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Fatal fluctuations? Cyclicality in infant mortality in India

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ABSTRACT

This paper investigates the impact of aggregate income shocks on infant mortality in India and investigates likely mechanisms. A recent OECD-dominated literature reports the provocative finding that mortality at most ages is pro-cyclical. Similar analyses for poorer countries are scarce, and both income risk and mortality risk are greater in poor countries. This paper uses data and methods designed to avoid some of the specification problems in previous studies and it explores mechanisms and extensions that have not been previously considered. It uses individual data on infant mortality for about 150,000 children born in 1970–1997, merged by cohort and state of birth with a state panel containing information on aggregate income. Identification rests upon comparing the effects of annual deviations in income from trend on the mortality risks of children born at different times to the same mother, conditional upon a number of state-time varying covariates including rainshocks and state social expenditure. Rural infant mortality is counter-cyclical, the elasticity being about −0.33. This is despite the finding that relatively high-risk women avert birth or suffer fetal loss in recessions. It seems in part related to recessions stimulating distress labor amongst mothers, in contrast to the case in richer countries, where they discourage labor market participation. Health-care seeking declines in recessions, and this appears to be related to the opportunity cost of maternal time. Disaggregation reveals that the average results are driven by rural households in which the mother is uneducated or had her first birth in teenage, and that it is only girls that are at risk; boys are protected from income shocks. Exposure to poor conditions in the fetal and neonatal period appears to have a larger effect on infant mortality than similar exposure in the postneonatal period.

1. Introduction

In poor countries, about 30% of all deaths occur in childhood compared with just 1% in richer countries (Cutler et al., 2006). The proximate cause of these excess deaths is infectious disease combined with inadequate nutrition (Jones et al., 2003). A question of long-standing policy interest concerns the extent to which improving incomes (or reducing poverty) will lower the risk of childhood death. Since longer-range growth is often entangled with a lot of other change — in technology, education, infrastructure and political and social institutions — the effects of income are more easily isolated by studying the effects of short-range changes. This paper studies the effects of annual fluctuations in the state-level economy in India on infant mortality.

This is topical in view of recent provocative evidence that mortality rates, for adults and infants alike, are pro-cyclical; see Ruhm (2000) and Dehejia and Lleras-Muney (2004), who analyse panel data for the United States.[1] A behavioural explanation offered to support this result is that, in recessions, when the opportunity cost of time is low, people (mothers in the case of infant mortality) substitute their time away from the labor market and towards health-preserving activities, and this substitution effect overwhelms the income effect, so that the net effect on health is positive. However, absent any behavioural mechanism, this outcome may arise on account of selectivity in the sample of births. In particular, if adverse shocks induce women to defer fertility, and this effect is stronger amongst women with inherently higher risks of infant mortality, then the composition of births in a recession will be selectively low-risk (Dehejia and Lleras-Muney, 2004). This paper provides evidence on both the behavioural and selection effects, using microdata on infant mortality. It argues that a similar selection effect, hitherto neglected in the literature, arises if higher risk women are disproportionately subject to fetal loss during recessions.[2] Fetal loss (spontaneous abortion or stillbirth) may be more relevant than conscious deferral of fertility in societies where women are under-nourished and have limited control over birth-spacing.

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[1] This literature uses the terms "business cycle" and "recessions" to refer to deviations of the unemployment rate from trend. Here recessions are deviations of aggregate income from trend. Given the informal nature of labor markets in poor countries and survival constraints on unemployment amongst the poor, aggregate income fluctuations are not as clearly mirrored in unemployment as in richer countries.

[2] In other words, there is the same problem at birth as at later ages of having to condition upon survival until the start of the exposure period. Dehejia and Lleras-Muney suggest selectivity in the timing of *births* and I suggest that, in addition, there is selectivity in the timing of *deaths*. Both will influence the composition of live births.

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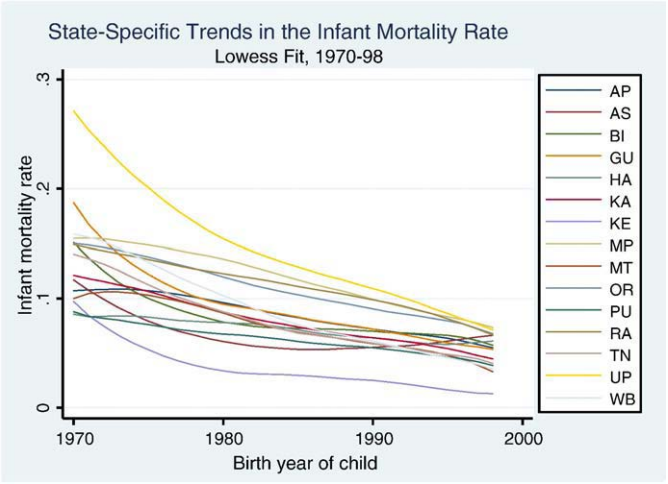

Fig. 1. State-specific trends in the infant mortality rate.

The questions analysed are especially relevant in poorer countries, where income volatility tends to be greater, and cyclical fluctuations larger and more abrupt (Pritchett, 2000; Koren and Tenreyro, 2007). The consequences of income variation are also likely to be more severe as neither states nor markets provide much insurance against shocks. State social expenditure tends to decline when aggregate income falls (Frankenberg et al., 1999). At the same time, a weaker financial infrastructure makes it difficult for individuals to borrow to smooth fluctuations in their incomes (Koren and Tenreyro, 2007). As a result, households resort to other, often more costly sources of insurance (Morduch, 1995). Of particular interest here are adjustments induced by macroeconomic shocks to fertility and maternal labor supply, as these adjustments may directly impact upon the health and survival of children.

The analysis uses individual data on infant mortality for about 152,000 children born to some 50,000 mothers across the 15 major Indian states in 1970–1997. The microdata are merged by cohort and state of birth with a state-level panel containing information on aggregate income. The essence of the research strategy is to compare the risk of dying in infancy of siblings, one born in a recession and one not. The micro-panel (of children within mother) is exploited to control for endogenous heterogeneity in the composition of live births, which is pertinent if either of the selection mechanisms described above prevails. I also directly analyse cyclicality in the composition of births. The state panel (years within state) is exploited to control for state-level heterogeneity and omitted trends in a more flexible way than in previous studies. The microdata are further exploited to investigate heterogeneity in the income effect, which helps confirm identification since any omitted variables would have to behave differently for different slices of the data in order to exhibit the sorts of heterogeneous effects that we find. The analysis is extended to investigate asymmetry in the income effect and to try to identify its timing in fetal and infant life. In contrast to most related studies, this paper allows dynamics and investigates the possibility that the results are driven by stochastic trends.

I find that recessions increase rural infant mortality and booms decrease it; I cannot reject symmetry in this effect. An income shock of median size (4.4%) is estimated to raise infant mortality by 0.136 percentage points. This is almost half of the total annual decline in mortality in India in 1970–99 (which I estimate at about 0.3%-points p.a.). As the median positive income shock in these data, at 5.8%, is larger, the simulated beneficial effects of booms are accordingly larger. The effects of income fluctuations are not evenly distributed. Children of uneducated or teenage mothers are most vulnerable. Within households, girls are more vulnerable than boys, reinforcing previous findings that girls' welfare is put second to that of boys in lean times. There is some indication that income in the fetal and neonatal stages of life has a larger effect on infant mortality than income in the postneonatal period.

Pro-cyclicality of child survival is associated with counter-cyclicality in maternal labor supply, especially amongst women who work in agriculture. Recessions in India stimulate distress work, in contrast with richer countries, where they dampen economic activity. This effect is concentrated amongst rural mothers, amongst whom a 5% decrease in state income is associated with a 4.7% increase in agricultural work participation. Antenatal and post-natal health seeking are lower in recessions, and lower amongst mothers who work in agriculture. To complete the causal chain, the paper shows that there is a strong negative correlation between maternal labor supply and a range of child health and survival outcomes. There is no similar effect of father's labor supply. So it seems that maternal time is an important determinant of the demand for inputs into child health. State health and development expenditure decline in recessions and despite the efforts of mothers to insure consumption, average household consumption also falls, illustrating failure to achieve full insurance.

Overall, the results indicate that cyclical variation in the relative price of child health — *via* maternal time — reinforces direct income effects. We further identify evidence that lower-risk women are more likely than others to produce live births in a recession. To the extent that this is a result of conscious deferral of fertility on the part of high-risk women, rather than a consequence of spontaneous fetal loss, it suggests that fertility, if not labor supply, is timed to avert infant mortality.

1.1. Previous evidence and Contributions

Previous evidence of negative income effects in developing countries is not overwhelming. Pritchett and Summers (1996), Cutler et al. (2002) and Baird et al. (2007) report a negative impact but Palloni and Hill (1997) argue that short-term mortality responses to recessions in the second half of the 20th century in nine Latin American countries were erratic, and statistically insignificant (also see Deolalikar, 2005, chapter 2). Other researchers have found positive associations between income and mortality in Chile, Argentina and Colombia, for example (Ortega and Reher, 1997; Abdala et al., 2000; Miller and Urdinola, 2007). For a recent review, see Ferreira and Schady (2008).

