in wages worsen health. Similar results are reported in Granados (2005), Neumeyer (2004) and Gertrad and Ruhm (2004) for the US, Germany and a panel of OECD countries. Together these studies suggest that substitution effects and externalities induced by temporary income shocks are large enough to swamp the income effect. Earlier work, using time-series data, had found the opposite pattern (e.g. Brenner 1979). An advantage of panel data in this context, emphasized by Ruhm (2000), is that correlated omitted variables can be captured in state and year fixed effects. An important "omitted variable", emphasized in a series of papers by Angus Deaton and co-authors (e.g. Deaton 2003, Cutler et al 2005), is health technology. This has trended upwards and, by all accounts, has had a causal impact on the upward trend in health (or the

downward trend in childhood mortality). In time series analyses, the favourable effect of (omitted) health technology will tend to load onto included trended variables like income, overstating their impact. In panel data, year dummies and region-specific trends will control quite effectively for health technology and a range of other relevant omitted variables (e.g. fertility, services). In fact they will also control for trends in national and regional aggregate income, so that the model estimates the impact on health (or mortality) oscillations of temporary income shocks or deviations from (a flexible) trend. For these reasons, results of the more recent panel data analyses now appear to hold sway.

Similar evidence from developing countries is still relatively scarce, although we may expect that the income effects of shocks will dominate in regions where incomes are low and volatile and neither markets nor social institutions provide sufficient opportunities to smooth consumption (e.g. Morduch 1999).[1] Indeed, the effect of macroeconomic shocks on childhood mortality may be seen as an indicator of the extent of vulnerability in a region.

A frequently cited study by Pritchett and Summers (1996) shows that increases in GDP are significantly associated with a decline in under-5 mortality in a panel of 58 developing countries. They explain that international statistics on mortality are collected on a five-yearly basis, the published annual series being obtained by interpolation. They therefore use quinquennial data, with at most five observations per country in the period 1960-85. The elasticity is about -0.2 in a model estimated in five-year first-differences and about -0.3 in a model estimated in levels with country fixed effects. These estimates may be biased for the following reasons. Although they include time dummies, they do not include country-specific time trends. Any country-time variation in technological change in their diverse set of countries will therefore tend to load onto the GDP variable, magnifying its effect on mortality. The effect may be further overstated if high-risk mothers delay birth in recessions, as this study does not allow for the endogenous timing of births. To the extent that the study does not adjust the reported standard errors for clustering by state, or for autocorrelation induced by differencing, it may also overstate the significance of the GDP effect. Examination of the properties of the error terms is

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[1] A lively literature has emerged on the scope for informal insurance in village economies, the conclusion of which is that most people are only imperfectly insured, and the poorest least so.

especially relevant since this study estimates a static model without any reported investigation of dynamics.

An advantage of sub-national analyses is that it is more likely that the regions (states) have a common technology (and common components of other omitted variables) that the time dummies will capture. I nevertheless include state-specific trends in the model. I also allow for the endogenous timing of births. The use of annual as opposed to quinquennial data make it more likely that the estimates presented in this paper reflect the effects of temporary income shocks.[2] Pritchett and Summers instrument GDP with an indicator of terms of trade shocks. An advantage of regressing individual mortality risk (rather than the state-level mortality rate) on aggregate income at the state level is that it mitigates concerns regarding possible reverse causality. For comparison, I present results from a panel data analysis in which I instrument GDP with its lags, and investigate rainfall shocks as a further overidentifying restriction. I also investigate dynamics (lagged dependent variables) and lagged effects of income.

Most of the other evidence available for developing countries looks at the impact on health of a single large crisis, typically using time-series data. Rucci (2004) and Cutler and Brainerd (2004), who analyse the crises in Argentina and Russia respectively, find no impact. However, severe adverse effects associated with macroeconomic crises are identified in Peru (Paxson and Schady 2005), Mexico (Cutler et al 2002) and Indonesia (Rukumnuaykit 2003). The different findings of these studies may reflect differences in methodology or differences in the nature of the shock, initial conditions like initial inequality, and the institutional buffers available to people. While the study by Paxson and Schady, for example, produces incontrovertible evidence that a severe negative shock to aggregate income in Peru raised mortality, it is harder to predict the likely effects of smaller shocks or economic cycles. Crises interventions are likely to be of a specific one-off nature, whereas most countries, especially developing countries, would benefit from mechanisms that shield the most vulnerable from suffering irreversible consequences (like child death) from transient shocks (like temporary increases in unemployment or in food prices).

A notable study uses Dutch data on individuals born between 1812-1912 in Utrecht, and followed up until 1999 (Lindeboom *et al.* 2005). These data are matched

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[2] The paper by Lant Pritchett and Larry Summers is called Wealthier is Healthier, reflecting the underlying notion of an association of permanent income and health.

with time series on GDP, with the particular aim of looking at the impact of recessionary conditions in childhood on survival chances later in life, although contemporary effects are also estimated. For a long stretch of these data, the population was predominantly agricultural and baseline living conditions were poor. The main result of the study is that children were more likely to survive at the time, and also later in life (especially after the age of 50) if, in their early childhood (age 1-7), GDP was relatively high. The approach in this paper is to estimate hazard models on individual data, allowing flexible age and time effects, with GDP varying across cohorts. Given the unusually long span of data, there is only a limited set of covariates, and the results for infants and young children are subject to the caveat that they will reflect not only the direct causal effect of GDP on mortality but also any effects that operate *via* the effects of GDP on the composition of mothers giving birth.

Overall, the following broad patterns emerge from the existing literature. At the family level, permanent income tends to exhibit a positive correlation with child health in most countries, while income shocks seem to affect child health in poor countries more clearly than in richer countries. At the aggregate level, most of the evidence on child health relates to childhood mortality. Secular trends in income exhibit a clear positive relationship with trends in childhood mortality. However, mortality appears to be pro-cyclical in rich countries. In poor countries, there is more limited evidence of the effect of the cycle but the available evidence suggests that childhood mortality is counter-cyclical. Against this backdrop, the observation made by Deaton and Paxson (2004) following their analysis of individual and aggregated data from the US and the UK is pertinent: "*understanding the effect of income on mortality presents many puzzles, between countries, and between analyses at different levels of aggregation.*" Also see Fuchs (2004), Sen (1998) and Dreze & Sen (1989)).

Some of the studies show, by estimation of independent auxiliary equations, that relevant behaviours (e.g. use of antenatal care, alcohol consumption) or the composition of births (e.g. percent black) respond to cyclical income (see Ruhm 2000, Dehejia and Lleras-Muney 2004, Paxson and Schady (2005). However, these studies do not complete the chain- in other words, they do not quantify the impact of, say, antenatal care on the health outcome. Nor do they explicitly condition upon the "channel variables" and observe the change in the coefficient on the cyclical income term (unemployment rate or GDP, as the case may be). As a result, we do not know how much of the main effect the suggested mechanisms account for, although the

auxiliary analyses do indicate their plausibility. I am aware of two studies that do include "channel variables" as regressors. Using cross-country data, Anand and Ravallion (1993) find a significantly negative relation of mortality and GDP, but they show that this is eliminated once they condition upon poverty and public spending. A recent report on India reports a positive effect of GDP on mortality conditional upon social expenditure and relevant indices of public infrastructure – but it does not report how the GDP coefficient changes when these variables are dropped (World Bank 2004).

3. Data

The micro-data are from the second round of the National Family Health Survey of India (NFHS), which recorded complete fertility histories for ever-married women aged 15-49 in 1998-99, including the time and incidence of child deaths.[3] Individual mortality data are thus available for cohorts of children followed over time from birth. Children in the sample are born in 1961-1999. To diminish selectivity bias arising from an upper limit on the age of the mother at the time of interview, I drop children born in the 1960s. The sample contains 163907 children of 50379 mothers. These data have (unexploited) potential to shed light on trends in fertility, mortality and related demographic change.

The analysis is conducted separately for neonatal, infant and under-5 deaths. To allow every child full exposure to mortality risk, the effective sample of births for the infant mortality estimation is 1970-1997, and for under-5 mortality, is 1970-1994. In both cases, since GDP before and after birth is allowed to matter, the GDP data are for 1969-1999. A rich set of micro-demographic controls is derived from the NFHS.[4] Data on household income are not available and are difficult to estimate for the entire retrospective period. However, using recent data, for which these data are available, I find that the parental education and demographics (which I include) explain about 40% of the cross-sectional variation in income.

