Does Demographic Transition Influence Development Outcomes?

An age-cohort analysis of fertility decline and infant mortality in India

SUMIT MAZUMDAR
Future Health Systems RPC – India
Institute of Health Management Research
Kolkata, India
sumitmazumdar@gmail.com

M. HAFIZUR REHMAN
Johns Hopkins University
Baltimore, MD
hrehman@jhsph.edu

Abstract: While progressive stages of demographic transition often correlate well with broader development processes, how the constituent demographic processes influence specific development outcomes is yet to be clear. With contemporary demographic transition in India as case-study, this paper examines relative effect of fertility decline on infant mortality. Using state-level age cohorts in a pseudo-panel approach, we test for the hypothesis that fertility decline significantly influences infant survival prospects. Unconditional demand models allow corrections for potential problems of measurement error and endogeneity and incorporate temporal changes in the gradient of influence. Extending the comparative statics of the analytical model, we delineate possible reductions in infant mortality under alternative scenarios of fertility decline. As we find, recent fertility decline in Indian states have significantly, and positively influenced infant survival, with a largely constant effect over the last 15 years. Hence, intensification of fertility reduction efforts can be expected to stimulate early-childhood survival prospects.

Keywords: Cohort analysis, pseudo-panel data; fertility decline; infant mortality; India

I. INTRODUCTION

The forces of demographic transition have historically characterized evolution of all human populations across the globe, its varying pace and nature being the structural ingredients of national demographic characteristics. The progressive stages of demographic transition often correlates well with broader development processes; populations in advanced stages of demographic transition generally enjoy better quality of life. But to what extent demographic transition or its constituent demographic processes influences development outcomes? For example, how far can we expect steady fertility decline to influence health status among the newborn and children?

With contemporary demographic transition in India as a case-study, this paper considers this broader issue with a specific focus: the relative effect of fertility decline on infant mortality. Reducing infant mortality rates constitute one of the prime development targets under the Millennium Development Goals (UN XXXX), and widely considered as an important summary indicator of human development. Using state-level age cohorts in a pseudo-panel approach, we test for the hypothesis that fertility decline significantly influences infant survival prospects in India using unconditional demand models for child health. Corrections are allowed for potential

problems of measurement error and endogeneity in the econometric specifications, and we examine whether the degree of such influence changes over the last decade and half. As an extension of the comparative statics of the analytical model, an attempt is made to delineate the possible reductions in infant mortality under alternative scenarios of fertility decline across the states. As the paper finds, recent fertility decline in Indian states have significantly, and positively influenced infant survival, with the effect being largely constant over the last 15 years.

Demographic transition refers to the process of population growth conditioned by the vital events of birth and death. In the earliest stage, that of pre-industrial (or 'Malthusian') societies, both births and deaths are high and population growth is slow. With the advent of medical technologies (vaccines and antibiotics for e.g.), life expectancy increases steadily, depressing the death rates in the next stage of the transition. However, with little spread and popularity of birth-control methods, fertility remains at higher levels causing a so-called stage of 'population explosion'. In the final stage of the transition, combined effect of socioeconomic development and improved medical technologies causes both fertility and mortality rates to fall steadily and ultimately settle to a constant, low-level. Population growth decelerates and an ageing society predominates (for further discussion on demographic transition, please refer Debraj Roy (XXXX) and Dudley Seers (XXXX)). While almost all the developed nations are well-past the era of population explosion, much of the developing world including India are yet to stabilize population growth and reduce fertility to the 'replacement-level' , where a couple is 'replaced' by their children (i.e. each couple having approximately two children). Apart from this broad association between economic development and demographic transition, countries with lower levels of fertility and mortality are also the ones with better indicators of socioeconomic and human development, as compared to their counterparts (mostly in South Asia and Sub-Saharan Africa) in the early, or middle-stages of demographic transition having higher levels of fertility, lower life expectancy and higher incidence of childhood mortality.

Population dynamics governing the nature and pattern of demographic transition are, in turn, primarily conditioned by intertemporal changes in fertility and mortality. For populations like that of India, with a younger age-structure (in terms of lower median age of the population), fertility transition is the major contributor to the process of demographic change (see footnote). Declining fertility directly depresses birth rates and slows population growth, assuming approximately constant average longevity of life. Recent demographic history of such populations clearly indicates the pattern; a relatively sharper trajectory of birth rates along with a flatter time-trend of death rates (Figure required). Hence, considering fertility decline to be the driving force of demographic transition, and examining its relative influence on development outcomes is in order.

Fertility & Infant mortality in India during the 1990s (add here)

II. Fertility decline and Infant Mortality: The Theoretical Linkages

It is widely acknowledged that fertility behavior and child survival prospects are interlinked. However, the causal mechanisms between the two remain a debated issue. The theory of demographic transition suggests that mortality decline, involving higher survival possibilities in the early childhood, usually precedes, and in fact be a precondition for fertility reduction (Simmons et. al. 1982). This makes fertility a function of mortality (Preston 1978).