Most studies of income effects on mortality in developing countries have exploited macroeconomic crises or big one-off shocks, with mixed results (e.g. Paxson and Schady, 2005; Miller and Urdinola, 2007). These crises are often associated with a collapse of public expenditure, so the role of liquidity constraints at the household level is unclear. Moreover, the effects of big one-off shocks are likely to be different from the effects of smaller more regular fluctuations, limiting the external validity of these results.[3] In their seminal paper, Pritchett and Summers (1996) use a cross-country panel for 58 developing countries. As they themselves state, the international mortality statistics that they use are unreliable (also see Ross, forthcoming). Also, international statistics are, at best, quinquennial, smoothing over some of the fluctuations that we are

[3] In general, smaller negative shocks will tend to generate smaller adverse effects but, at the same time, they tend to stimulate less of a reaction amongst governments and donors, so that their net impact may be more or less adverse. Interventions following crises are of a more specific one-off nature, easier to finance, and more likely to be consistent with political incentives, given heightened media activity around a crisis, and the relative ease with which the impact of an intervention can be observed (see Sen, 1981; Besley and Burgess, 2002). In a different context, Adda et al. (2006, pp. 20) observe that the effects of big changes in income may be different from the effects of smaller changes.

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interested in. The use of consistent microdata on mortality from a single survey in this paper is a considerable improvement. Baird et al. (2007) similarly exploit survey data from several developing countries, and they find similar results.[4]

This paper contributes to the literature by improving controls for potential confounders and by analysing mechanisms. It systematically controls for the potentially endogenous composition of births in analysing infant mortality, while also directly investigating this using observable indicators of risk like education and ethnicity. Although Dehejia and Lleras-Muney (2004) motivate this, their analysis of infant mortality is conducted using a US state panel. They present mother fixed effects estimates for prenatal care and birth weight but these are largely insignificant, making it difficult to assess the empirical relevance of selectivity. Baird et al. (2007) mostly analyse country-year level data, and do not analyse mechanisms. This paper controls more comprehensively for trended unobservables and shocks than related studies, also allowing their effects to vary by the season of birth of the child.

With a view to illuminating transmission, this paper explores cyclicality in maternal labor supply, social expenditure and health-seeking behaviour. No previous study investigates labor supply, although Dehejia and Lleras-Muney (2004) stress its potential importance in explaining cyclicality in infant mortality. No previous study directly investigates cyclicality in social spending, although Paxson and Schady (2005) underline its significance. A handful of studies analyse health seeking in the context of infant mortality, but they are unable to condition upon social spending; see Paxson and Schady (2005) and Miller and Urdinola (2007). This study exploits, for the first time, informative distinctions between public *v* private birthing facilities and between antenatal care sought and received.

The paper is organized as follows. Section 2 describes the data and Section 3 sets out the empirical strategy. Descriptive statistics are presented in Section 4. Results, extensions and robustness checks are presented in Section 5. Section 6 analyses mechanisms, and Section 7 concludes.

2. Background and data

The data on linked siblings are constructed from retrospective fertility histories provided in the National Family Health Survey of India (NFHS-2).[5] Ever-married women aged 15–49 in 1998–99 recorded the time and incidence of all births and any child deaths. Individual mortality data are thus available for cohorts of children (implicitly) followed over time from birth. Children in the sample are born in 1961–1999. These microdata are merged by state and year of birth with a state-level panel of data on income, rainfall, state social spending and other relevant macroeconomic indicators.[6]

To allow every child full exposure to infant mortality risk, children born in the twelve months before the survey date are dropped. I drop mothers that have ever had a multiple birth since death risk for multiple births is known to greatly exceed that for singletons and, as I am using sibling data, it is cleaner to compare singletons. An upper limit to the age of the mother at the time of the interview induces selectivity — as we go further back in time, the births captured in the sample are not only fewer but disproportionately of relatively young (possibly high risk) mothers.[7] Since most Indian mothers give birth

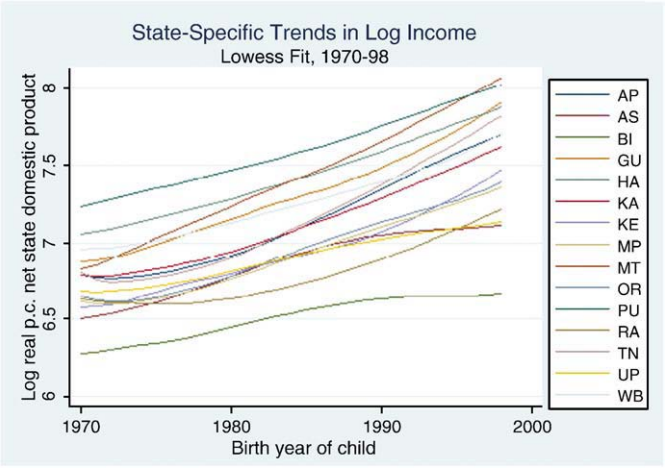

Fig. 2. State-specific trends in log state income.

relatively young, truncation in this sample of mothers who are relatively old at the time of birth is relatively unimportant, but is nevertheless accounted for by conditioning upon maternal age at birth.[8] Since the sample cell sizes by state and year for the 1960s are quite small, the sample is restricted to births occurring 1970 onwards. I also investigate truncating at 1980. Left truncation of the data also limits recall bias.[9] Another recall-related issue is rounding off of age. Since the data reveal age-heaping at six-month intervals, I define infant mortality as inclusive of the twelfth month. We may have a selectively healthy sample of mothers because mothers who do not survive to the date of interview are not in the sample. In this case, we may under-estimate the effect of income. Retrospective data demand that we account for inter-state migration. The survey records where the mother lives at the time of interview rather than at the time of each birth. Exploiting a question in the survey that asks the mother how long she has lived in her current place of residence (village, city or town), I limited the sample to births that occurred in her current location. Applying this strict criterion, I retain 86% of births. The reported results use the restricted sample, but since migration may be correlated with survival, I also confirm that failing to control for migration does not make a significant difference to the results. This is possibly because inter-state migration in India is very small (e.g. Topalova, 2005).

The sample analysed contains 117,088 rural children of 36,068 mothers (average of 3.3 per mother, including 1-child families) and 35,783 urban children of 13,414 mothers (average of 2.7 per mother) born during 1970–97 in one of the 15 major states.

3. Empirical strategy

This section describes how the unusually rich data structure of a micro-panel nested within a regional panel is exploited to avoid some of the specification problems in previous studies and achieve an estimate of the effect of income on mortality that might be deemed to be causal. Much of the literature has focused on identification problems relating to reverse causality, since the health of adults may be expected to influence their income through productivity (Adda et al., 2006; Halliday, 2006; Smith, 1999). Here, this problem is limited by looking at child rather than adult health and by analysing

[4] Baird et al. (2007) was written in parallel with the current study, an earlier version of which appeared as Bhalotra (2007a).

[5] For details on sampling strategy and context, see IIPS and ORC Macro (2000).

[6] These data were assembled by Ozler et al. (1996) and extended by Besley and Burgess (2002). For variable definitions and sources, see http://sticerd.lse.ac.uk/eopp/research/indian.asp. I use revised state health expenditure data and rainfall data from www.indiastat.com, both kindly collected by Juan Pedro Schmid.

[7] For example, a woman who gave birth at age 15 in 1965 and was interviewed in 1999 will be 49 at the date of interview, and her birth will be recorded. However, births to women any older than 15 years in 1965 will not be recorded since they will be older than 49 years in 1999.

[8] Maternal age at birth is potentially endogenous. In the mother fixed effects model we may assume it is not. I examined results that suppress it and found no significant change in the income effect.

[9] This refers to the possibility that mothers are more likely to forget the incidence or dates of events the further back in time they are. In fact, the DHS surveys have numerous checks built in to ensure the quality of birth history data (see ORC Macro, 2006, p.14).

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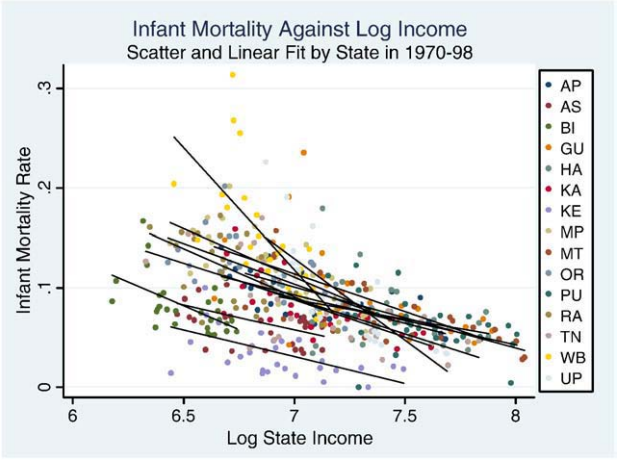

Fig. 3. Infant mortality against log income by state.

individual risk as a function of aggregate income (as in van der Berg et al., 2006).[10] However, there remain potential problems of selection bias and of omitted variables, and this section discusses how these are addressed.