Using sampling weights, I have aggregated the micro-data from the NFHS to the state level to produce annual mortality rates. These data are merged with a panel

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[3] For details on sampling strategy and context, see IIPS and ORC Macro (2000).
[4] Time-variation in these variables arises through following successive cohorts: the educational level of mothers of children born in the 1990s is higher than that of children born in the 1970s.

of data on real net state domestic product per capita (abbreviated, if inaccurately, as GDP) and other relevant statistics for the 15 Indian states, over the chosen period. These data were assembled by Ozler, Datt and Ravallion (1996) and then extended by Besley and Burgess (2002, 2004), who were kind enough to supply me with their database. The merge is done by state and time, where calendar time in the panel is matched to the year of birth of the child in the micro-data (henceforth t). So for children born in 1980 and exposed to the risk of under-5 death during 1980-85, we have matched information on GDP in 1980. In the estimated model, I regress the under-5 mortality rate for children born in year t on GDP averaged over the period (t, t+5), which is the relevant exposure period.

The NFHS contains information on a number of relevant characteristics of the mothers, their partners and the children. Control variables included in the analysis include the gender and birth-order of the child, the religion, ethnicity and sectoral (rural/urban) location of the household, the educational level of each parent and the age of the mother at birth of the child. These characteristics have been shown to be significant predictors of mortality risk in a number of previous studies, and also on these data (see Arulampalam and Bhalotra 2004). Some of these characteristics are potentially correlated with economic growth, for example the educational level of parents. For this reason, I present estimates of the effect of GDP on mortality obtained before and after introducing the control variables.

4. Descriptive Statistics

In 1990, the under-5 mortality in India was 8.7%, as compared with 17.9% in 1970 & 11.2% over the sample period. Infant [neonatal] mortality is, on average, 72% [46%] of under-5 mortality. Under-5 mortality decreased at 3.44% p.a. over the sample period, the rate of decline having slowed in the last decade. State-specific trends exhibit convergence over time. GNP increased at 2.87% p.a., and there was an acceleration in growth in mid-80s. The state-series show no clear tendency towards convergence.

There are vast differences in the level of mortality across the Indian states, which demonstrate the scope for reduction in the overall level. Averaging over the period 1970-98, the incidence of mortality ranged from 4.8% in Kerala to 17.3% in Uttar Pradesh. The average linear rate of decline in mortality during the period

analysed in this paper, 1970-95, is estimated at 2.83% p.a. It is more accurate to look at averages up to 1995, because only then do all children have full exposure to under-5 mortality risk. The average linear rate of growth during 1970-95 is estimated at 2.61% p.a., the rate in the period 1970-98 being 3% p.a. State-specific rates of growth vary considerably, ranging between 4.4% in Maharashtra (the industrial capital) and 1.8% in Bihar (one of the very poor states) (see Table 1).

(All figures are currently for under-5 mortality but figures for infant mortality are similar). Figure 1 shows trends in under-5 mortality for each of the 15 major states of India. The first panel shows the actual data by state, and the second the population-weighted average for India. Mortality has declined fairly steadily in India as a whole, and in every state. The graphs show that mortality rates across states have tended to converge over time, although there is clearly a role for (unobservable) "state effects" on mortality on top of a role for the initial level of mortality. Thus, some states with initially low mortality (like Kerala) have achieved further declines at a rapid pace, while other states (like Assam or Punjab) that started out with relatively low levels of mortality have progressed at a more sluggish pace.

Figure 2a shows trends in the logarithm of real per capita net state domestic product (henceforth GDP) for every state. The picture indicates a fairly steady increase, with some acceleration in most states starting in the 1980s. The figure suggests limited if any convergence across the states. In Figure 2b are trends in the fifth difference of the log of GDP for every state. This graph shows the extent of volatility in growth, and confirms that there is no clear indication of convergence in GDP levels. *The fact that there is convergence in mortality rates across states but not in GDP may be explained by improvements in health technology that, independent of GDP, have diffused across the states.*

Figure 3a puts the all-India trends in mortality and GDP on the same axes. Since both series are trended, with mortality declining and GDP increasing over time, any correlation between these series will be spurious to the extent that it picks up common trends. For this reason, Figure 3b plots the two time series after *de-trending* both. This is done by regressing each of mortality and GDP on a set of time dummies and saving the residuals. The plot is of these residuals. This is equivalent to regressing mortality on GDP and a set of time dummies. So what we have in Figure 3b is the relationship that we are really interested in identifying: the relation of growth and mortality after taking out any other trended variables that might otherwise confound

the relation. A casual glance at Figure 3b makes it difficult to discern any clear relation. In other words, controlling comprehensively for other trended variables like advances in health technology and services, and for temporal shocks like floods or famines, there is, in the aggregate, no *evident* relation of growth and mortality.

5. Research Strategy

5.1. The Estimated Equations

These are displayed as (1) and (2) below, where subscripts *s* and *t* indicate state and year and *i* and *f* indicate the individual child and mother (/family) respectively; ln is logarithm. Let M denote mortality, Y is income at the state level (GNP), X is a vector of j other state-level variables (e.g. inequality, indicator of the business cycle, rainfall shocks) and Z is a vector of k control variables obtained from the microeconomic data (e.g. parental education, religion). State and year fixed effects are denoted $\boldsymbol{a}_s$ and $\boldsymbol{a}_t$ respectively. The elasticity of central interest is $\boldsymbol{b}$ although, as discussed, we are also interested in other macroeconomic indicators and therefore in $\boldsymbol{q}_j$. To avoid clutter, I do not show dynamics or interaction terms, though these are specified in the following section.

Logit (/probit) on micro-macro data

The basic model can be written as

$(1)\ M^{*}_{ifst} = \boldsymbol{a}_s + \boldsymbol{a}_t + \boldsymbol{b}\ lnY_{st} + \boldsymbol{q}_j\ lnX_{jst} + \boldsymbol{l}_k\ lnZ_{kifst} + \boldsymbol{n}_f + \boldsymbol{e}_{ifst}$

Here, M is a latent variable, an indicator for whether the index child i in family f who was born in time t in state s died before a specified age. For *neonatal* mortality, this is by the age of one month, for *infant* mortality it is 12 months, and for *under-5* mortality, it is 60 months. Unobservables common to siblings are represented by a family random effect, $\boldsymbol{n}_f$. This will capture factors like genetic frailty or environmental risks. Marginal effects are calculated for each observation & then averaged (rather than being calculated at means of the data); see Greene (2003). There are 163053 children located in 15 states and observed over at least 25 years. So the individual data are "nested in" a state panel.

Panel data model (on aggregated data)

$(2)\ ln\ M_{st} = \boldsymbol{a}_s + \boldsymbol{a}_t + \boldsymbol{b}\ lnY_{st} + \boldsymbol{q}_j\ lnX_{jst} + \boldsymbol{l}_k\ lnZ_{st} + u_{st}$

Now mortality is the incidence of mortality (from birth up until a specified age) in state s for children born at time t. It is in logarithms. All of the regressors are also at the state level, X being the economic variables that are at the state level in equation (1) as well, and Z being aggregates that I construct from the microdata (e.g. proportions of mothers in the state-year with different levels of education). The number of observations is at least 375, (T=25 x S=15).

Well-determined estimates of the impact of GNP on mortality conditional on fixed effects depend upon the states exhibiting substantial independent macroeconomic fluctuations across states over time. This condition appears to be met in the Indian data (e.g. Ravallion and Datt 1996, Besley and Burgess 2005)

A panel data version of the Granger-causality test is conducted, which involves testing the null that coefficients on all of the GNP terms (i.e. various lags) are *each* zero. If the sum of the coefficients on all of the GNP terms is nonzero, then there is a long-run effect of GNP on mortality. If the sum is zero, there may nevertheless be a short-run effect- in other words, mortality may depend upon the change in GNP and not on its level. As discussed earlier, distinguishing level from change effects is of some substantive interest.

Intuition and preliminary data analysis suggest a decreasing and convex relation between mortality and GNP. This is allowed by including the square of GNP in a variant of the main model.

5.2. The Variables

In the equations for under-5 mortality, Y is defined as GNP averaged over the five years of exposure of the child to mortality risk. In the case of neonatal and infant mortality, it is GNP in the year of birth. As I have information on the month of birth of the child, I will investigate if it makes any difference to use an average of GNP over the relevant exposure period, where the weights are the fraction of the child's life spent in each year. [For instance, in the case of infant mortality, the child's 12 months of exposure can span two years].

The vector X includes the following variables, although they are not all necessarily included in the same specification:[5] inequality (the Gini), positive & negative GDP shocks, ratio of manufacturing to agricultural output, relative prices, inflation in consumer prices, public spending, poverty.[6] Z includes gender, birth-order, religion, ethnicity, rural/urban, location, education of mother & father and age of mother at birth of child.

The state fixed effects control for initial conditions and for persistent elements of history, climate, culture [e.g. status of women] & other institutions [e.g. public service delivery, corruption]. The year fixed effects control comprehensively for aggregate time-variation associated with, for example, improvements in health technology, or episodes like famines, floods, epidemics.

