Abolishing User Fees, Fertility Choice, and Educational Attainment*

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November 2015

Abstract

This study examines the effect of abolishing user fees from health care services on fertility and educational attainment as a test of the quantity-quality tradeoff model. Exploiting sudden improvements in nutritional status among South African children post-apartheid as producing an exogenous decline in the price of quality investments, we document evidence consistent with the model showing that parents lowered fertility and increased their investment in education. The absence of treatment effects on both fertility and education among children not subject to the health policy eliminates channels through heterogeneous preexisting trends or unobserved concurrent changes. In addition, we explore and reject a number of alternative hypotheses that may account for the observed relationship. Important policy implications of our findings are: (i) parents in developing countries successfully adjust their fertility behavior in response to increased returns to child learning; and (ii) health policy serves as a motivating force underlying the demographic transition and human capital development.

Key words: user fees, fertility, education, South Africa
JEL codes: J13, I15, O15, I18

* We appreciate Jenny Aker, Laurie DeRose, Nancy Hite, Seema Jayachandran, Ryo Kambayashi, Daiji Kawaguchi, Takashi Kurosaki, Dilip Mookherjee, Daniele Paserman, Adam Storeygard, Rodrigo Wagner, Hiroyuki Yamada, Wesley Yin, and numerous participants at seminars and conferences for helpful comments. Financial support from the Grant-in-Aid for Young Scientists (Start-up) (Grant No. 21830058) from the Japan Society for the Promotion of Science is gratefully acknowledged. All remaining errors are our own.
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I. Introduction

Economists have long been interested in explaining the historical demographic shift from high to low fertility rates. The stylized fact that this demographic transition has almost always been coupled with human capital development and economic growth has given rise to the theoretical conjecture of a tradeoff between child quantity and quality (QQ) (Becker 1960; and Becker and Lewis 1973; Becker and Tomes 1976; Hanushek 1992; Galor and Weil 2000; Kalemli-Ozcan 2002, 2003; Tamura 2006).

However, the extent to which, or even whether, fertility can be portrayed as a matter of parental choice among poor households in developing countries remains at the heart of development policy debates. An empirical test of these theories has been impeded by difficulties in isolating or even measuring variations in prices of child quantity/quality that are endogenously and jointly determined. For example, the conventional model explains that an increase in quality of children raises the shadow price (marginal cost) of quantity of children and vice versa, and thereby making it difficult to establish a causal relationship. More importantly, the shadow prices that play the central role remain unobserved.

This study presents empirical evidence to assess the theoretical prediction that the price change in child quality investments induces parents to substitute quantity with quality. The mechanism we focus on in this study differs from those of the existing empirical studies in three distinct aspects. First, a number of economic theories have attributed the decline in fertility to historical changes in factors raising the opportunity costs of a marginal child, namely the price of child quantity, due, for instance, to increased adult wages (Becker, Murphy, and Tamura 1990; Hazan and Berdugo 2002), or elevated women's status (Mincer 1963; Galor and Weil 1996; Lagerlöf 2003). Empirical evidence regarding the interaction between quantity and quality has also predominantly concentrated on research contexts involving constraints on the *quantity* of children, i.e., using exogenous changes in the number of children parents choose to have due to unexpected incidences, such as twin births or same-gender siblings (Rosenzweig and Wolpin 1980; Angrist and Evans 1998; Angrist, Lavy, and Schlosser 2005; Black, Devereux, and Salvanes 2005) or economic incentives for child-bearing, such as changes in relative female wages (Schultz 1985, 1986) or financial support for obstetric care (Boyer 1989; Whittington, Alm, and Peters 1990; Zhang, Quan, and Van Meerbergen 1994; Acs 1996; Kearney 2004; Milligan 2005; Laroque and Salanie 2008; Cohen, Dehejia, and Romanov 2013).