The *basic equation*, which is then generalized, is

$$M_{imst} = \beta y_{st} + \alpha_m + \eta_t + \gamma_s t_{st} + Z_{imst}\rho + \varepsilon_{imst}. \quad (1)$$

M is a dummy that indicates whether index child i of mother m born in year t in state s died by the age of 12 months and y is the logarithm of per capita net domestic product in state s and year t deflated by the consumer price index for agricultural workers (henceforth *income*). β is the parameter of interest and it measures the change in infant mortality associated with a 100% change in income. α_m denotes mother fixed effects. Since, by construction (see Section 2), mothers do not migrate between states, the mother fixed effect incorporates a state fixed effect. Heterogeneity in death risk within mother is allowed for by including the child-specific Z_{imst}, specified as dummies for gender, birth-order, birth-month and age of mother at the birth of the index child. η_t are year (or cohort) dummies that control for aggregate shocks and $\gamma_s t_{st}$ capture omitted trends that vary by state.

Estimation uses the linear probability model since fixed effects probit estimates are inconsistent in short panels and the relevant panel in this case is the micro-panel, where T is the number of children per mother.[11] Standard errors are robust to arbitrary forms of heteroskedasticity and clustered at the state level to allow for correlation at a given time and across time within states (Bertrand et al., 2004). This also allows for correlation of the standard errors across siblings because, by construction, siblings are all in the same state.

Mother fixed effects are included to control for the possibility that the characteristics of women giving birth vary with the cycle (see Section 1).[12] They will capture, for example, differences across mothers in frailty, fertility, contraception preferences and awareness of health-related technology and services. To assess the empirical importance of controlling for such unobservables, I estimate a similar model that includes a rich set of observed mother characteristics. The Indian states have historical differences that are correlated with income and possibly also with health. The state fixed effects implicit in the mother fixed effects control for initial conditions and for persistent elements of history, climate, culture (status of women) and political institutions (public service delivery, corruption) that may simultaneously affect mortality and income. They also control for state-specific time-invariant sources of measurement error.

A problem that has marked historical and time-series analyses of the effect of income on health is the presence of trended unobservables, the effects of which will tend to load onto the income coefficient (Deaton and Paxson, 2004; Cutler et al., 2006). Regional panel data largely resolve this problem by allowing us to study the effects of deviations of income from trend. We start with a specification similar to that used in previous panel data studies (e.g. Ruhm, 2000; Dehejia and Lleras-Muney, 2004), which is to include year (cohort) dummies and state-specific linear trends. The year dummies control for aggregate time-variation associated with, for example, secular improvements in health technology, episodic shocks like famines, floods and epidemics and any economic or political regime changes. The state-specific trends allow for omitted trends that vary by state. A causal effect of income on mortality is identified in this specification on the assumption that the residual contains no state-time varying shocks that might drive a correlation between mortality and income. I therefore develop the specification in the following ways which, together, offer a substantial generalization upon previous studies.

The birth-month of the child is coded into quarter of birth dummies (q) which are interacted with year dummies to produce 112 quarter-year fixed effects ($\theta_q \eta_t$). This specification of aggregate shocks accounts for seasonal correlations of income and mortality. I allow for nonlinear evolution of health technology and other trended omitted variables at the state level by replacing linear with cubic state trends ($f_s(t_{st})$).[13] I control directly for the most likely confounding state-time varying shocks, namely, rainfall shocks and changes in public expenditure. Rainfall variation affects income through agricultural production and it affects health by altering the disease environment. Since about two-thirds of infant deaths in our sample occur in the first month of life (neonatal), I interact rainshocks with quarter of birth. Rainfall shocks (R_{st}) are measured as normalized deviations of state-level rainfall from its time-series mean (z-scores). I allow for the possibility that positive shocks have different effects from negative shocks by defining two terms, one equal to the z-score when the z-score is positive and zero otherwise, and another that is symmetrically defined.

In richer countries, fiscal policy tends to be counter-cyclical (Lane, 2003). However, in developing countries, it is not uncommon that state social expenditures are cut in response to large negative income shocks (Lustig, 1999). I therefore control for state expenditure on health, education (this may influence parental investments in child health) and other development (mostly on nutrition and other anti-poverty programs). Variations in state income now more closely reflect variation in household income. I then saturate the model with other state-time varying variables (X_{st}) that include the log and the change in the log of population, an indicator for urbanisation, income inequality and poverty. The augmented model can be written as

$$M_{imst} = \beta y_{st} + \alpha_m + \theta_q \eta_t + f_s(t_{st}) + \delta_q R_{st} + X_{st}\lambda + Z_{imst}\rho + u_{imst}. \quad (2)$$

[10] Their specification differs in other ways. In particular, they use time series rather than panel data on aggregate income so that their estimates of the effect of income are not clearly distinct from the effects of quality of public services or the relative price of different nutrients (see Banerjee et al., 2007).

[11] In fact, probit estimates of the income coefficient are insignificantly different.

[12] This problem of endogenous composition of inflow is analytically similar to that involved in modeling the effects of the business cycle on individual probabilities of exit from unemployment allowing for the possibility that the cycle also affects the composition of the inflow into unemployment (van den Berg and van der Klaauw, 2001).

[13] An advantage of a sub-national over a cross-country panel is that, given easier diffusion of technology within a nation, it is less likely that there are state-specific health technology shocks. Nevertheless, Figs. 1 and 2 suggest that it is restrictive to impose common trends across states.

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The notation is defined as in Eq. (1) or in the preceding text.[14] In this specification the identifying variation in income (y_{st}) is within state and nonlinear, being purged of any correlations with quarter-year dummies, cubic state trends, state and quarter specific rainfall variation, public expenditure, other state-level variables like inequality and state-specific trends in the micro-demographic variables (Z_{imst}), for example education of women and men and maternal age at birth. It is hard to think of an omitted variable and harder to think of one that would follow this nonlinear path.

The equation is estimated separately for rural and urban households. As the income effect is only significant in the rural sample, for this sample I further investigate heterogeneity in the income effect across and within households. As discussed in Section 1, the results of this exercise reinforce faith in the identification strategy.

We have exploited the microdata to many advantages. But the income data belong to a state-level panel, and this is thin and long ($N=15$, $T=28$). The length of the panel aids identification, making it more likely that there are independent macroeconomic fluctuations within states; indeed the standard deviation of within-state income variation in these data is almost identical to that of the between-state variation. However, when the panel is long, it becomes relevant to check that the mortality–income relationship identified in Eqs. (1) and (2) is not spuriously driven by stochastic trends. I therefore estimate a model with an equilibrium correction term that allows for the possibility that M and y are integrated of order one but may be cointegrated, and that models dynamics that, otherwise, will creep into the error term. The specification estimated is

$$\Delta M_{st} = \lambda y_{st} + \theta M_{st-1} + b(L)\Delta y_{st} + g(L)\Delta M_{st} + \alpha_s + \eta_t + \gamma_s t_{st} + \mu_{st} \quad (3)$$

where the notation is familiar, except that now the lag operator, L, is introduced and the state fixed effects, α_s are explicit. The demographic data (mortality, education, caste etc) at the individual level have been aggregated using sample weights to produce state-level proportions (M_{st}, Z_{st}). After experimenting with longer lags, I settled upon an ADL(3,3) model (translated to the ECM form shown) in which $b(L)=b_0+b_1L+b_2L^2$ and $g(L)=g_1L+g_2L^2$. The long run effect of income on mortality, $(b_0+b_1+b_2)/(1-g_1-g_2)$ can be directly read as the negative of the ratio of the levels coefficients, $-\lambda/\theta$, the standard error of which is computed using the delta method.[15]

Related studies in this domain, for example Ruhm (2000) and Dehejia and Lleras-Muney (2004) who use a long US panel, and Besley and Burgess (2002) and Burgess and Pande (2005), who use the same Indian panel, do not investigate non-stationarity. It is therefore useful to assess its relevance by comparing the ECM with standard panel data estimators. The cited studies use the within-groups estimator, which is what we get when Eq. (1) is aggregated up to the state level. This can be expressed as

$$M_{st} = \beta y_{st} + \alpha_s + \eta_t + \gamma_s t_{st} + \varepsilon_{st}. \quad (4)$$

