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Child mortality risk and fertility: Evidence from prevention of mother-to-child transmission of HIV☆

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ABSTRACT

A fundamental question in development and growth is whether and how fast fertility responds to reductions in child mortality risk. I use the expansion of prevention of mother-to-child transmission of HIV (PMTCT) in Zambia to provide some of the first quasi-experimental evidence on this question. My results suggest that the local introduction of PMTCT reduced pregnancy rates by approximately 10%, particularly among likely HIV positive women and women in locations where PMTCT was available for a longer duration, and that PMTCT substantially increased breastfeeding rates.

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1. Introduction

A fundamental question in development and growth is whether and how fast fertility responds to reductions in child mortality risk. As standards of living rise and public health efforts improve, poor countries have experienced large reductions in child mortality.[1] Standard economic models of fertility suggest that fertility may fall in response to increases in child survival and household investment in children's human capital may increase (Becker and Gregg Lewis, 1973; Lee, 2003; Schultz, 1997).[2] However, other theories of fertility do not necessarily generate this prediction and there is a classic concern that fertility may not respond to child mortality reductions, leading to rapid population growth and possibly reducing income per capita (Acemoglu and Johnson, 2007; Malthus, 1798; Preston, 1975).

A major obstacle to answering this question is the lack of experimental evidence on the effects of child mortality risk (Schultz, 1997). Many studies are purely theoretical (e.g., Becker and Gregg Lewis, 1973; Sah, 1991; Soares, 2005) or rely solely on non-experimental variation in child mortality and fertility (e.g., Angeles, 2010; Ben-Porath, 1976; Boucekkine et al., 2009; Hossain et al., 2007; Kalemli-Ozcan, 2002; Wolpin, 1984). Other studies that exploit quasi-experimental variation in disease risk (e.g., Acemoglu and Johnson, 2007; Durevall and Lindskog, 2011; Fortson, 2009; Juhn et al., 2013; Kalemli-Ozcan, 2012;

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[1] For example, consider countries in the two poorest regions of the world over the past 20 years. Between 1990 and 2010, under-5 child mortality (per 1000 live births) fell from 116 to 63 in Southern Asia and from 178 to 112 in Sub-Saharan Africa (UNICEF, 2012).

[2] Bleakley and Lange (2009) provide evidence from hookworm eradication in the American South that the reduction in child morbidity increased household investment in children's schooling.

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Kalemli-Ozcan and Turan, 2011; Lucas, 2013) are unable to isolate the effect of child mortality risk from adult mortality and morbidity risk.

I exploit the rapid expansion of a critical reproductive health technology in Zambia designed to reduce child mortality risk by reducing mother-to-child transmission (MTCT) of HIV.[3] Antiretroviral drugs for the prevention of mother-to-child transmission of HIV (PMTCT) reduce the cumulative probability of transmission from mother-to-child by as much as 45% in the absence of PMTCT to as little as 3% (Canning, 2006; Dabis and Ekpini, 2002). As shown in Fig. 1, between 2000 and 2007, the number of health facilities in Zambia offering PMTCT increased from fewer than 6 to nearly 600 (or 40% of facilities).[4] In a country where the representative woman should expect to experience roughly 0.4 child deaths due to HIV over the course of her adult lifetime, this expansion should have generated large reductions in expected child mortality risk. [5]Approximately 8% of children in Zambia are born HIV positive or acquire HIV through breastfeeding in the absence of PMTCT (Ministry of Health, Zambia, 2008; Torpey et al, 2010), of whom it is estimated that nearly 50% die by age 1 (Dabis and Ekpini, 2002).[6] A total fertility rate (TFR) in Zambia of 5.91 (Fortson, 2009) means that the representative woman in Zambia should expect to experience roughly 0.4 child deaths due to HIV over the course of her childbearing years.[7] Although PMTCT generally refers to a package of interventions including HIV testing and breastfeeding advice, during the period I examine in the current analysis (i.e., 2007 and before), the national PMTCT program in Zambia focused primarily on providing antiretrovirals for PMTCT.[8] Because the short course of antiretrovirals for PMTCT does not directly affect adult mortality risk and few women in Zambia who received PMTCT during the period I examine also received antiretrovirals as part of an ART program for adults (UNICEF, 2010), PMTCT plausibly reduced child mortality risk without directly affecting adult mortality risk.

My empirical strategy is a difference-in-differences approach. I augment the Japanese International Cooperation Agency 2006 Health Facilities Census, which includes the GPS coordinates of each health facility in Zambia, with newly assembled data on the month and year each facility began offering PMTCT. Data on reproductive behavior and fertility outcomes come from repeated cross-sectional national household surveys conducted before, during, and after the scale-up, where each survey year includes respondents from multiple interview months. Combined with detailed geographic information on the location of survey households, I use these data to measure how reproductive behavior changes when PMTCT is introduced at a local health clinic. Multiple rounds of survey data before the introduction of PMTCT, as well as multiple rounds after the introduction PMTCT, mean that I am able to identify the effect of PMTCT on fertility while controlling for unobserved time-invariant characteristics of locations receiving PMTCT and differential trends between PMTCT and non-PMTCT locations.