In contrast, there has been little empirical investigation regarding the fertility response to relaxing constraints on the *quality* of children.[1] Our research design takes advantage of sudden and exogenous improvements in nutritional status during early childhood in post-apartheid South Africa as a price decline in child quality investments. Improved nutritional status during early childhood increases efficiency in learning in an environment where low health status is a major impediment to learning in school, thereby effectively lowering the price of quality investments. We explore whether this shock in turn induces parents to invest more in human capital and reduce fertility.

Second, we shed light on a mechanism linking contemporaneous adjustments of fertility to predicted future welfare of children, whereas most previous literature focuses on adjustments to contemporaneous economic conditions. Our view is consistent with overlapping-generations models of endogenous fertility, in which parents invest in their children either as a form of parental altruism toward their offspring (Becker 1990; Becker and Barro 1988; Barro and Becker 1989; Becker, Murphy, and Tamura 1990) or for the sake of their own old age support (Ehrlich and Lui 1991; Boldrin and Jones 2002). Both of these mechanisms provide theoretical support to the fertility reduction along the path of adjustment when parental investments to child quality rise.[2] However, there is still little empirical evidence available regarding whether parents in the developing world make contemporary behavioral responses to future economic incentives.

Third, we identify a specific health policy as the trigger for the demographic transition, namely the abolition of user fees from healthcare services in the aftermath of the dismantlement of apartheid in 1994 in South Africa. Assessing the benefits of free health services is important in its own right, as there is increasing interest among development policy makers in the question of whether user fees should be charged or abolished from basic health services.[3] While a growing

[1] For example, Galor and Weil (2000) develop a unified endogenous growth model, in which increased returns to education due to technological progress are followed by reductions in fertility and increases in human capital investment. However, empirical evidence focusing on the fertility response to the quality shock is scarce. An exception is Bleakley and Lange (2009), who consider the hookworm eradication in the United States as the increased return to child quality when the burden of the disease considerably depressed the returns to learning. Along with the findings in Bleakley (2007), they show that a fertility decline was at the root of substantial improvements in school attendance and literacy a decade after the intervention took place. Also, Aaronson et al. (2014) show a smaller number of children along the intensive margin in response to greater schooling opportunities for children.

[2] For example, in models considering intergenerational effects from parents to children, the utility of children becomes an important component of the parental utility function. Increased investments in child education, therefore, raise the marginal cost of a child, as constrained by budget limitations, leading to the negative associations between quantity and quality of children. Moreover, in models in which intergenerational effects run from children to parents as parents seek care from children in their old age, parents maximize their future transfer from children in relation to the cost of rearing children, again producing the quantity-quality tradeoff.

[3] Whether free distribution or cost-sharing achieves an efficient allocation of basic health goods has recently been debated. Pricing has been widely adopted as a means to target individuals in need of a good and increase usage thereof. Recent evidence, primarily derived from randomized controlled trials, however, suggests that charging even a small price substantially reduces usage of health goods (i.e., insecticide-treated nets, deworming drugs, or water disinfectant), due in large part to resultant crowding out of the poor in need of these goods (Kremer and Miguel 2007; Holla and

number of African countries have begun abolishing user fees in the last decade, the introduction of such policies at the national level renders a rigorous assessment difficult. On the one hand, the estimated effects are likely to be overstated by unobserved time factors (Lagarde and Palmer 2008), while on the other hand, estimates will prove to be substantially understated if the health improvements experienced during early childhood lead to the onset of demographic transition and economic growth in the long run. We know of no other study that exploits a health care policy as a motivating factor underlying such fertility and human capital development process.

In this regard, our study also adds to a growing empirical literature on the long-run effects of early childhood health status. Existing studies find that health status, particularly early childhood nutrition, has substantial long-term and irreversible economic impacts on later outcomes. It has been shown that children who experienced positive (negative) health shocks during early childhood perform better (worse) in school, earn higher (lower) income during young adulthood, and achieve higher (lower) health and socioeconomic status up to middle age.[4] The critical-period hypothesis is often assumed to be the phenomenon whereby early childhood health correlates to later educational outcomes, but the precise mechanism remains unknown. We highlight the fertility reduction as a linkage between the two.