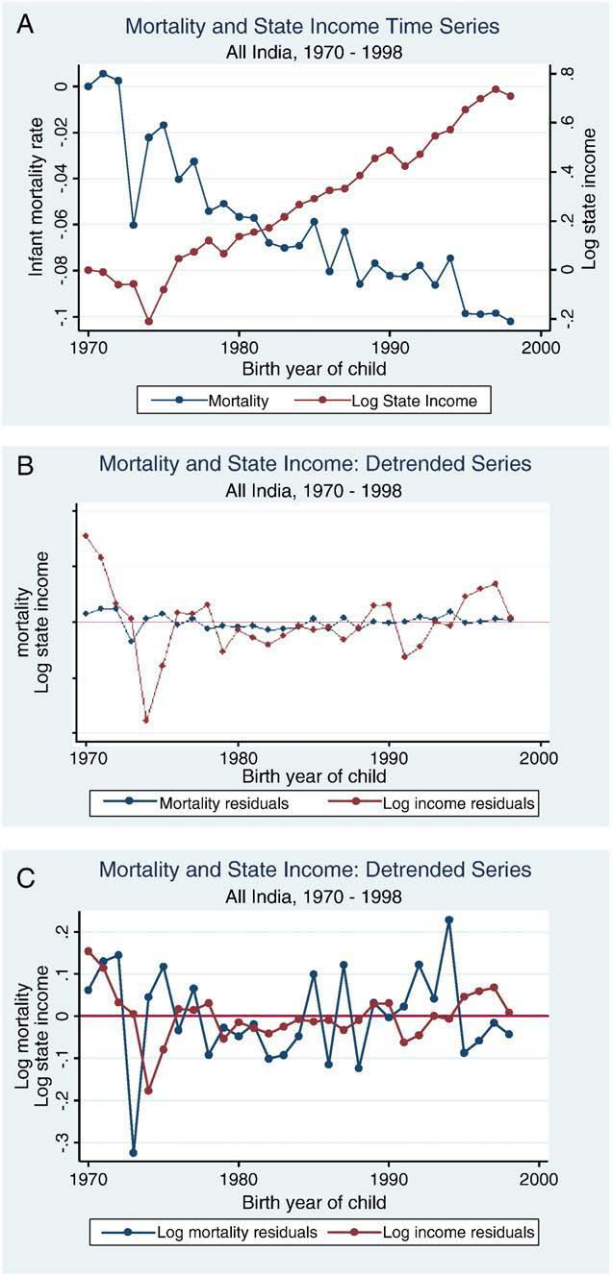

Fig. 4. A and B: infant mortality and log state income: population-weighted averages for all-India. A: raw, B: detrended. C: *Log* infant mortality and log state income: detrended series for all-India.

This is compared with the first-difference estimator which is a natural benchmark for the ECM model

$$\Delta M_{st} = \lambda \Delta y_{st} + \alpha_s + \eta_t + \mu_{st}. \quad (5)$$

Regressions of Eqs. (3)–(5) are weighted by the square root of the time-averaged state population.

The data and specifications employed for robustness checks, extensions and estimation of cyclicality in maternal labor supply and health-related behaviours are described in Sections 5.2 and 6, together with the findings.

4. Descriptive statistics

Fig. 1 shows state-specific trends in infant mortality rates derived from the microdata using sample weights. Averaging over 1970–1998,

[14] Here and in all equations that follow, the same Greek letters are used to denote parameters for convenience; they are of course not expected to be identical across equations.

[15] Consistent with Eqs. (1) and (2), state-specific trends are allowed to appear in the cointegrating vector. This allows that the level of mortality shows a trend decline (due to state-specific medical technical progress, say) and that the level of its path is determined by the log of state income. Control variables are initially included, like income, in levels and differences but dropped where insignificant since each regressor involves estimation of four additional parameters in this framework.

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the infant mortality rate in rural India was 9.4% and it decreased at 0.3%-points p.a. For urban India, the corresponding figures are 5.9% and 0.18%-points p.a. The median of the average yearly change in infant mortality at the state level was −0.33% in rural and −0.23% in urban areas. However, only in 52% (rural) and 51% (urban) state-year observations is the change negative.[16] There were vast differences in level and rates of change across the states. Although these differences have narrowed over time, Kerala achieved the fastest reduction even though its initial mortality was lowest, and its income growth was about average.

State trends in income are in Fig. 2. The average annual growth rate of state income over the period 1970–97 is 2.98% p.a., and most states exhibit acceleration after about the early-1980s, at which time an economic liberalization program was being phased in. State incomes are volatile. A regression of the log of state income on a linear trend in a pooled regression has an *R*-square of just 0.41, and the standard deviation of the residual is 0.30. This is typical of developing countries, the *R*-square for most industrialized countries being much higher (Pritchett, 2000). Recessions are frequent and quite severe. The annual change in log income is positive in only 65% of state-year cells. In these cases, the median size of the change is 6.2%, and the change at the 25th and 75th percentiles is 3.2% and 10.6% respectively. When negative, the median change is −4.4%, the 25th and 75th percentiles being −9.1% and −1.7% respectively.[17]

Figs. 3–5 explore the relationship between infant mortality and income. Fig. 3 plots a scatter and linear fit by state. The slope is negative in most cases. Aggregating these data to the state level with population weights, I find that, while the trends in the two variables are clearly inversely related (Fig. 4A), the relationship of the deviations from trend is weaker (Fig. 4B). Fig. 4C displays the detrended relationship with logged mortality. The proportional deviations are larger and the relationship with income stronger than in Fig. 4B; on which see Deaton (2006).[18] The dependent variable in the model I estimate is the individual risk of infant mortality, which aggregated to the state level produces the level (not log) of infant mortality. So I am less likely to find a significant relationship between income and mortality but, if I do, it is more likely to be causal. Although the analysis relies upon within-state variation, Fig. 5 exhibits the between-state relationship. The fitted line is negatively sloped but the data are quite dispersed around it.

In Section 1, we argued that women may consciously defer birth in recessions. This requires that they have some control over the timing of their births. Only 33% of women interviewed in rural India in 1992/3 reported current use of a modern contraceptive method, 63% reporting no method and the rest a traditional method. Amongst urban women, 44.6% used modern methods and 48.6% used no method. Average use in the period 1970–98 will have been lower.

5. Results

5.1. The baseline model

Table 1 displays results for the rural and urban samples. There is a significant negative effect of income shocks on mortality in rural households (which contain 73% of births). Once the data are detrended, income is insignificant in the urban sample. The urban

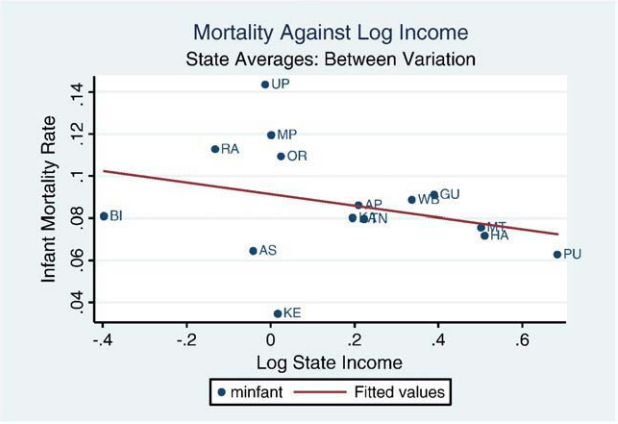

Fig. 5. Infant mortality against log income: the between-state variation.

coefficient is not small, but it is poorly determined.[19] The discussion here pertains to the rural sample and it focuses on the income effect. Full results are available on request.

5.1.1. Cross-sectional heterogeneity

The unconditional marginal effect of aggregate income on infant mortality risk is a significant −0.042 (column 1). The income coefficient identified on within-state variation is −0.084, twice as large (column 2). This implies that states that are relatively ineffective in translating income growth into lower mortality are not those with inherently high mortality risk, associated with the sorts of persistent historical or institutional factors that state fixed effects tend to capture. Introducing controls for observable mother-level traits lowers the income coefficient by one standard deviation to −0.075 (column 3). Every step up in the level of maternal education is associated with a gain in infant survival, and the effect doubles at the secondary and higher levels. Higher education amongst fathers is beneficial, but the marginal effect is smaller. Some previous studies have found that controlling for education diminishes any protective effect of income on health (e.g., Deaton and Paxson, 2001; Fuchs, 2004). The results in columns 1–3 show that there is an income effect conditional upon (parental) education. Note that although education does not vary within mother, it varies across cohorts and so exhibits state-time variation in line with the income variable.

Once we control for mother-level unobservables using mother fixed effects, the income coefficient doubles, to −0.15 (column 4). The percentage of the error variance contributed by mother-level heterogeneity is 39.[20] We are now comparing children of the same mother who, in their first year of life, were exposed to different economic conditions. This eliminates the potential concern that the income effect is simply a compositional effect. Indeed, the estimates are consistent with our hunch (see Section 1) that births in recessions are selectively low-risk, either because higher risk women are more likely to consciously defer fertility or because they are more likely to suffer miscarriage or stillbirth; this is confirmed in Section 5.2. Including child-specific characteristics brings the coefficient down to −0.069 (column 5). Birth-month dummies are jointly significant. Children of mothers under the age of 18 are significantly more likely to die. Like mother's education, maternal age at birth and birth order are trended because the data stack cohorts of children observed in age 0–1.