5.3. Estimator

Specification (1) is estimated using a logit (or probit) model, with standard errors adjusted to be Newey-West. (Exogeneity of GNP was not rejected in an IV-probit using Amemiya's GLS estimator). Specification (2) uses a fixed effects panel data estimator. The benchmark estimator is within-groups OLS. This is compared with first-difference OLS and first-difference IV. A potential problem with the latter is that lagged levels of the series tend to be weak instruments for first-differences (FD) if the series exhibit persistence or if the variance of the fixed (state) effects is large relative to the variance of transient shocks at the state-year level. In this case, identification can be improved by making additional assumptions on the initial conditions of the process, and using the systems estimator (Arellano and Bover (1995), Blundell and Bond (1998)). This basically involves adding a levels equation to the FD equation and using lagged first-differences of the two series dated t-1 as instruments for the levels equations. Although GMM (Arellano and Bond 1991) is more efficient than IV (Anderson and Hsiao 1981), the latter is more appropriate given the dimensions of my

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[5] For instance, public spending and poverty are only included as a special case to investigate mechanisms by which GNP may act upon mortality.
[6] I will consider alternative poverty indices: the headcount, poverty gap, squared poverty gap, per capita mean consumption, all available separately for rural and urban areas. Although health expenditure is a natural candidate for public spending, alternative indicators considered are social expenditure, development expenditure and expenditure on food and famine relief.

panel.[7] So the preferred estimator for (2) is the systems-IV estimator. As a specification check, I also show estimates of GLS panel data models.

5.4. Specification Issues

Dynamics: I allow persistence in mortality. Any effect of GNP may not be instantaneous, taking a year or more to play out. Since the NFHS data permit us to follow cohorts of children over time, I also include lags of GNP to allow macroeconomic conditions at birth or in pregnancy to affect mortality risk in early childhood. Most related studies estimate static models (a notable exception being Ruhm 2000), which may be mis-specified. Omitted dynamics will tend to creep into the error term, but previous studies typically do not test for residual autocorrelation (see Bertrand et al (2004) for a similar critique).

Heterogeneity in the growth elasticity of mortality is investigated both by including interaction terms and by estimating the model on sub-samples of the data (inequality, education, gender, caste, birth-order).

Endogeneity: A causal effect running from child mortality to GNP seems unlikely *a priori.* However, it cannot be ruled out. In microeconomic studies, individual income is endogenous to health because healthier people tend to earn more. Direct feedback of this sort is eliminated by focusing on child health (e.g. Case *et al* 2005). However, there remains the problem that both health and income may be jointly affected by unobserved heterogeneity, inducing simultaneity. Using aggregate income to explain individual mortality, as in this study, avoids this problem.

In panel data models run on aggregates, endogeneity is mitigated by using fixed effects estimators (as in Ruhm 2000), but there remains the possibility that mortality and aggregate output are simultaneously subject to idiosyncratic shocks. For example, a rainfall shock or high tobacco use may simultaneously reduce GNP and increase child mortality. These effects are minimised in this analysis by including such variables as regressors. I also try instrumenting GNP with its lags (Anderson and Hsiao 1981). Over-identifying restrictions associated with rainfall shocks and adult education are investigated using Hansen's J statistic- they turn out to be valid although not to have much additional power.

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[7] A nice survey of alternative estimators for dynamic panel data models is Bond (2002).

Selectivity: A problem with retrospective data, when an age cut-off is used to select the interviewees, is that they may be an unrepresentative sample for earlier years. This is addressed by conditioning on maternal age at birth, and dropping children born in the 1960s as they are not just unrepresentative but few. To further assess whether the main results are sensitive to the data structure, I included interaction terms between all trended variables (like education) and year (Rindfuss et al 1982). Finally, I estimate the model on a sample of more recent data, selecting the sample by looking at the distribution of mother's age by birth year of child.

Adjustment of Standard Errors: In specification (1), the variance-covariance matrix for the error term may be block diagonal, the error terms within each state-year cell being correlated. For example, economic shocks may affect all individuals in a state on an annual basis. I adjust the standard errors for this sort of clustering, failure to do which can bias inference (see Moulton (1990), Donald and Lang (2001)). Another solution to this problem that is used in the literature is to collapse the data to the group level of aggregation (state-year cells). This corresponds to my specification (2). A further issue that is pertinent to long panels is of serial correlation in the errors. Although this will be reduced by my modelling dynamics, I also test for it and compare block bootstrapped with Newey-West standard errors (allowing for an unrestricted covariance structure over time within states, with or without assuming that the error process is the same across states); see Arellano (2003). In a survey of applied research that uses difference-in-difference estimators, typically on US panel data, Bertrand *et al* (2004) remark that most do not adjust for clustering or autocorrelation. Indeed, properties of the error term are not discussed in Pritchett and Summers (1996), the only available guide to the impact of GNP on mortality in developing countries.

Aggregation: In comparison of specifications (1) and (2), I replace the logit with a linear probability model. The comparison is a useful exercise because previous research uses cross-country data without comparable micro-data analysis. The aggregate model may be less subject to measurement error bias, but more subject to heterogeneity bias. Also, if the effect of micro-demographic controls is non-linear, then simple averages of these variables in the aggregate model will fail to capture the desired effect.

6. Results

The unconditional elasticity of under-5 mortality with respect to aggregate income (GDP) is –0.71, significant at the 1%. level[8] Once time and state dummies are included in the model, this falls to –0.59, and remains significant (Table 3). The other rows of Table 3 show that this elasticity is fairly robust to inclusion of other covariates, including public health expenditure and poverty (see section 1).

Precise definitions of the all covariates are in Bhalotra (2006). Here, I summarise the main findings. The state and time dummies are each jointly significant at the 1% level. Conditional on state and time effects, within and between sector inequality, poverty, relative prices (agriculture relative to industry) and inflation are all insignificant. The sectoral composition of GDP is significant. In particular, agricultural growth has a greater mortality-reducing effect than non-agricultural growth. So, at a given level of total GDP, the relative growth rate of the agricultural sector takes a significantly negative coefficient in the mortality equation. In contrast, analyses of the effect of sectoral shifts in GDP on poverty in India find that the greater impact has flowed from non-agricultural growth (see Besley et al 2005, Ravallion and Datt 2002). Public expenditures on health and family welfare have a significant mortality-reducing effect only at high levels of expenditure. A disaggregate analysis, discussed in more detail in Bhalotra (2006), shows that this effect is significant only in four of the fifteen states, these being Uttar Pradesh, West Bengal, Tamil Nadu and Maharashtra. The only significant compositional effects in the model are secondary-level education amongst fathers, which reduces child mortality, and belonging to a Scheduled Tribe, which increases mortality.

Differences in the growth elasticity across the states

I allow the coefficient on GDP to be state-specific by interacting GDP with state dummies. I find that it is significant in only 8 of the 15 states (see Table 4). In these eight states, the elasticity varies between -0.5 and -0.9, with the exception of Kerala, where the elasticity is a remarkable -1.7. Comparing state GDP effects with state fixed effects, we find that the states that were relatively ineffective in translating growth into lower mortality (i.e. states with a small absolute elasticity) were not those

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[8] This happens to be almost exactly the same as the unconditional elasticity reported for the UK and the USA in Deaton and Paxson (2004).

with an inherently high mortality risk (i.e states with large fixed effects).[9] This is encouraging for policy because it suggests that the states in which growth does not significantly reduce mortality can more easily make their growth "pro-poor" (i.e. mortality-reducing) than would be the case if their observed inefficacy were tied to the sorts of persistent historical or institutional factors that state fixed effects tend to capture.

Was the growth elasticity larger in the post-reform era?

The average growth rate of GDP per capita was barely 1% p.a. in the 1960s and 70s but, since the early-1980s and especially since about 1993, it has been distinctly higher, averaging 4.8% p.a. between 1993/4 and 1999/00. The upturn in the growth rate coincided with the onset of economic liberalization in India. A gradual process of reform was set in motion in the early to mid-1980s and this accelerated in the 1990s. Whether the reforms caused higher growth, and how, is debatable (see Virmani 2004, Clark and Wolcott 2002, DeLong 2002, Bhalotra 1998) and, in any case, is not the subject of this paper. However, it is interesting to investigate whether the additional growth and the structural change associated with reform altered the growth-elasticity of mortality. For a review of concerns about the impact of structural adjustment on mortality, see Hill and Pebley (1989). Their discussion underlines that the effect can go either way, making this an important question to investigate empirically.

To do this, I split the sample at 1981. A break-point in 1980/1 or 1981/2 is indicated by the analysis in Virmani (2004), who tests for structural breaks in GDP growth in India over the period 1950-2002. Possibly relevant is that the Congress party returned to power in 1980/1, initiating a new approach to economic management in view of growing awareness of the growth-inhibiting constraints of its earlier regime. Table 2 summarises rates of growth of GDP and rates of decline in mortality for the two periods created by a break in 1981. It is clear that, even as the GDP accelerated, mortality decelerated.