To establish causality, it is critical to identify the source of health improvements as plausibly exogenous. To this end, we exploit salient features of South Africa's history of apartheid. For more than four decades up to 1994, blacks in South Africa had limited political representation as well as limited ability to choose their place of residence. The control exercised by whites over the allocation of health resources, unrelated to the demand of black Africans for these resources, resulted in little correlation between availability of health facilities and household characteristics across communities, limiting the scope of heterogeneous preexisting trends. Using a similar research design, a previous study by Tanaka (2014) shows that the removal of user fees in South Africa in 1994 led to substantial improvements in nutritional status among children in communities with a health facility relative to those without, in the period between 1993 and 1998. The present study investigates the longer-term effects a decade later of that earlier removal of user fees on educational outcome and fertility decisions as a test of the QQ model.

Kremer 2009; Cohen and Dupas 2010; Ashraf, Berry, and Shapiro 2010; J-PAL 2011). These studies lend strong support to free distribution of health goods over cost-sharing as a way to enhance equity in access to health goods and services and to improve health status.

[4] The literature along with the fetal origins hypothesis is summarized in Almond and Currie (2011). Further evidence for the long-term impacts of early-life health status is found in Alderman et al. (2001), Glewwe, Jacoby, and King (2001), Glewwe and King (2001), Case, Lubotsky, and Paxson (2002), Case, Fertig, and Paxson (2005), Alderman, Hoddinott, and Kinsey (2006), Almond (2006), Bleakley (2007), Dinkelman (2008), Yamauchi (2008), and Maccini and Yang (2009).

Our findings are consistent with the theoretical prediction that abolishing user fees from health services would induce parents to increase educational investment and to reduce fertility between 1993 and 2004. We conduct falsification tests that show the absence of treatment effects on both child quantity and quality among the cohorts not subject to the health policy, which eliminate potential channels such as heterogeneities in preexisting trends or unobserved concurrent changes in educational reforms. In addition, we explore and discard a number of other potential mechanisms that may account for the observed relationship, such as effects through mortality, access to family planning and contraception practices, a price change of quantity, an educational reform, and migration. Overall, our findings suggest that improved nutritional status affected child quality and quantity by means of its effect on the price of investment in child quality. These findings hold important implications for fertility behavior among parents in developing countries by demonstrating that appropriate economic incentives can induce demographic transition, obviating the necessity of resorting to intense population policies to curb fertility growth.

The remainder of the paper is structured as follows. Section II describes the historical background of health policy and education in South Africa and the effect of new health policy on nutritional status among children. Section III illustrates the conceptual framework that highlights the key mechanism through which health improvements in early childhood affect fertility and educational attainment via the impacts on the price of quality investment. Section IV describes the data set used in the main analysis, its summary statistics as initial evidence, an econometric framework, and discusses the validity of its identification assumptions. Section V reports empirical results from the main and falsification/robustness analyses, and Section VI explores and tests additional alternative hypotheses. Section VII concludes.

II. Background

A. History of Health Policy and Education in South Africa

Apartheid in South Africa has an enduring legacy as one of the most discriminatory regimes in modern history. In a society characterized by extreme racial segregation, black Africans constituted the poorest and most underserved group, suffering the greatest oppression by whites in all aspects of their lives. For the purpose of our research context, two salient features of that regime were: restricted residential choice and unequal allocation of resources among black African communities.

The Bantu Authorities Act of 1951 confined black Africans within impoverished so-called "homelands," not only externally determining their place of residence but also prohibiting them from freely migrating within the country. Further, the white minority in urban areas controlled almost all resources without representation from the black majority. As a consequence, few resources were allocated to black African communities, and the meager resources thus allocated were distributed in a random fashion unrelated to local demand. Under these circumstances, the overriding issue among children was the disparity and inequality in nutritional status and educational performance across races and geographical areas.