[16] The median change when positive (negative) is 2.2% (−2.6%) in rural areas and 2.6% and (−2.8%) in urban areas.

[17] State NDP (income) data may be subject to measurement error. In the econometric analysis, time-invariant measurement error specific to the state and arising, for example, on account of different accounting conventions, is captured by the state fixed effects.

[18] Deaton argues that if income growth has no influence on absolute changes then it follows that the relationship is really between income growth and the level of mortality and, in this case, it is hard to argue for a causal effect of income on mortality.

[19] The rural/urban difference is consistent with rural households having lower and more volatile incomes, and with their facing more limited access to markets and public services.

[20] These estimates do not use information from the 3.7% of children born in families that record only one child. The omitted children come from 12% of women who have only one child (rural sample). Using the specification that includes observable mother traits, I compared the income elasticity in the full sample with that in the sample of mothers with at least two children and they are the same.

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Table 1
The baseline model: the impact of state aggregate income shocks on infant mortality risk.

	Unconditional		Controlling for cross-sectional heterogeneity		
	(1)	(2)	(3)	(4)	(5)
	No controls	State dummies	Mother characteristics	Mother fixed effects	Child characteristics
Rural income	−0.042 [0.021]	−0.084* [0.011]	−0.075* [0.009]	−0.154* [0.014]	−0.069* [0.012]
Urban income	−0.045* [0.013]	−0.054* [0.010]	−0.046* [0.009]	−0.132* [0.018]	−0.062* [0.022]

	Controlling for aggregate shocks and state-specific trends				Controlling for state-specific shocks			Baseline
	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
	Time dummies	Time dum × birth quarter	Linear state trends	Cubic state trends	Rainfall shocks, + and −	Rainshocks × birth quarter	Population terms	Baseline model
Rural income	−0.055*	−0.056*	−0.035*	−0.034*	−0.027*	−0.027*	−0.027	−0.031*
	[0.020]	[0.020]	[0.011]	[0.012]	[0.012]	[0.012]	[0.012]	[0.013]
Urban income	−0.043	−0.043	−0.037	−0.031	−0.039	−0.039	−0.040	−0.041
	[0.031]	[0.031]	[0.038]	[0.047]	[0.047]	[0.047]	[0.047]	[0.036]

Notes: The dependent variable is an indicator for infant mortality and *income* is the log of real per capita net state domestic product. The number of children (mothers) is 117,088 (36,068) in the rural sample and 35,783 (13,414) in the urban sample. Once mother fixed effects are introduced the effective samples, which exclude 1-child families, are 112,760 (31,740) and 34,068 (11,699). Robust standard errors clustered at the state level are in parentheses. Elasticities are at the mean infant mortality rate, 0.09481 (rural) and 0.0599 (urban). Mother characteristics refer to her education, her partner's education, her ethnicity, religion and height. Child characteristics include gender, birth-order, birth-month and age of the mother at the birth of the child. Rainshocks are allowed to have different effects according to whether they are positive or negative. Population terms are the log level, its difference and the ratio of the rural to the urban population. Changes are cumulative in moving from col. 1 to 12. In col. 13, I revert to linear state trends, drop interactions of time dummies and rainshocks with quarter of birth, and drop population shocks and the inverse urbanisation rate.
* Significance at the 5% level.

5.1.2. Time-varying unobservables

Including year dummies drives the income effect down to −0.055 (column 6) although generalizing using interactions of year with quarter of birth does not alter the effect (column 7). Allowing for state-specific trends brings the coefficient down to −0.034 (columns 8–9). Most of this drop occurs with linear trends. These results confirm that failing to control for trended unobservables would result in over-estimation of the income effect, consistent with the mortality-reducing influence of trends in medical technical progress. The mother and year fixed effects and the state-specific trends are each jointly significant at the 1% level.

Controlling for rainfall reduces the income effect to −0.027 (column 10). A one standard deviation increase in rainfall decreases mortality risk by 0.004. As this is conditional upon income, it would seem to represent epidemiological effects, possibly conflated with effects on subsistence consumption that are not captured by state domestic product.[21] Since about two-thirds of infant deaths occur in the first month of life (neonatal), we may expect that the risk of death following a rain shock depends upon the birth-month of the child. However, conditional upon birth-month and the birth-quarter-year dummies, interacting quarter of birth with rainshocks makes no further difference to the income effect (column 11).

5.1.3. The specification of income-robustness checks

A possible concern is that, at a given level of aggregate income, an increase in fertility lowers *per capita* income and, given a positive association of fertility and mortality, this shows up as a negative impact of income on mortality. To control for state-specific fertility shocks, I included the logarithm of the state population and its first difference in the model. Both are insignificant, and the income elasticity is unchanged (column 12). I explored the alternatives of conditioning upon the state population under the age of 15 as this will proxy fertility better, and upon the number of births per mother recorded in the data, but in neither case was there a significant change in the income effect.[22] Adding the rural share of the state population similarly made no difference.

To allow big income shocks to have a different marginal effect from smaller ones, I included the square of log income in the model. This was insignificant, indicating that the semi-log form approximates the curvature in the mortality–income relationship sufficiently well. I also investigated an alternative generalization in which I included, together with log income, its first difference, allowing different coefficients according to whether this difference is positive or negative. These variables were insignificant, suggesting that mortality risk is sensitive to deviations of log income from trend but not to deviations in the growth rate of income.

5.1.4. Baseline model: size of effect

In column 13, I report a parsimonious form of the model obtained by dropping from column 12 terms that did not change the income effect (see notes to Table 1). The marginal effect of income in the baseline model is −0.031. Since the average infant mortality rate in the rural sample is 0.094, the elasticity at the mean is −0.33. Averaging over positive and negative shocks, annual income growth in the sample period was about 3% p.a., and our estimates suggest that this lowered infant mortality by 0.093 percentage points.

5.2. Robustness checks

This section discusses robustness of the results to controls for social expenditure, replacing current with future income, removing outliers, changing the window of time, and allowing for cointegration and dynamics.

5.2.1. State social expenditure

Column 2 of Table 2 introduces state-year varying variables that may be correlated with both mortality and income. These are the logarithms of real per capita state expenditure on each of health, education and other development. The income coefficient is robust to this; it rises from −0.031 to −0.035, by just less than a standard deviation. In auxiliary models, I regressed each of the state expenditures on state income. Within-group estimates of the elasticities are all significantly positive, 0.32 for each of health and education and 0.43 for other development spending. Thus social expenditure in India is pro-cyclical, declining in recessions. In principle, this may be a mechanism through which the identified effect of income on mortality operates. However, conditioning upon

[21] The finding of direct effects of rain on mortality conditional upon income invalidates the use of rain as an instrument for income in this setting.

[22] Another possibility is that infant mortality raises the share of the working age population and, in this way, raises productivity and income. Failing to control for this, we will be less likely to see a negative effect of income on mortality. Although this mechanism seems less important in the analysis of fluctuations than trends, I investigated it by including the share of the working age population in the model. The income coefficient was unaffected.

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Table 2
Robustness checks.

Panel A: Mother fixed effects estimates on the microdata

	(1) Baseline	(2) State expend.	(3) Drop Kerala	(4) 1980–
Income	−0.031*	−0.035*	−0.030*	−0.028
	[0.013]	[0.015]	[0.013]	[0.015]
Elasticity	−0.327	−0.369	−0.311	−0.318
N (children)	117,088	117,088	114,294	97,000
N (mothers)	36,068	36,068	34,837	34,366
Mean (dependent var)	0.0948	0.0948	0.0964	0.0879

Notes: The dependent variable is an indicator for infant mortality and this is the rural sample. The baseline is from col. 13, Table 1; see notes to Table 1. In column 2, the state variables included are logarithms of real per capita state expenditure on each of health, education and "other development".