Refer Table 5, where row 1 reports the benchmark estimate of -0.60 from row 4 in Table 3. Rows 2 and 3 show the "pre-reform" and "post-reform" elasticities to be -0.82 and -0.44 respectively, and I am able to reject the null that these are equal at the

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[9] A similar result is reported in World Bank (2004).

10% significance level.[10] This result suggests that the Indian reforms were anti-poor (childhood mortality is concentrated amongst the poor: see Victora *et al* 2003, for example). In fact, since the mortality rate is bounded, we may expect the elasticity to decrease as the level of mortality decreases even in the absence of any structural change. This is especially the case since, as the incidence of under-5 mortality declines, the fraction of neonatal deaths in all under-5 deaths tends to rise, and neonatal deaths are less closely tied to fluctuations in GDP. All that can be safely concluded is that the post-81 period was not associated with growth becoming evidently more pro-poor than before.

7. Conclusions

(to be extended in next draft of paper)

Cyclical fluctuations do impact on infant and under-5 mortality. The impact varies across regions, indicating the importance of initial conditions such as inequality (e.g. Ravallion 2005), and of political variables. Also, a given level of growth is consistent with different rates of mortality reduction, indicating the importance of other factors that are unrelated to growth.

Policies that increase the relative growth rate of the agricultural sector will contribute to reducing mortality. After the mid-1970s, agricultural income has grown much more slowly than non-agricultural income in India (e.g. Besley et al 2005). The analysis in this paper suggests that this has constrained reductions in mortality. Public health expenditure only has a beneficial effect on mortality at high levels of expenditure. Although this study does not investigate the allocation of public expenditure, it is clear from previous research (e.g. World Bank 2004) that the composition of expenditure and its effective delivery are key to its effectiveness. Time-varying unobservables that most likely reflect technological change (e.g. medical progress) and improvements over time in health services have contributed

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[10] The regression for 1982-94, like the regression for the full period, 1970-94, allows every child in the sample full exposure to the risk of under-5 mortality, and the GDP variable corresponding for births in 1994 is the average of GDP over 1994-99. To similarly allow for full exposure for every child in the period 1970-81, I re-estimate this model on data for births in 1970-77, with death rates for births in 1977 being modeled as a function of GDP averaged over 1977-81. The pre-reform elasticity is now –1.37 rather than –0.81, and its difference from the post-reform elasticity of –0.44 is significant at the 5% level.

significantly to mortality decline, and to the convergence of mortality rates across the Indian states.

Five Indian states account for more than half of all childhood mortality (World Bank 2004). Interventions need to be concentrated in these states. Although this was not specifically investigated in this study, the data show that under-5 death probabilities are higher amongst girls, first-born children, and children of scheduled-tribes. Targeting these relatively vulnerable groups will bring down average mortality incidence.

In this study, I interacted GDP with state dummies to obtain state-specific growth elasticities from a panel data model. In work in progress, I replace the state dummies with a vector of variables denoting initial conditions such as female literacy and the initial level of inequality. I will further investigate how the welfare gains from growth may depend upon inequality, media activity and political representation, all of which may be expected to influence the pro-activeness of the state government.

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Table 1
Level and Change of Under-5 Mortality and GDP:
All-India and States: 1970-1998

State	Mean mortality (%)	s.d. (income)	Linear rate of change in mortality p.a. (%)	Linear rate of change in income p.a. (%)
Andhra	10.9	0.36	-3.4	3.8
Assam	7.8	0.23	-1.7	2.6
Bihar	12.1	0.20	-3.0	1.8
Gujarat	12.0	0.38	-3.8	3.6
Haryana	9.3	0.41	-2.5	3.0
Karnataka	10.8	0.31	-4.1	3.3
Kerala	4.8	0.32	-7.1	3.0
Madhya	17.5	0.30	-3.6	3.1
Maharashtra	8.7	0.41	-4.9	4.4
Orissa	14.2	0.30	-3.3	3.1
Punjab	6.9	0.35	-2.1	2.8
Rajasthan	15.2	0.25	-3.2	2.2
Tamil Nadu	9.9	0.41	-5.3	4.2
West Bengal	9.8	0.27	-5.1	2.7
Uttar Pradesh	17.3	0.21	-4.4	2.0
All India	**11.1**	**0.31**	**-3.9**	**3.0**

Notes: Income is the logarithm of real per capita net state domestic product. The means and standard deviations (s.d.) are for the period 1970-98 for each region. The growth rates in the last two columns are obtained as coefficients in a regression of the mortality rate or log income, as the case may be, on a linear trend term. The rank correlation coefficient between mortality and lnGDP is **–0.50.**

Table 2
Changes in Under-5 Mortality and GDP:
All-India and States: 1970-1981 & 1982-94

State	Under-5 Mortality		Aggregate Income	
	1970-81	1982-94	1970-81	1982-94
Andhra Pradesh	-1.79	-0.94	1.39	4.86
Assam	-4.28	-1.58	2.49	1.30
Bihar	-4.77	-0.66	1.30	1.36
Gujarat	-6.77	-3.05	3.14	3.02
Haryana	-1.98	-2.57	2.30	3.11
Karnataka	-2.78	-3.56	1.58	3.74
Kerala	-9.46	-9.86	3.07	3.00
Madhya Pradesh	-2.03	-2.36	1.53	2.80
Maharashtra	-6.80	-2.63	4.66	4.58
Orissa	-4.49	-2.82	1.68	2.93
Punjab	-0.59	-0.66	2.25	3.39
Rajasthan	-3.39	-1.28	-0.73	2.75
Tamil Nadu	-6.14	-4.03	1.74	5.46
West Bengal	-4.31	-4.58	1.62	2.01
Uttar Pradesh	-5.50	-2.69	1.25	1.41
ALL INDIA	-4.40	-2.88	1.95	3.05

Notes: These are linear rates of growth obtained by a simple regression of the log of mortality or GDP, as the case may be, on a trend. The All-India regression includes a set of state dummies. All figures are percentages.

Table 3
The Growth Elasticity of Under-5 Mortality
Alternative Sets of Control Variables

	Covariates other than log GDP	Growth Elasticity	t-statistic
1	None	-0.71	13.8
2	State dummies	-0.97	12.4
3	Year dummies but no state dummies	-0.51	6.6
4	State & year dummies	-0.59	3.5
5	+ inequality (gini)	-0.55	3.5
6	+ health expenditure	-0.51	3.1
7	+ poverty gap index	-0.56	3.2
8	+ sectoral composition of GDP, relative prices, price inflation, GDP shocks	-0.64	3.0
9	+ maternal age at birth, maternal & paternal education, gender, ethnicity, religion	-0.50	2.2

Notes: The additional regressors shown are cumulative. In other words, unless otherwise indicated, row j has all of the regressors shown in row j-1 and also those named in row j. Precise definitions of the covariates are in a companion paper, Bhalotra (2006). The absolute t-statistics reported in the last column are based on Newey-West standard errors that allow for heteroskedasticity and autocorrelation.

Table 4
Growth Elasticities and Fixed Effects by State

	Elasticity	Fixed Effect
Andhra Pradesh	-0.20	0.00
	[1.03]	n.a.
Assam	-0.05	-0.44**
	[0.15]	[7.18]
Bihar	-0.36	-0.08
	[0.76]	[0.40]
Gujarat	-0.53*	0.24*
	[1.99]	[2.41]
Haryana	-0.18	-0.13
	[0.66]	[1.02]
Karnataka	-0.57*	0.04
	[2.14]	[1.07]
Kerala	-1.69**	-0.97**
	[4.03]	[11.71]
Madhya Pradesh	-0.24	0.43**
	[1.01]	[9.24]
Maharashtra	-0.71**	0.035
	[3.43]	[0.45]
Orissa	-0.56*	0.20**
	[2.35]	[4.57]
Punjab	0.77	-1.11
	[0.91]	[1.69]
Rajasthan	-0.27	0.23**
	[0.91]	[2.67]
Tamil Nadu	-0.72**	-0.048
	[3.01]	[1.31]
West Bengal	-0.89**	0.12
	[2.94]	[1.69]
Uttar Pradesh	-0.94*	0.40**
	[2.51]	[7.95]

Notes: The reported figures are estimates from a model of under-5 mortality that includes additive state and year fixed effects, and interacts GDP with each of the 15 state dummies. The elasticity is significant in only 8 of the 15 states. The fixed effects coefficients are relative to Andhra Pradesh (normalized to zero), and 7 states are estimated to have significantly different fixed effects as compared with Andhra. Absolute t-statistics are in brackets and significance is indicated by asterisks, * denoting significance at the 5% level and ** denoting significance at the 1% level.