With respect to health, the absence of coordination and integration among as many as eighteen central and regional departments of health resulted in unequal distribution of health resources across and within provinces. In addition, there was neither a comprehensive health policy nor a central institution to coordinate health plans and practices at the national level. As a result, black Africans suffered from substantially low health status due to poor access to health services, constrained by costly out-of-pocket payments for health services as well as by a dearth of facilities, doctors, and medicines in the public health sector.

The new democratic government established in 1994 undertook immediate efforts to ensure equal access to public services. The most remarkable policy in the health sector was the abolition of user fees from healthcare services to pregnant women and to children under 6 years of age at public health facilities such as hospitals and clinics. These services included pre- and post-natal services until 42 days after delivery as well as primary care services to children, such as maternal nutrition, breastfeeding assistance, nutrition education, child immunization, growth monitoring, nutritional promotion, and micronutrient supplementation.

Among black Africans, for whom the cost of medical services had been a major impediment to accessing treatment, the abolition of user fees contributed to substantial increases in the utilization of health facilities among pregnant women and children under 6 years of age in (McCoy 1996; Department of Health 1998; Schneider and Gilson 1999; Wilkinson et al. 2001; Cooper et al. 2004; Morestin and Ridde 2009).

In the educational sector, on the other hand, progress in eliminating racial disparities was more limited, despite significant efforts to equalize the allocation of government funds. Two facts are especially important for our research purpose. First, most black Africans continued to perform poorly in school due in large part to differences in school resources, fees, and curricula that began under apartheid (Fiske and Ladd 2004; Yamauchi 2005; van der Berg 2007; van der Berg and Louw 2007; Bhorat and Oosthuizen 2008). Although the demise of apartheid freed black African students from severe restrictions on school choice, most remained in schools with poor

infrastructure (Lam, Ardington, and Leibbrandt 2011). This averts endogenous sorting of black African students across schools, creating little correlation between household characteristics (i.e., the income and educational background of parents) and the quality of schools their children attended (Case and Deaton 1999).

Second, the degree of variation in student performance remains high, even after controlling for household and school characteristics (Case and Deaton 1999; Crouch and Mabogoane 1998, 2001; Hoadley 2007; van der Berg 2007; Bhorat and Oosthuizen 2008; van der Berg and Shepherd 2008), a phenomenon which Lam, Ardington, and Leibbrandt (2011) call "schooling as a lottery."[5] The weak explanatory power of school inputs and household attributes in relation to educational performance leaves the identification of key factors contributing to better educational performance an open question.

B. Free Healthcare Services and Nutritional Improvements Among Children

Access to health services is one of the most important contributors to nutritional status, particularly among poor black African children who were not previously reached by such services.[6] Tanaka (2014) provides three important findings for our present research context. First, he finds that the abolition of user fees from health services led to immediate and substantial improvements in nutritional status among newborns between 1994 and 1998, allowing us to exploit such nutritional improvements as a shock to price of child quality investments. Second, he identifies the source of variation in health improvements as plausibly exogenous, namely variation in infrastructure across communities under apartheid. We present similar evidence below that household characteristics are *ex-ante* similar, reflecting the historical fact that infrastructure was predominantly controlled by whites unrelated to demand by black Africans. Third, these improvements in health status were not concentrated on the lower tail of the health status distribution, indicating that the mean shift in health status was not entirely due to shrinkage of the low health status cohort. The important implication is that free healthcare does not appear to have contributed to mortality reductions, ruling out a mechanism through which fertility

[5] There are several possible explanations for such variation. On the one hand, Hoadley (2007) points to the frequently chaotic classroom environment as well as ineffective classroom and school management, and on the other hand, van der Berg and Shepherd (2008) attribute the problem to poor internal assessment of student performance. Lam, Ardington, and Leibbrandt (2011) establish a model with evidence to support the claim that a stochastic linkage between actual ability and measured performance has led to high enrollment and high repetition rates among black African schools.