Panel B: Alternative estimators on the state-level panel

	(1) Within groups	(2) First differences	(3) ECM
L.mortality			−1.371* [0.064]
income	−0.034* [0.015]		−0.091* [0.029]
D.income		−0.050 [0.025]	0.068* [0.031]
L. D.income			0.055* [0.023]
L2. D.income			0.053* [0.016]
L. D.mortality			0.185* [0.069]
L2. D.mortality			0.076 [0.045]
Long run income effect	−0.034* [0.015]	−0.050 [0.025]	−0.067 [0.021]

Notes: *Income* refers to the log of real per capita state domestic product and *mortality* to the state infant mortality rate. The dependent variable is *mortality* in column 1 and its first difference in columns 2 and 3. *L* is the lag operator, *D* is the first-difference operator. Every column includes year and state dummies and state-specific trends. In brackets are standard errors that are robust and clustered at the state level. These equations are estimated on the state-level panel ($N=15, T=28$). After taking lags and differences for the error correction model (ECM) in column 3, $T=25$. The sample in columns 1 and 2 is restricted to be the same as in column 3 and hence $NT=375$. In column 3, the long run effect is computed as the ratio of the coefficient on the level of current income to the coefficient on the lagged dependent variable. The differenced income terms are jointly significant at the 2% level and the differenced mortality terms are jointly significant at the 5% level. The table presents a parsimonious equation absent rainfall shocks and demographic indicators (elements of Z_{st}). These were initially included, like income, in levels and in current and lagged differences. The long run income effect was −0.073, which is also insignificantly different from the baseline estimate. If I restrict the ECM by dropping the second lags of the first-differenced terms, the long run income effect (on the same sample) is −0.044.

social expenditure does not diminish the effect of aggregate income shocks on mortality (see Anand and Ravallion, 1993).[23] It would therefore seem that the relevant mechanisms pertain to liquidity constraints at the individual level. This is supported by the direction of heterogeneity in the income effect but it does not show up, as we may expect, in asymmetry of the income effect (see below).

5.2.2. Placebo check

Infant mortality risk should not be affected by future income. I estimated the baseline specification using income in period (t + 1) in place of contemporary income. The coefficient on lead income is 0.025 and its *p*-value is 0.15.

5.2.3. Outliers and retrospective data

The data show, across India, a dip in mortality in 1973 (Fig. 4A), and Kerala has unusually low mortality through the period (Fig. 1). The year and state dummies capture these effects to the extent that they are additive. I now allow for slope differences by excluding Kerala and 1973 from the estimation sample. Rather than simply drop the year 1973, I drop all years in the 1970s so as to check, at the same time, for any selectivity associated with the retrospective nature of the mortality data (Section 2). The income effect is insignificantly different in every case (Table 2: panel A).[24]

5.2.4. Stochastic time trends — refer to Table 2: panel B

The ECM estimates show evidence of persistence and dynamics and the long run effect of income is −0.067, which is larger than but not significantly different from the baseline estimate (column 3). The within-group estimate is, as expected, almost identical to the estimate obtained on microdata. The first-differenced model produces a bigger coefficient, of −0.05, which is insignificantly different from the baseline or WG estimate. Overall, there is no evidence here that the estimated effect of income on mortality is spurious.

5.3. Extensions

This section discusses cyclicality in the composition of births by mother's SES, heterogeneity and asymmetry in the effect of income on mortality, and its timing.

5.3.1. Composition of births by mother's SES

We have seen that controlling for mother-level unobservables increases the size of the income effect on mortality (Table 1). This suggests that high-risk mothers disproportionately avert birth or suffer fetal death in recessions. Here I investigate this using *observable* indicators of risk. With individual data aggregated to the state level with sample weights, I estimated the following model

$$C_{st} = \beta y_{st} + \alpha_s + \alpha_t + \mu_{st} + \delta_q R_{st} + \varepsilon_{st} \quad (6)$$

where *C* is the percentage of births in a state-year to mothers with a given characteristic (e.g., no education), *y* is log state income and the other terms denote state and year dummies, state-specific trends and rainshocks. Explicit results are in an earlier version of this paper (Bhalotra, 2007a); here I summarise the findings, and put significant income coefficients (β) in parentheses. In rural areas, births to uneducated women (0.048), women with uneducated husbands (0.048) and women of scheduled tribes (0.042; ST are a disadvantaged ethnic group) are significantly under-represented in a recession. A similar result obtained by Dehejia and Lleras-Muney (2004), who show that the proportion of US births contributed by black women is smaller in recessions. On the other hand, using Peruvian data, Paxson and Schady (2005) find that higher risk women are *less* likely to avert birth in recessions.

5.3.2. Heterogeneity in the income effect

The data are split by level of mother's and father's education [none *v* some] and by maternal age at birth [9–18 *v* 19–42] as the baseline estimates show that these variables have large effects on mortality risk. In the rural sample, 74% mothers and 40% fathers have no education, and 60.5% of mothers had their first birth by the age of 18. Recall that the average income effect in the rural sample is −0.031 (Table 1). Analysis of sub-samples (see Table 3) shows that the income effect is significantly larger for children with uneducated mothers (or fathers) and mothers who initiated fertility before the age of 18 [−0.04 *v* insignificantly different from zero]. Comparing brothers and sisters, I find a much bigger impact of income shocks on girls (−0.06); indeed, boys are fully protected. This is consistent with a previous literature showing that investments in girls are more sensitive to

[23] Social expenditure has small if any effects on mortality. The contemporaneous effects are all insignificant although, in Bhalotra (2007c), I show that health expenditure exhibits a protective effect with a lag.

[24] I have confirmed that the income effect is not significantly different if, instead, I start the sample in 1974 or 1975 rather than in 1980.

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Table 3
Heterogeneity in the effect of income shocks on mortality.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Sector		Mother's education		Father's education		Ma age at first birth		Child gender	
	Rural	Urban	None	Some	None	Some	9–18	19–42	Girls	Boys
Income	−0.031*	−0.041	−0.042*	0.007	−0.044*	−0.019	−0.039*	−0.018	−0.057*	−0.004
	[0.013]	[0.036]	[0.013]	[0.035]	[0.017]	[0.017]	[0.018]	[0.022]	[0.023]	[0.022]
Elasticity	−0.327	−0.684	−0.403	0.102	−0.411	−0.219	−0.378	−0.220	−0.595	−0.043
Mean dep var	0.095	0.060	0.104	0.069	0.107	0.087	0.103	0.082	0.096	0.094
N (children)	117,088	35,783	86,305	30,783	46,990	70,098	70,888	46,200	61,002	56,086
N (mothers)	36,068	13,414	24,378	11,690	13,205	22,863	20,461	15,607	30,567	28,213
% of group	76.6	23.4	73.7	26.3	40.1	59.9	60.5	39.5	52.1	47.9

Notes: The dependent variable is an indicator for infant mortality. The baseline specification is that in column 13, Table 1. Except in column 2, the sample is restricted to *rural* households. The sub-groups % are of the child sample, so for mother characteristics they are implicitly weighted by the number of children that the group contributes to the sample. Of children whose fathers have no education, 93% of mothers have no education. In the sample whose mothers have no education, 51% of fathers have no education.

income in difficult times (e.g. Behrman and Deolalikar, 1989; Rose, 1999; Bhalotra, 2007b). The results in this section make it unlikely that any omitted variables drive our main findings since they would have to interact with characteristics (like being a girl or having an uneducated mother) in just the same way as income does.

5.3.3. Asymmetry in the income effect

It is conceivable that the effects of recessions are different from the effects of booms under survival constraints. I investigated this asymmetry by splitting the (log) income variable into one variable that records income when it is positive and is set to zero otherwise, and a second that similarly switches on when income is negative. Their coefficients are almost identical. I also checked asymmetry using the first-differenced model estimated on the state panel, as in Table 2: panel B. I defined separate terms for positive and negative income differences. Again, the coefficients on these terms were insignificantly different.

5.3.4. The timing of the income effect

The results so far report the effect of annual changes in state income in the year of birth, in line with previous studies in this literature. I now exploit information on the birth-month of the child to investigate the stage of life — fetal, neonatal or postneonatal — at which exposure to adverse conditions is most likely to result in infant death (death in age 0–12 months). The fetal period is the 9 months *in utero*, the neonatal period is the first month of life and the postneonatal period is months 2–12 of life. This is crude because the income data are annual, birth-month may be measured with error, and if income operates on mortality with a lag (see the ECM above) then these distinctions are blurred. With these caveats in mind, I create a child-specific average of state income for each exposure period, where the weights are the fraction of the period spent in each of two fiscal years.[25] Results are in Table 4. Including each income term separately, I find that income in the fetal (−0.027) and neonatal periods (−0.036) has a significant negative effect on mortality in infancy, but income in the postneonatal period is insignificantly small (−0.015); a related result is reported in Chay and Greenstone (2003). It looks like the effects of income on mortality may work more through maternal health (nutrition, antenatal care) and delivery practices than through post-natal investments in the nutrition and healthcare of the child; this is investigated further in the following section.[26]

6. Mechanisms

This section considers mechanisms by which the identified negative effect of income fluctuations on mortality may operate. We have seen that recessions are associated with a decline in public (state) expenditure. Here we note that they are also associated with a decline in private (household) expenditure [and wages]. We find that they stimulate an increase in maternal labor supply and, related, a decline in the demand for health inputs.