Table 5
Was There A "Structural Break" in the Growth Elasticity?

	Sample	Elasticity	t-stat
1	1970-1994 (entire period)	-0.59	3.5
2	1970-1981 ("pre-reform")	-0.82	2.8
3	1982-1994 ("post-reform")	-0.44	1.9

Notes: The dependent variable is the log of under-5 mortality, as in Tables 3 and 4. The equations includes state and year fixed effects. Standard errors are Newey-West. The elasticities -0.81 and -0.44 are significantly different (F(1,167)=2.7, p>F=0.103).

Figures

Figure 1: Trends in Under-5 Mortality
Figure 1a: Trends in Under-5 Mortality by State
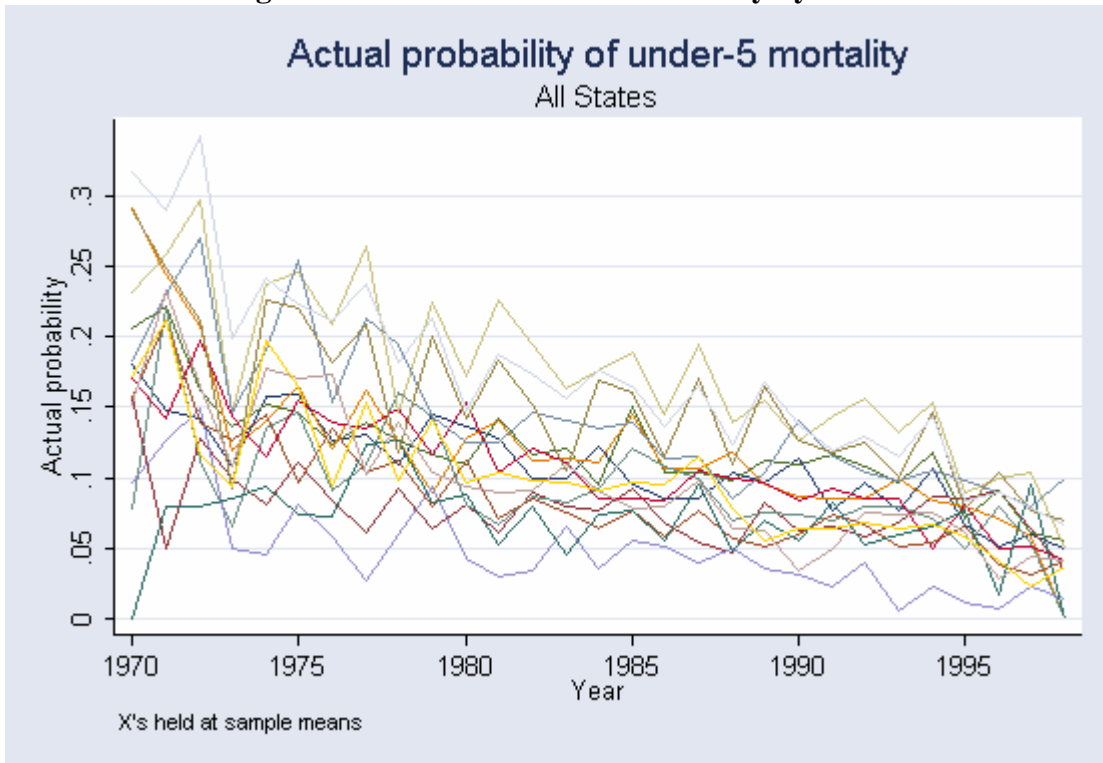
Figure 1b: Trend in Under-5 Mortality, All India (Weighted Average)
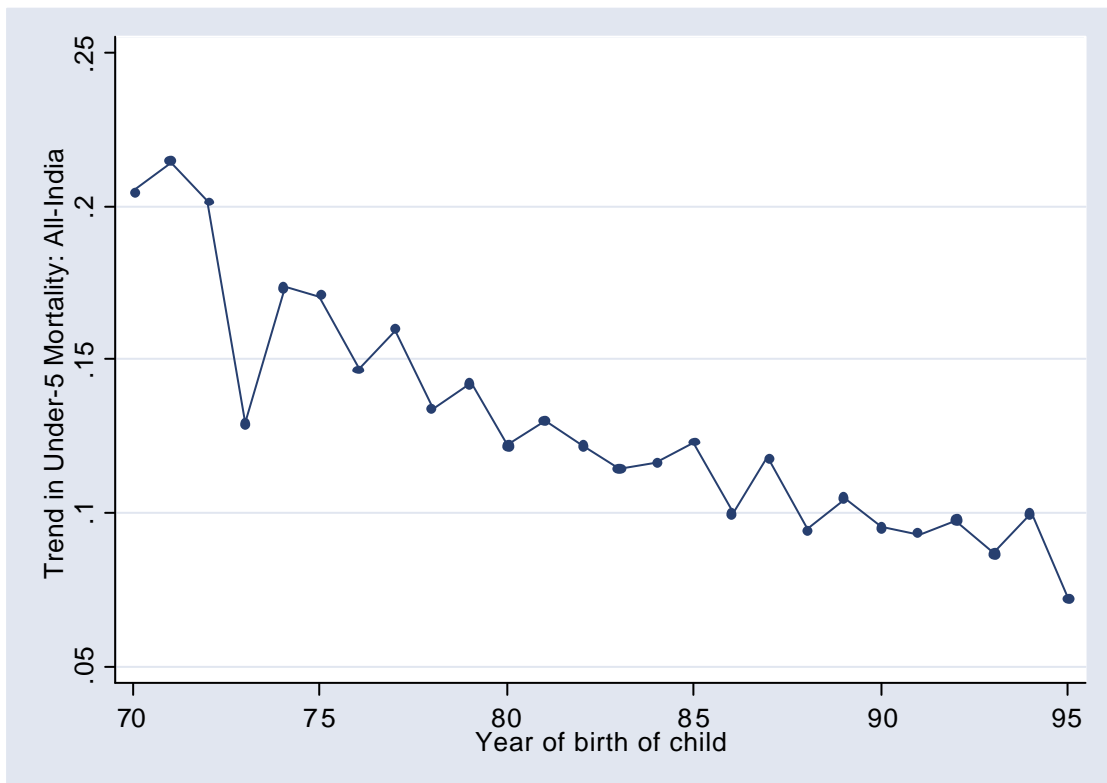
Figure 2: Trends in ln GDP and D_5 ln GDP: By State, 1970-1995
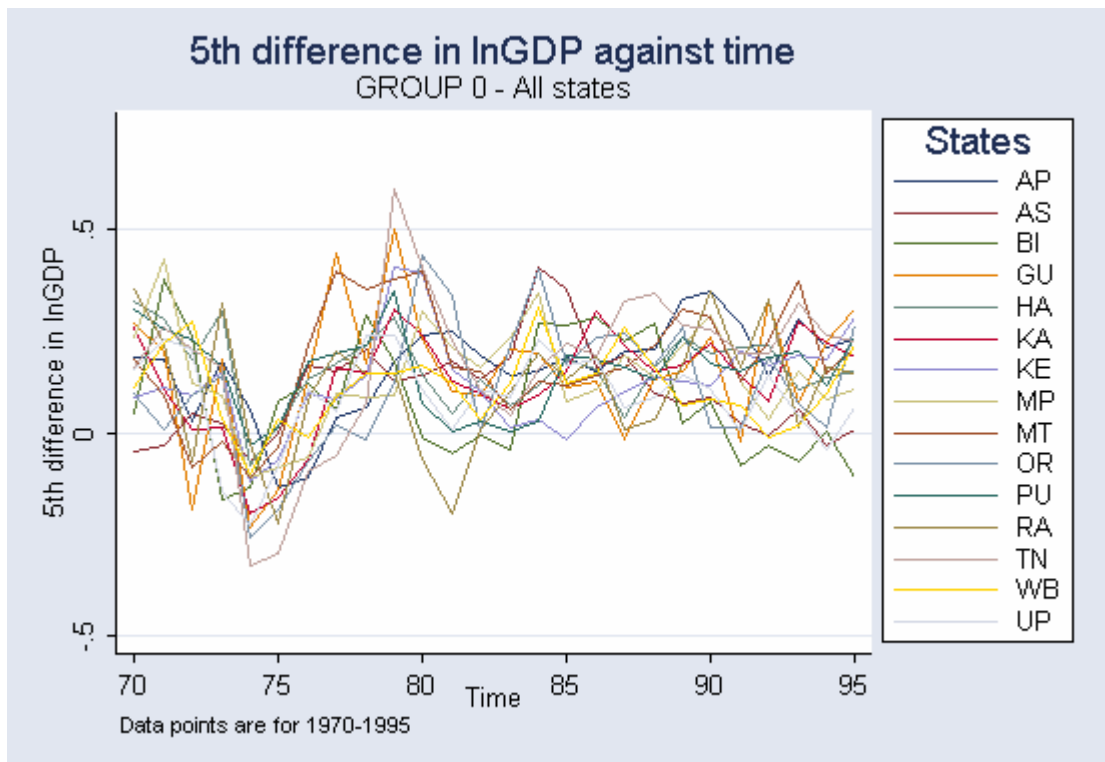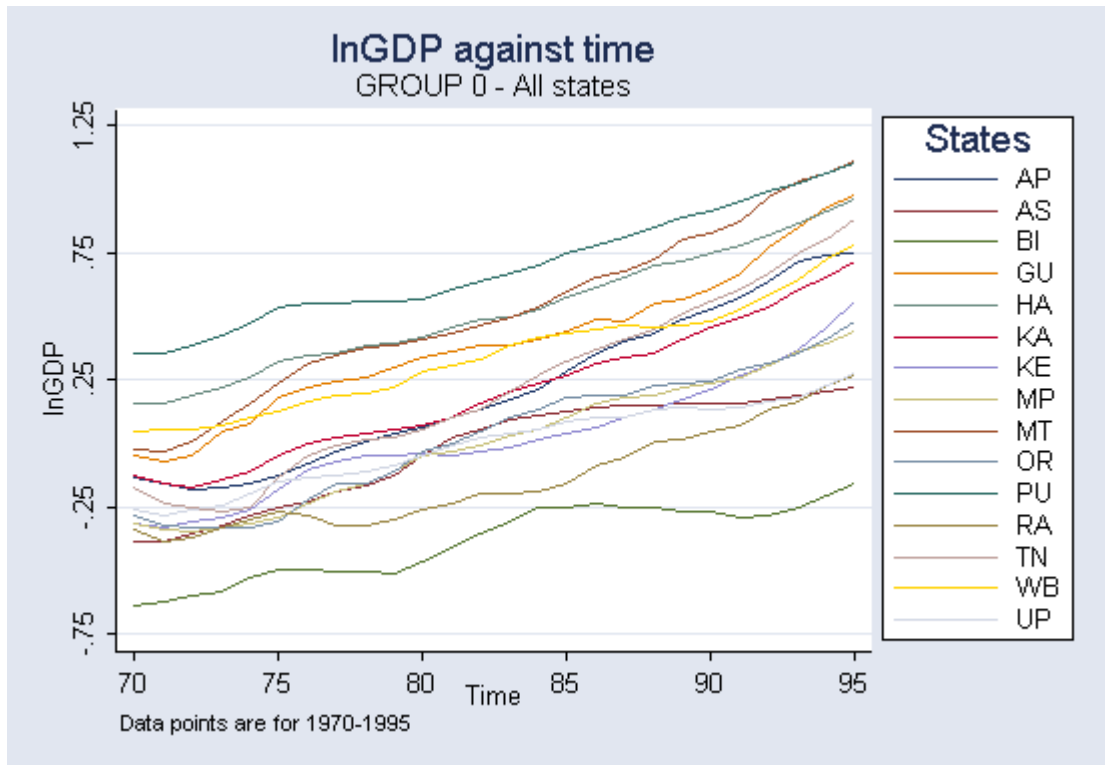
**Figure 3: Under-5 Mortality and Ln GDP p.c., both Against Time
All-India: Population-Weighted Averages, Time-Variation**
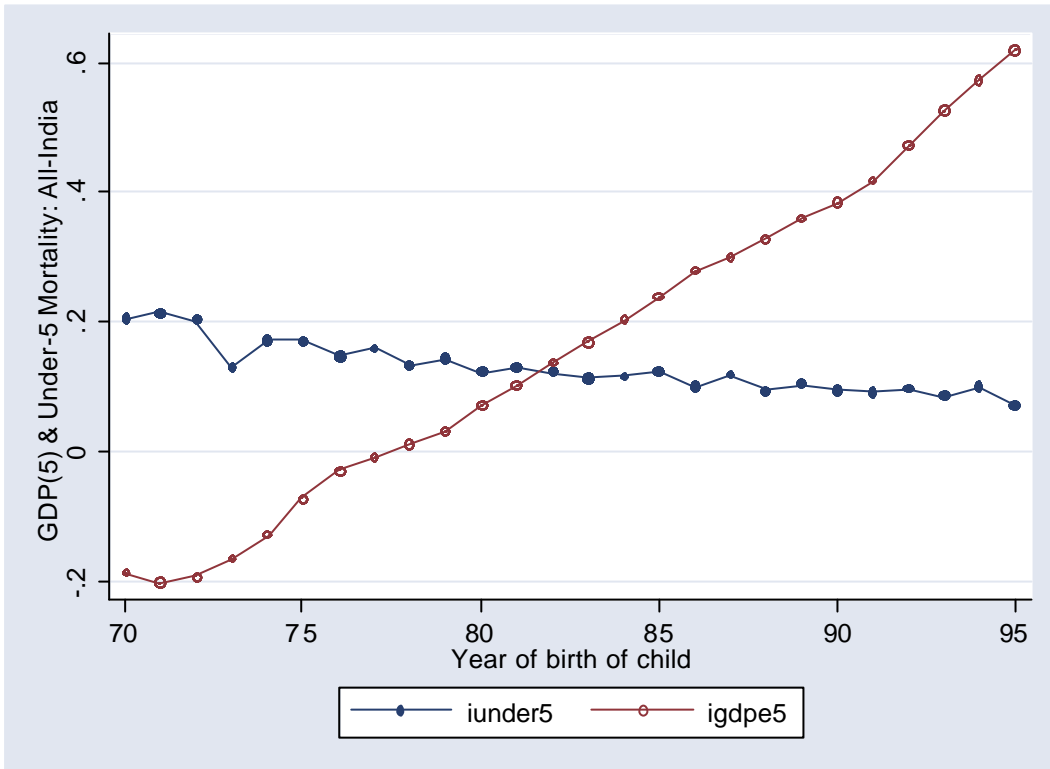
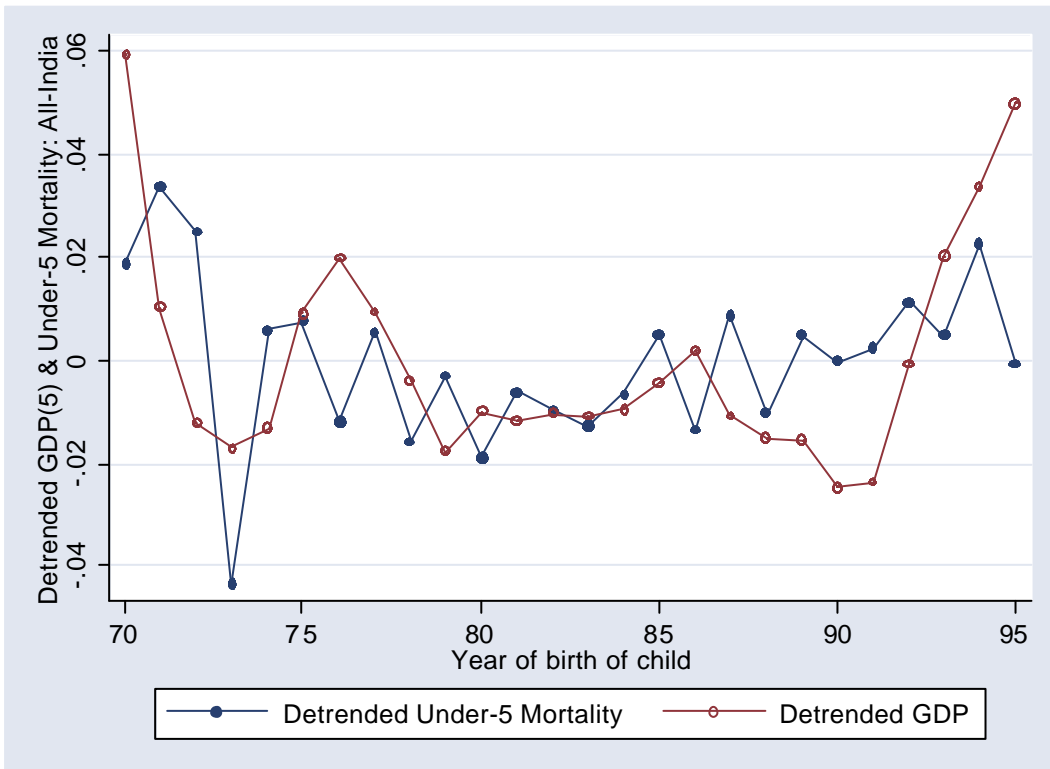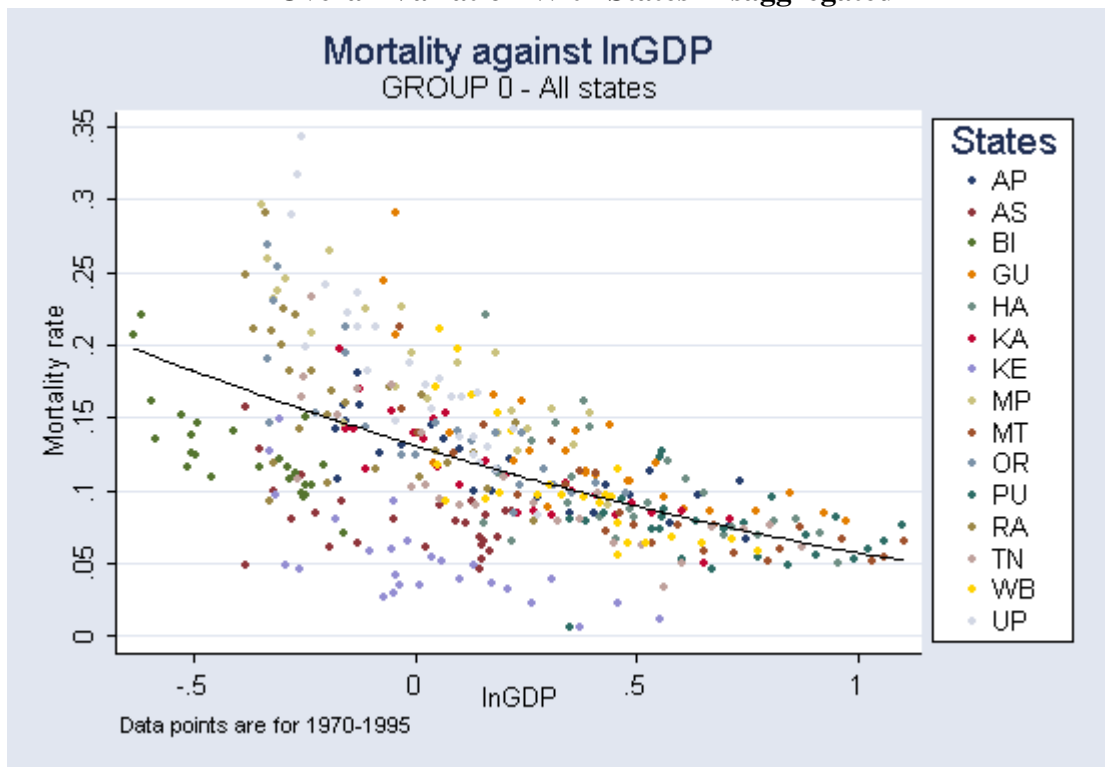**<u>Detrended Series (M$_{st}$-M$_t$ & Y$_{st}$-Y$_t$)</u>**