[6] Clearly another important contributor is the quality of medical services, including factors such as the range of medical services, the availability of drugs and equipment, the knowledge and skill levels of physicians and staff, and the quality of facilities. It is important to note here that the importance of the quality of healthcare should not be underestimated as the interactions between quantity and quality are key to promoting further improvements in public health. However, we emphasize access, or the quantity-side, more in a circumstance where prospective patients have poor access to begin with. A small increase in the utilization of health facilities from virtually zero visits should result in substantial improvements in health status whether we assume linearity or concavity in the relationship between access and nutritional status.

reductions responded to the likelihood of child survival. We investigate the effect on mortality in greater detail in Section VI.

III. Conceptual Framework

Originating with Becker and Lewis (1973), the parental utility function typically considers child quantity, *n*, and quality, *q*, in which parents decide the optimal *n* and *q* simultaneously:

$$\text{Max } U = U(n,q,x),$$

where $\partial U/\partial k = U_k > 0, \partial^2 U/\partial k^2 = U_{kk} < 0$ for $k = (n,q,x)$, and x is a set of other goods and services, subject to the budget constraint:

$$I \leq \pi_n n + \pi_q nq + x,$$

where I is the household income, $\pi_n n$ represents fixed expenditures on child quantity that is independent of child quality, and $\pi_q nq$ represents costs of quality investments into children (with the standard assumption that child quality is identical across children within family).

The equilibrium conditions at (n^*,q^*) highlight the marginal costs, or the shadow prices or quantity and quality to be;

$$U_n = \lambda(\pi_n + \pi_q q^*) = \lambda p_n$$
$$U_q = \lambda \pi_q n^* = \lambda p_q,$$

where λ denotes the marginal utility of income. These two equations illustrate an important tradeoff between child quantity and quality. In particular, the first equation shows that an increase in child quality, *q*, raises the marginal costs of having additional children, whereas the second equation shows that an increase in the number of children, *n*, raises the marginal costs of investing in child quality.

Most studies have focused on pathways from quantity to quality. For example, increases in relative female wages are associated with increases in the marginal costs of child quantity, p_n, which in turn decreases *n*, while the marginal costs of quality fall as *n* falls from the second equilibrium condition, and thus the quality of children is upgraded. Financial incentives to having additional children work in a similar way: a decrease in marginal costs of child quantity increases *n*, and thus increased marginal costs of quality will lead to lower quality of children. The literature that exploits exogenous changes in the number of children due, for instance, to multiple births, directly affects *n*, without referring to changes in the marginal costs of child quantity, which then leads to an increase in marginal costs of quality and thus a decrease in quality.

The causal pathway we investigate in this study differs by focusing on quality change as it affects quantity. Improvements in health status are expected to improve education outcomes and lower barriers to investment in child quality (in a typical environment where low health status is a major impediment to learning). This effectively lowers the marginal costs of quality investments, p_q, which should increase the level of such investment. Increased investment in quality is associated with increased marginal costs of quantity, as described in the first equilibrium condition, and thus fertility is expected to fall.[7]

IV. Research Design

A. Data Sources

We use longitudinal datasets from the KwaZulu-Natal Income Dynamics Study (KIDS).[8] KwaZulu-Natal province is the second largest province in South Africa, representing approximately 20 percent of total population in 2011, the majority of whom are black Africans. Importantly, KwaZulu-Natal province shares common attributes with other former homelands, such as high rates of poverty and absence of basic services (Klasen 1997; Leibbrandt and Woolard 1999; May et al. 2000).