That high fertility is a behavioral response to high mortality is supported through the 'child replacement hypothesis' which states that parents try to replace their children who die (footnote on demand for children; children viewed as 'normal' goods in the utility framework) and the 'child survival hypothesis' stating that parents aim to produce enough children so that the desired number of children reaches adulthood (Mari Bhat XXXX, others). Hence, it is argued, that fertility levels would not drop until parents are convinced that prevalent mortality conditions have sufficiently improved. However, mortality can sometimes be a response to high fertility instead of being a stimulus to it (Scrimshaw 1978). A rich demographic literature suggests a strong, significant relationship between mortality and birth spacing (review in Panis & Lillard 1995), where children of shorter birth-intervals and/or of higher parity are found to face increased mortality risks. Several hypothesis and rationale have been forwarded to justify the observed correlation and causality: these include mother's depletion due to rapid, successive conceptions (Rosenzweig & Schultz AER paper) and increased risks of premature birth. Within a household, competition for limited family resources among the siblings can intensify proportionately with increasing family size (Olsen & Wolpin 1983) and even reflect parental preferences for 'wantedness' of their children (Simmons et. al. 1982). Again supply-side interventions that help reducing fertility, for e.g. family planning programs, can also improve availability and accessibility of healthcare services, which in turn can improve child health and survival chances as well. This follows the 'simultaneity' hypothesis (Choudhury et.al. XXXX) which argues that a couple's fertility decisions and choice over health inputs are jointly made (Rosenzweig & Schultz XXXX), and accounting for such types of selectivity, fertility and child mortality are simultaneously determined (Panis 1992, as cited in Panis & Lillard 1996). Driven by the testable hypothesis that forms the major focus of this paper, we model infant survival as a function of fertility (and intertemporal changes), but apply heuristic corrections for 'feedback effects' of mortality on fertility. The reduced-form specification employed also addresses concerns over selectivity and simultaneity of demand for child health inputs. This is explained in greater detail while introducing the model and empirical specification followed.

III. Data and Methods

We use data from consecutive rounds of National Family Health Survey in India, using the state-level datasets to generate age-cohorts of ever-married women. Sample averages for these age-cohorts were used as model parameters. We then match the age-cohorts to their counterpart from the following round of NFHS to derive a pseudo-panel dataset of age-cohorts of women following the approach outlines in Deaton (1997). For e.g. data for women of age-cohort 20-25 from NFHS-I (1992-93) is matched with age-cohort data of women aged 26-31 from NFHS-II (1998-99) allowing the initial age-cohort to ‘grow’ by the interval, as in the case of a conventional panel. Such approach is however fraught with possible biases caused due to averaging age-cohort values, and such measurement error calls for estimating errors in variables models. In the initial exercise, we rather have *a-priori* approach of allowing a 10% error in our major variable of interest rates, viz. the age (cohort)-specific fertility rates. Later analysis tests for our models with an alternative source of data which allows simultaneous assessment of the robustness of initial model parameter estimates as well as have a more grounded correction for measurement errors.

The empirical model which serves as the theoretical underpinning for our analysis is a modified form of the child health production function approach, due initially to Becker (1969)

and formalized by Schultz (1984). We start with reduced form models under strong assumption of exogeneity of model predictors, but later relax to accommodate and correct for potential problems of endogeneity. Instrumenting fertility change with lagged values of our fertility variable, we test for rejection of a non-zero coefficient on the variable.

Summary results from the model are as below:

Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
x1hat					0.467	
					0.0001	
x2hat						0.449
						0.0002
delasfr	0.017	0.006	0.474	0.438		
	0.9200	0.9469	0.0067	0.0067		
delfemale	-113.567	-125.329	-67.046	-55.708	-74.150	-51.978
	0.1097	0.0604	0.3898	0.4758	0.2484	0.4554
delurban	-152.682	-167.643	-98.217	-60.568	-110.378	-69.510
	0.0411	0.0013	0.2024	0.5590	0.0295	0.3728
delnorm_as~x	3.254	4.284	2.916	-0.066	3.848	0.036
	0.0219	0.0192	0.0396	0.9666	0.0350	0.9921
delmam			-32.438	-31.370	-31.286	-28.628
			0.0006	0.0014	0.0000	0.0012
delscst	114.529	114.788	113.168	212.089	113.395	220.084
	0.0010	0.0000	0.0019	0.0002	0.0000	0.0000
dellfpr	36.501	44.856	51.617	143.931	59.717	138.076
	0.5822	0.3507	0.4416	0.1071	0.1949	0.0206
deledu_sy	-17.585	-19.512	-5.869	2.115	-6.457	3.283
	0.0135	0.0011	0.4791	0.8025	0.2983	0.7280
N	336	336	336	336	336	336

Model 1 is a pooled OLS, Model 2 assumes strict exogeneity, but allows errors in variables of the fertility change ('delasfr') variable, Model 3 allows for endogeneity - instrumenting fertility change with lagged value of ASFR, Model 4 Additionally allows state-level fixed effects, Model 5 recognizes both measurement error & endogeneity, Model 6 allows state-level fixed effects in EIV models