Figure 4: Under-5 Mortality Against GDP

Overall Variation With States Disaggregated

Figure 4b: Mortality Against Ln GDP:
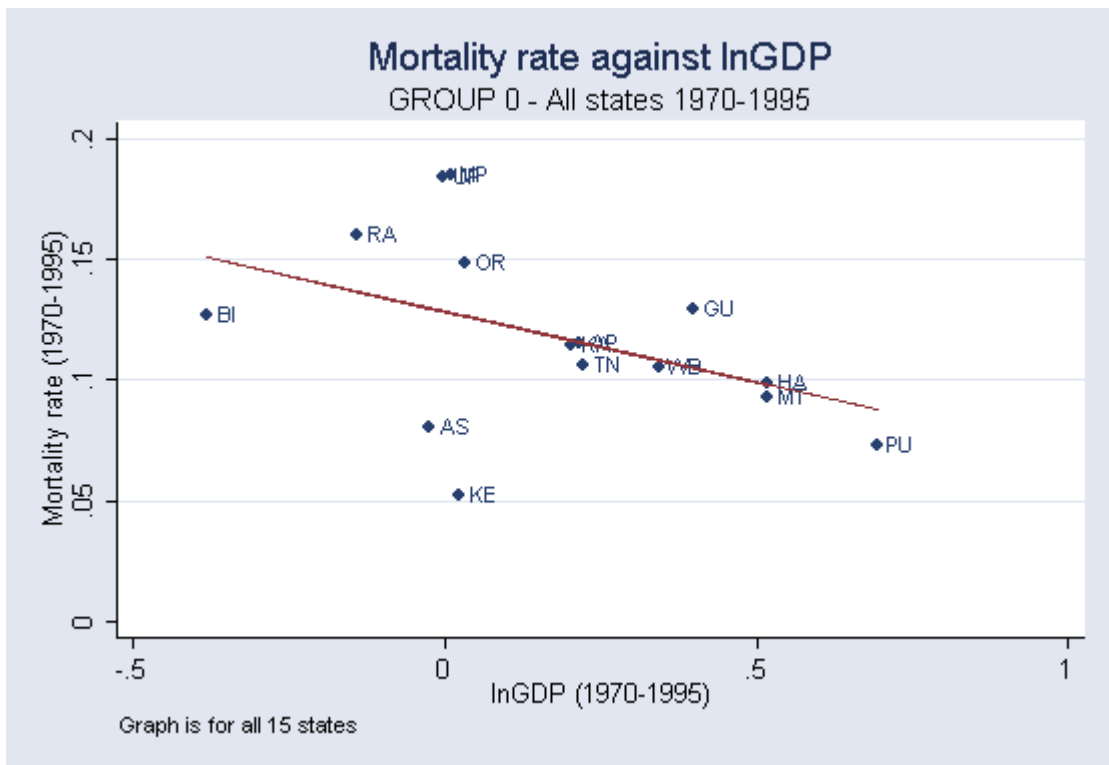
State-Variation (or Between-Variation)
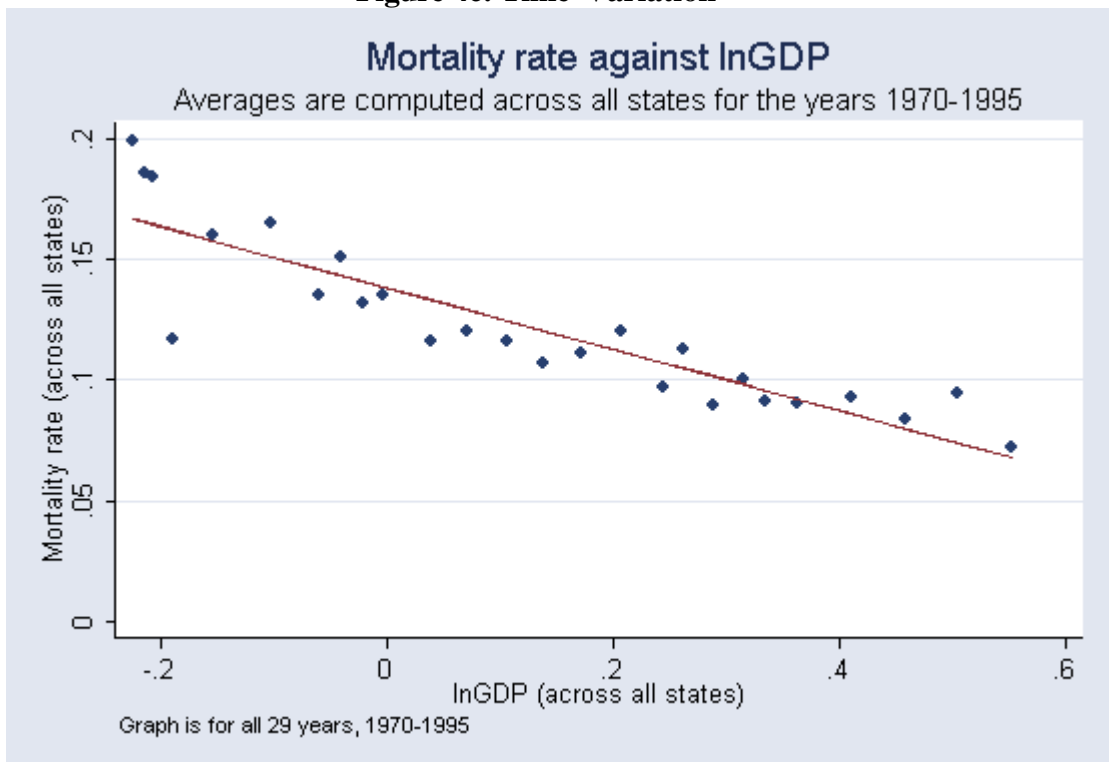