6.1. Health-seeking behaviours

Interventions relevant to infant mortality in poor countries involve improvements in antenatal care, skilled attendance at delivery, immunization, and treatment of diarrhea, malaria and respiratory infections (Black et al., 2003; Jones et al., 2003). This section analyses cyclical variation in the use of these inputs. Information on health seeking is available in the second round of the NFHS for children born in the three years preceding the date of the survey. To gain more time-variation in these data, I pool them with similar data from the 1992/93 round which recorded this information for children born in the four years before the survey. The resulting data have range 1988–1998, in which years 1994 and 1995 are empty. Children in the sample are aged 0–47 months at the time of the interview. These microdata are merged by state and year of birth of the child with the state-level panel that includes income. Information on infectious disease refers to the two weeks before the survey date. To investigate this, I merge the micro- with the macro-data by state and year of interview (rather than year of birth); year of interview is one of 1992, 1993, 1998, and1999.

Definitions of the dependent variables analysed are in notes to Table 5. For immunization, which occurs through infancy, I use income in the year of birth, and for place of delivery (birth) and antenatal care,

[25] The fiscal year in India runs from 1 April to 31 March. For children born in January–April, income in the fetal period is simply lagged income. For children born in May it is 8/9(lagged income) + 1/9(current income) and so on, so that for children born in December it is 1/9(lagged income) + 8/9(current income). Income in the post-neonatal period is similarly adjusted. For children born in January, it is 2/11(lagged income) + 9/11(current income) and so on. Since the neonatal period is a single month, income in the neonatal period is lagged income for children born in January–April and current income for children born in May–December.

[26] With the data available here and in other similar studies, it is difficult to draw unambiguous conclusions; ideally we would have month of birth and monthly income data and no measurement error in either. As a check, I swapped dependent variables, estimating the effects of unadjusted income on neonatal and under-5 mortality. The neonatal income elasticity is an insignificant −0.10. The marginal effect of income on under −5 is larger than on infant mortality (−0.04 *v* −0.03), although the elasticity at the mean is the same (−0.33). Dehejia and Lleras-Muney (2004) separate infant mortality into neonatal and post-neonatal and find larger effects of current income shocks in the post-neonatal period; but estimates for post-neonatal mortality condition upon neonatal survival and this selectivity may bias the results. They use the first lag of the shock, which is approximately like the shock in the fetal period, to instrument the current value of the shock. This strategy is not valid if fetal resources have a direct effect on infant survival.

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I use income in the year before birth. For immunization, the sample is restricted to children who are at least a year old at the time of survey to allow for the natural course of vaccinations spread through the first year of life. For treatment of diarrhea and respiratory infection, I report results for a sample restricted to children who the mother reports contracted the infection, as well as unconditional estimates (see Dow, 1996). As before, I use the linear probability estimator but I cannot use mother fixed effects because only 19.5% of births are from mothers who have at least two children. The sample of siblings is not only small but also endogenously selected, including only women with very short birth intervals (i.e. two births in a 3 or 4 year span). Standard errors are robust and clustered at the state-year level; they are smaller if clustered at the mother level (Table 3, Bhalotra, 2007a). Estimates are conditional upon gender, whether first-born, current age of the mother, level of maternal and paternal education, caste and religion and, in a variant, a proxy for the supply of public services. As the equations include state dummies, state-specific trends and time dummies (which encompass a survey-year dummy), we are identifying the effects of deviations in state income from trend.

In parallel with the analysis of mortality, the analysis was conducted separately for rural and urban households, with sample sizes of 50,195 and 15,819 children respectively. Urban results are in the Online appendix[27], and rural results are in Table 5. They are consistent with our findings for infant mortality. In downturns, rural Indian mothers are significantly less likely to deliver outside the home, seek antenatal care and get children immunized and treated for illnesses. It is striking that an increase in income increases the probability of delivery in a *private* facility by exactly as much as it decreases the probability of delivery at *home* (0.11), while there is an insignificantly small effect on delivery in a *government* facility (0.02). This is suggestive of constraints on the affordability of private care. We also find that income shocks have a larger impact on antenatal visits *sought* than on whether a visit from a health worker is *received* (coefficients of 1.98 *v* 0.05). These results indicate demand effects. To investigate directly the role of cyclicality in the supply of public services, I included state health expenditure as a proxy for this (panel B, Table 5).[28] It has a significant positive effect on some indicators of health seeking. However, the effect of income on health seeking is undiminished.

It seems therefore that recessions lower family resources available for child health. These will tend to include private expenditure and maternal time. Absent individual income or consumption data, we used state-level consumption, measured to include expenditure on nutrition and health inputs (Ozler et al., 1996; Besley and Burgess, 2002). The income elasticity of rural consumption is estimated to be a significant 0.13; in contrast, urban household consumption is invariant to aggregate income changes. This is consistent with previous evidence that rural households are not fully insured (Townsend, 1995). Cyclicality in maternal time is investigated next.

6.2. Parent's labor supply

We are primarily interested in the labor supply of mothers because their health and health-seeking behaviour matters for child survival. However, to confirm this presumption and also as a robustness check, parallel results for father's labor supply are presented. Information on work participation is available at the time of interview so, using two rounds of the NFHS as in Section 6.1, we have information for 1992, 1993, 1998 and 1999. All women in the sample are mothers, aged 13–49. Fathers are identified as partners and 98% are in the same age bracket. The individual data are merged with the state-level panel by year of interview. Regressors include a quadratic in current age, indicators for level of education of the woman (man) and their partner, caste, religion, state dummies, time dummies and state-specific trends. The trends are adjusted to reflect the jump between 1993 and 1998. It is unlikely that changes in the workforce participation of rural women will have any quantitatively important feedback effect on changes in income. The work variables are defined in notes to Table 6. Estimates are of the linear probability model, with standard errors clustered at the state-year level.

Table 4
The timing of income shocks on infant mortality income in the fetal, neonatal and postneonatal periods.

	(1)	(2)	(3)	(4)
Exposure period	Infant	Fetal	Neonatal	Postneonatal
Income	−0.018 [0.017]	−0.027 [0.014]	−0.036* [0.012]	−0.015 [0.017]

Note: The equation specification is as in col. 13, Table 1, for the rural sample, with 117,088 children of 36,068 mothers and the estimation uses mother fixed effects. The dependent variable is an indicator for infant mortality. These estimates use information on the child's birth-month to construct average state income at different times of life; see the text. So income now varies not only across 28 years and 15 states but, further, across 12 birth-months. In col.1, it is income in infancy, age 0–12 months inclusive. In col. 2, it is income in the 9 months *in utero*. In col. 3, it is income in the neonatal period, 0–1 month, and in col. 4 it is income in the postneonatal period, 2–12 months.

6.2.1. Mother's labour supply

See Table 6 for rural results. Urban results are in the Online Appendix. About a third of rural women work, amongst whom two-thirds work in agriculture. Less than a sixth of urban women work, amongst whom less than a fifth are in agriculture. Even with the fairly limited variation available, we see significant effects of aggregate income shocks on work participation. Rural Indian mothers increase their labor force participation in recessions. A 5% increase in state income is associated with a 4.7% decrease in work participation. The elasticity is similar for urban women, but poorly determined. So the labor supply curve of Indian women is forward falling (negative at low wages).[29] The likely reason is that women are secondary workers who increase work participation in response to an adverse shock to the main earner's income; also see Bhalotra and Umana-Aponte (2009).[30] In other words, the added worker effect dominates the discouraged worker effect for women within couples. This temporal effect is consistent with cross-sectional evidence on women's labor supply in India, which is U-shaped, with participation rates being highest amongst the very poor and the highly educated, and quite low in-between (Das and Desai, 2003; also see Goldin, 1995; Mammen and Paxson, 2000).

Disaggregation of work as agricultural/not and unpaid/paid reveals that the overall result is driven by informal work. Paid and non-agricultural work in rural areas behave conventionally, decreasing in downturns, but the opposing effects of unpaid and agricultural work dominate. These results make sense — there are fewer jobs on offer in downturns, so we would be unlikely to see formal sector employment rise, but the informal sector can expand to accommodate subsistence work. Since unpaid and agricultural work are more often performed by less educated/poorer women, the results also tie in with the interpretation of women's work being used as an insurance mechanism by poor households. Some previous studies adduce related evidence (Kochar, 1995; Frankenberg et al., 2003; Halliday, 2006). In their seminal analysis of cyclicality in mortality, Dehejia and Lleras-Muney (2004) argue, on the grounds of conventional wisdom, that the opportunity cost of mother's time is higher in upturns, but they do not investigate this. I find no evidence to support the conventional wisdom, not even for urban women in India.

[27] See http://www.efm.bris.ac.uk/ecsrb/bhalotra.htm.

[28] Conditional upon state income and trends, this variable will reflect the effect of changes in state expenditure *share*. It is potentially endogenous as governments may raise health expenditure in response to a health shock. Although we have controlled for non-linear trends and rainshocks, there may, for example, be an epidemic that is detached from rain variation. However, in this section, we are primarily seeking to establish correlations that tie in with the main results.

[29] For discussion of the shape of the labor supply curve under subsistence constraints, see Barzel and McDonald (1973) and Bhalotra (2007b).