I find evidence that the local introduction of PMTCT may have reduced pregnancy rates, particularly among demographic subgroups for whom theory predicts that we might observe a larger response. Point estimates from a variety of empirical specifications suggest that local PMTCT availability reduced pregnancy rates by approximately 2 to 4 percentage points, or roughly 10% relative to the mean pregnancy rate. Consistent with a causal effect of local PMTCT introduction on fertility, I find evidence suggesting that the fertility reduction was concentrated among women who were more likely to be HIV positive and among women closer to the health facility where PMTCT was locally introduced. Likewise, a semi-parametric difference-in-differences analysis suggests the absence of a pre-local introduction "effect" and that the medium term response to local PMTCT introduction was larger than the short term response. I also find that local PMTCT availability increased breastfeeding rates, an important input into child quality, by between roughly 5 and 20 percentage points, or approximately 10% relative to the mean breastfeeding rate.[9]

There are at least two important caveats about these findings. First, in the full sample of respondents (i.e., including likely HIV negative women and women in locations where PMTCT has only been available a short period of time) simultaneously controlling for the availability of the two other main HIV/AIDS services (i.e., VCT and ART) and for two other major determinants of child mortality (i.e., piped water and bed nets) reduces the magnitude of the point estimate and increases the estimated standard error. This makes the fertility response statistically insignificant from zero in the full sample. Of course the results indicate a large and statistically significant reduction in fertility among the two subgroups for whom theory suggests we should observe the largest responses to local PMTCT introduction (i.e., likely HIV positive women and women in locations where PMTCT has been available longer). Moreover, VCT, ART, and PMTCT expansion in Zambia was highly correlated, making it difficult to separately identify the effect of PMTCT while conditioning simultaneously on ART and VCT availability.[10] Notably, controlling for either ART or VCT alone does not dramatically affect the estimated effect of local PMTCT availability.

Second, although pregnancy rates appear to have fallen in response to local PMTCT availability, population growth may have increased, particularly in the short term. As I discuss in Section 6.5, the fertility reduction implied by the estimated average effect of local PMTCT availability on pregnancy rates (i.e., equivalent to a 0.4 child reduction in TFR) is approximately equal to the expected child mortality reduction if PMTCT were to eliminate MTCT of HIV.

Although there is little quasi-experimental evidence on the effect of child mortality risk on fertility, this paper contributes to several related bodies of empirical literature. First, numerous studies (e.g., Aksan and Chakraborty, 2012; Angeles, 2010; Ben-Porath, 1976; Boucekkine et al., 2009; Hossain et al., 2007; Kalemli-Ozcan, 2002; Wolpin, 1984; Young, 2005, 2007) examine the effect of child mortality risk on fertility using non-experimental data. Second, other research (e.g., Bleakley and Lange, 2009) examines the effect of child morbidity risk on fertility. Third, there is research (e.g., Lucas, 2013) that examines the effects of diseases (e.g., malaria) that simultaneously affect child mortality/morbidity risk and adult mortality/morbidity risk or examine the effects of

[3] Several facts make Zambia an interesting case study. Adult HIV prevalence in Zambia is approximately 15% (Central Statistical Office et al., 2009), making it the 5th highest HIV prevalence country in the world (UNAIDS, 2010). With a total fertility rate (TFR) of approximately 6, Zambia is the 10th highest fertility country in the world (World Bank, 2013).

[4] This paralleled a broader scale-up of PMTCT throughout much of Sub-Saharan Africa. Between 2005 and 2008, the proportion of HIV positive pregnant women receiving PMTCT increased from 15% to 45% and the proportion of infants born to HIV positive mothers receiving PMTCT increased from 11% to 31% (WHO, 2010a).

[5] Adult HIV prevalence in Zambia is nearly 15% (Central Statistical Office et al., 2009).

[6] There appears to be limited evidence on the mortality-age profile for HIV positive infants who live past the age of 2 (Dabis and Ekpini, 2002). Presumably in this study setting the vast majority do not survive into adolescence.

[7] HIV/AIDS accounts for 12% of under-5 child mortality in Zambia (WHO, 2010b). Although data do not appear to be available for mortality shares at younger ages, HIV/AIDS likely accounts for a higher fraction of under-2 mortality than under-5 mortality because roughly one-half of HIV positive infants die by age 2 (Dabis and Ekpini, 2002).

[8] In Section 2 and in Section 6.5, I describe in more detail the evidence supporting this claim.

[9] This is consistent with the predictions of a quantity–quality model, in which a decrease in child quantity should be associated with an increase in child quality (Becker and Gregg Lewis, 1973). However, there exist at least three alternative interpretations. First, the increase in breastfeeding could be a response to the decreased risk of infecting one's child through breastfeeding. Second, widespread use of lactational amenorrhea as a contraceptive method in the developing world means that breastfeeding may simply be the mechanism by which women are decreasing pregnancy rates. Third, PMTCT services may include recommendations to breastfeed.

[10] Among female respondents in the 2007 Demographic Health Survey (i.e., the latest survey round I use in the analysis), the correlation between access to PMTCT, ART, and VCT (i.e., living with 20 km) is as follows. The correlation between PMTCT and VCT access is 0.80. The correlation between PMTCT and ART access is 0.68. The correlation between VCT and ART access is 0.59.

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