Figure 4c: Time -Variation
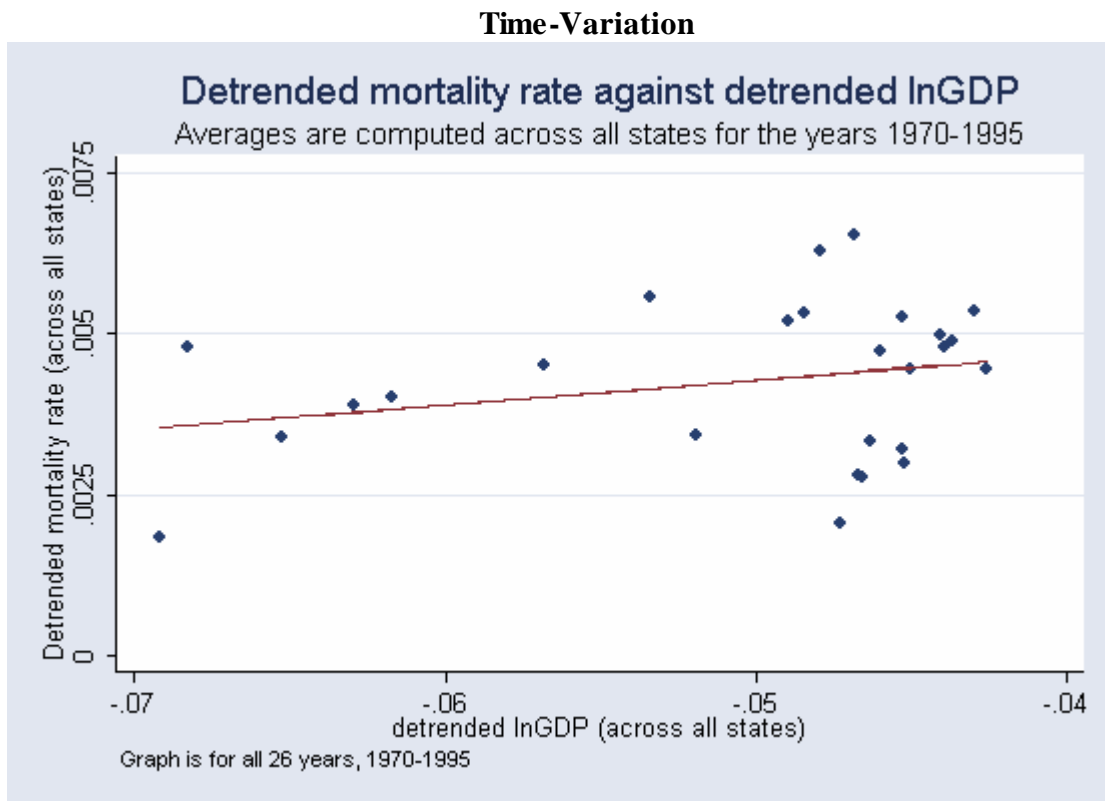
Figure 5a: Mortality Against Ln GDP: *Both Series Detrended*

Time-Variation

Detrended mortality rate against detrended lnGDP
Averages are computed across all states for the years 1970-1995

Figure 5b: State-Variation

Detrended mortality rate against detrended lnGDP
GROUP 0 - All states 1970-1995
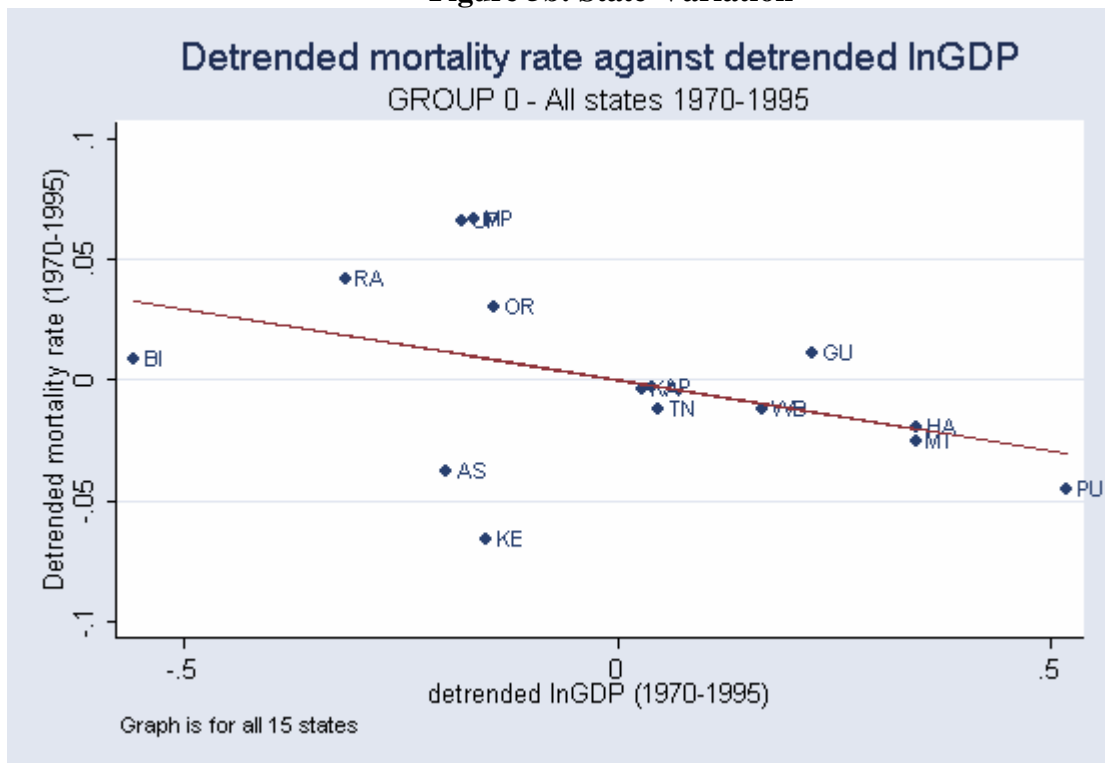
Figure 6: Relationships By Decade
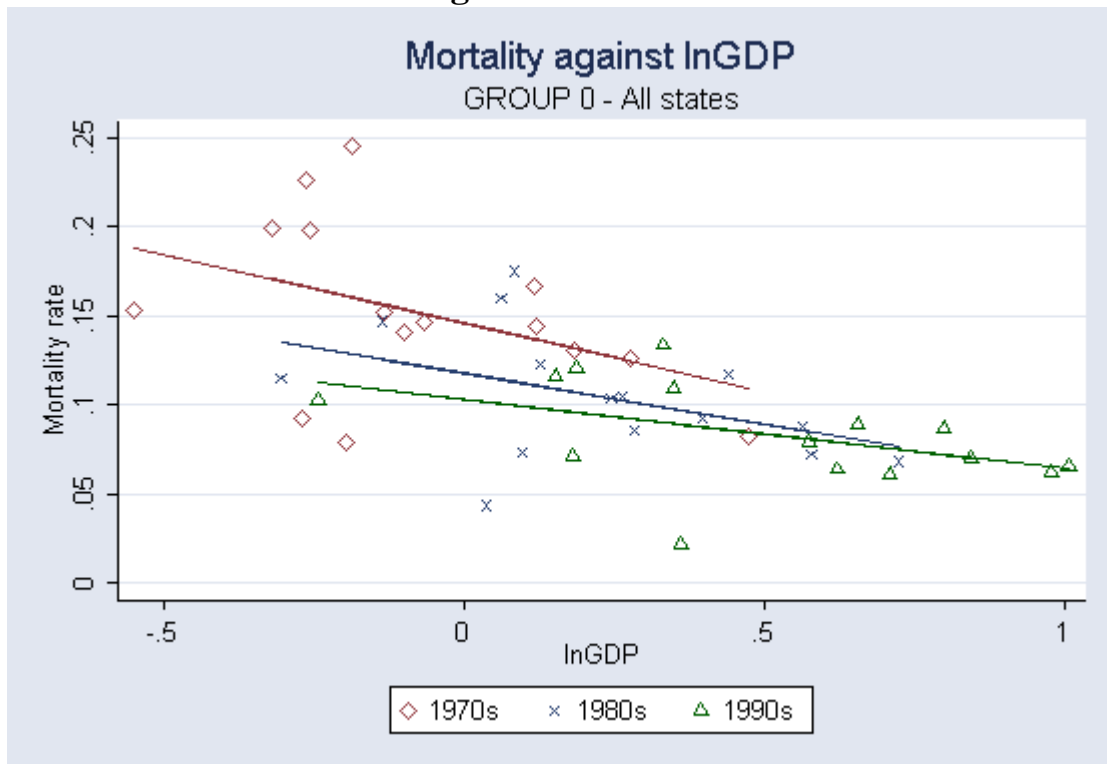
Figure 6a: Ln GDP

Mortality against lnGDP
GROUP 0 - All states

Figure 6b: **Detrended Series**

Detrended mortality against detrended lnGDP
GROUP 0 - All states