[30] Although the data do not contain individual wages or earnings, there are state-level time series data on the average daily rural agricultural wage. This declines in downturns; the income elasticity is 0.38.

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Table 5
The impact of income shocks on health-seeking behaviour.

	Place of delivery					Antenatal care	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Home	Government facil.	Private facility	Complete	First trimester	No. visits sought	1(visit received)
A. baseline							
Lagged income	−0.112* [0.032]	0.019 [0.029]	0.107* [0.030]	0.019 [0.040]	0.089* [0.042]	1.975* [0.298]	0.046 [0.063]
B. Add state health expenditure							
Lagged income	−0.111** [0.037]	−0.008 [0.035]	0.124** [0.033]	0.022 [0.051]	0.094 [0.048]	1.643** [0.347]	0.134* [0.067]
State health exp	−0.036* [0.017]	0.046* [0.021]	−0.005 [0.022]	0.016 [0.038]	0.053* [0.025]	0.042 [0.259]	−0.014 [0.048]
Mean dep var	0.805	0.108	0.083	0.278	0.222	1.87	0.224
Observations	49,515	49,515	49,515	49,060	49,592	43,451	49,357

	Child immunizations		Treatment: conditional		Treatment: unconditional	
	(8)	(9)	(10)	(11)	(12)	(13)
	Number	Full	Diarrhea	Respiratory	Diarrhea	Respiratory
A. baseline						
Income	−0.129 [0.299]	0.163* [0.050]	0.679* [0.172]	−0.061 [0.087]	0.160* [0.035]	0.110 [0.099]
B. Add state health expenditure						
Income	−0.097 [0.402]	0.198* [0.082]	1.026** [0.213]	−0.013 [0.146]	0.267** [0.065]	−0.006 [0.108]
State health exp	0.449 [0.307]	0.057 [0.046]	0.192* [0.095]	−0.085 [0.080]	0.161** [0.036]	−0.054 [0.066]
Mean dep var	3.67 [max 8]	0.223	0.588	0.533	0.082	0.174
Observations	32,873	35,028	5593	13,060	40,092	40,092

Notes: The dependent variables are binary except in cols. 6 and 8 where they are counts. Columns 1–7 use the sample of rural children born in 1988–1998 aged 0–4 years, cols. 8 and 9 restrict this sample to children aged at least 1. Treatment in cols. 10–11 is conditional on contracting the infections and in cols. 12–13 it is unconditional. Complete antenatal care in India is defined as at least 3 antenatal care visits, at least one tetanus shot and a 3-month course of iron folic tablets. In first trimester is unity if the woman made an antenatal visit in the first 3 months of pregnancy (as advised) and zero otherwise. Number of visits sought is the number of antenatal visits that the woman went in for. Visits received is 1 if the woman has ever received a home visit from a health worker. "Full" immunization refers to 1 BCG, 3 DPT, 3 Polio and 1 measles shot, expected to be had by age 1. Treatment may be at a public or private facility, we only measure whether or not treatment was sought.
*,** Significance at the 5% level.

6.2.2. Father's labor supply

In rural and urban areas alike, about 97% of fathers work, so there is only small room for manoeuvre. The average income elasticity is positive but insignificant. Men have a larger positive elasticity in non-agricultural work than women, and a smaller negative elasticity in agricultural work. Men are also almost four times as likely as women to work in non-agriculture. So distress work stimulated by recessions is primarily located amongst rural agricultural women, consistent with adverse effects of recessions on rural infant mortality if women are the primary caregivers.

Overall, the results in this section contribute to the evidence that, faced with limited consumption smoothing opportunities, poor families attempt to achieve income smoothing, but not without costs. The literature has highlighted costs in terms of sub-optimal production and profits (see Morduch, 1995) rather in terms of health, although see Artadi (2005), who focuses on seasonal variation; also see Attanasio et al. (2005) and Kochar (1995).[31]

6.3. Maternal labor supply, infant mortality and health inputs

If recessions (booms) are associated with increased (lower) maternal labor force participation and if this means less (more) time dedicated to maintaining child health then this may be a mechanism by which income shocks impact infant mortality. To complete the "chain", I investigated the effects of being a working mother on a range of health outcomes. With the limited cross-sectional data available for this analysis, the estimates cannot be regarded, *a priori*, as causal. In particular, the innate health of the child may influence the mother's decision to work. In practice, if "feedback" of this sort or omitted variables were driving the results presented here, they would have to exhibit the pattern of differences across rural/urban, agri/nonagri and mother/father that I find. For infant mortality, I select children born exactly 12 months before the date of interview and for health inputs, I use the most recent birth of each

Table 6
The impact of income shocks on parent's work participation.

	(1)	(2)	(3)	(4)	(5)
	Any work	Agriculture	Non-agri	Unpaid	Paid
A. Mothers					
Income	−0.273*	−0.380*	0.105	−0.397*	0.207*
	[0.090]	[0.086]	[0.055]	[0.077]	[0.079]
Mean (dependent variable)	0.291	0.196	0.095	0.114	0.173
Observations	40,092	40,030	40,030	40,032	40,032
B. Fathers					
Income	0.015	−0.266*	0.275*		
	[0.028]	[0.094]	[0.095]		
Mean (dependent variable)	0.972	0.506	0.471		
Observations	39,454	39,263	39,263		

Notes: The dependent variables are all defined against "no work". These are results for rural households, for urban households, see the Online appendix. Estimates are for women aged 13–49 and their partners for whom labor force participation is recorded at the time of interview. Women are interviewed in 1992, 1993, 1998 and 1999. The dependent variables are binary, these are LPM estimates. A question on type of employment is used to construct the paid/unpaid classification and a question on occupation is used to construct the non-agriculture/agriculture classification. Participation in paid and unpaid work adds up approximately to total work (in column 1), as do participation in nonagri/agri work. This adding up is not exact because the unclassified people in the employment and occupation questions are not identical and because the employment question has a third response, which is self-employment (less than 5% of women). Unpaid and agricultural work are likely to be mostly subsistence work. Income is log real p.c. state net domestic product. The models include state dummies, time dummies and state-specific trends, a quadratic in current age and indicators for the woman's level of education, the level of education of her partner, her caste and her religion.

[31] Women's labor supply as an insurance mechanism is discussed in Attanasio et al. (2005). Using data from rural India, Kochar (1995) demonstrates that households with more able-bodied people and especially men achieve more insurance.

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mother amongst births in the last 3 (if NFHS2) or 4 (if NFHS1) years. Results are in the Online appendix; here I present a summary of the (conditional) correlations. Rural infant mortality risk is higher by 0.05 (50%) if the mother works in agriculture. There are no adverse effects associated with mothers working in non-agriculture or with fathers working in either sector, and there are no adverse effects in urban areas. Mothers' participation in rural agricultural activity also has consistently adverse effects on each of the ten indicators of health seeking studied in Table 5. The chances of delivering the child outside the home and of seeking antenatal care in the first trimester decrease by 0.05 (25%) and 0.03 (13.5%) respectively, and the probability that the child is fully immunized decreases by 0.03 (13.5%). Children of mothers in agricultural work are more likely to contract both diarrhea and respiratory disease, and less likely to be treated. It is striking that the agricultural activity of fathers has almost no adverse effects on survival or health. Indeed, father's employment, especially in non-agriculture has beneficial effects. This no doubt reflects an income effect where employment, conditional upon education increases resources available to purchase inputs into child health.

7. Conclusions

Shocks to state-level income in India cause substantial variation in infant mortality in rural Indian households, even after adjusting for possible selection associated with heterogeneity in fertility timing or in survival until birth. We may expect income effects to be larger in poorer countries, but the direction of cyclicality in mortality is unclear *a priori* because of the uncertain sign and size of relative price effects. This paper identifies counter-cyclicality. A recession involving a one standard deviation change in log income (0.39) is estimated to raise mortality risk by 1.2%-points, other things equal. Taking the UN estimate of live births in India in 1990 of 26.3 million, this implies an additional 0.32 million infant deaths. These are large effects and the effects on lifetime health will tend to be even greater since, where children survive income shocks in childhood, early exposure to poor living conditions has lasting adverse effects on their health (e.g. Almond, 2006; Banerjee et al., 2007; van der Berg et al., 2006).

Analysis of cyclical variation in birth composition suggests that Indian mothers experience fetal loss or time fertility in a manner that lowers average death risk (and enables greater labor supply). However, they appear to be constrained in the extent to which they can time their labor supply to maximize infant survival chances. Although there is a large literature on consumption smoothing in poor countries there is limited evidence on maternal labor supply as a smoothing mechanism. This paper has brought together two empirical features of poor countries that distinguish them from richer countries: their astounding scale of childhood mortality and their greater income volatility. It demonstrates that, even if income fluctuations are temporary, they cause irreversible damage.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.jdeveco.2009.03.006.

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