We merge the first wave in 1993 (hereafter KIDS93) and the third wave in 2004 (hereafter KIDS04) to trace the long-run adjustments over time. The first wave was conducted as part of the first comprehensive national household survey, the Project for Statistics and Living Standards and Development, and thus it provides information prior to the new health policy reform. The subsequent waves in the post-reform period revisited only black Africans and Indians in KwaZulu-Natal province. Although the second wave was conducted in 1998, the longer period of time between the first and the third waves enables us to trace children from early childhood to primary school age. These datasets report detailed information on key variables, including educational attainment and the number of children, as well as other important individual and household characteristics, such as age, gender, and education of all household members. Moreover, we can link the household surveys to the community surveys, which report infrastructure information, such as the number and types of healthcare facilities and primary schools in communities.[9]

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[7] Precisely speaking, fertility falls in response to increased educational return if child quality is elastic with respect to the cost of child quality (i.e., $\partial n / \partial p_q > 0$ if $[\partial q / \partial p_q][p_q / q] < -1$. Alternatively, the optimal number of children *increases* when the cost of educating a child falls if $[\partial q / \partial p_q][p_q / q] > -1$. See Galor (2012) for the discussions.

[8] For more detailed description of KIDS, see May et. al. (2000), and May et. al. (2007).

[9] Throughout the paper, we use the term "community" to refer to a census enumerator sub-district, the smallest geographical unit at which we can identify the health/education facility information.

The sample used in the main analysis is restricted to black Africans only in an effort to remove heterogeneities that may cause bias in our estimates. This is appropriate as the goal of the health policy was to reach out to disadvantaged groups, making black Africans particularly subject to the effect of the policy.

B. Summary Statistics

The baseline information regarding community-level infrastructure as of 1993 is presented in Table 1. Observations are at the community level. The first column shows the mean values of each respective variable, and the second column its standard deviation.

All variables for health facilities (clinics, dispensaries, hospitals, family planning clinics, and maternity homes) are dummy variables, being equal to one if there was at least one facility in the community. Regarding clinics, there was either only one or zero clinic in each community in 1993, and thus the mean value indicates 48 percent of communities had clinical availability as well as 0.48 clinics existed on average per community. On the other hand, dispensaries or hospitals barely existed in black African communities. These observations are consistent with evidence that clinics served as the main facility where black Africans received pre-natal services and initial treatments. For our purposes, as discussed below, high-treatment regions are defined as communities with at least one medical center (clinic, hospital, or dispensary); all other communities are defined as low-treatment regions. The last row indicates that 41 percent of the total 56 communities (equivalent to 23 communities) constitute the high-treatment region. The variables for primary and secondary schools are in numbers; the table shows 1.86 primary schools and 0.82 secondary schools on average per community. It is clear that these communities lacked both educational and health facilities.

Panel A of Table 2 presents summary statistics on variables used for child quality analyses for the sample of black African children aged 7 to 14, using observations from KIDS93 and those from KIDS04 separately. Note that those aged 14 or younger in 2004 were 4 years old or younger in 1994 and thus had full exposure to free health services including a prenatal period. We use educational attainment, that is, grade level completed, to measure the parental investments in child quality. It is worth noting that the use of educational attainment instead of years in school avoids bias arising from grade repetition, which is highly prevalent among these students. Average educational attainment is about 3 years for cohorts of children with an average age of 10 years old in 1993, whereas average educational attainment had increased to 3.8 years for a similar age group in 2004.

Panel B of Table 2 presents summary statistics on key variables for child quantity for the sample of black African women aged 31 to 45. A comparison of children aged 8 or less between 1993 and 2004 measure the fertility dynamics in the post-reform period. We also present children aged 11 to 19 to characterize a fertility trend in the pre-reform period. On average, there were 0.95 children aged 8 or less and 1.25 children aged 11 to 19 per mother in 1993. Both of these figures had fallen in 2004; there were 0.53 children aged 8 or less and 0.97 children per mother. The reduction in fertility even among cohorts born before 1994 suggests that a simple comparison of fertility over time may confound a preexisting time trend. We describe our econometric framework that constructs a counterfactual and adequately controls for a time trend below.

C. Econometric Framework

Two important sources of variation constitute our empirical framework to identify the effect of free health services on child quantity and quality outcomes. First, user fees for health services were abolished in 1994, allowing us to observe pre-reform conditions from KIDS93 and to observe long-term outcomes in the post-reform period from KIDS04. Second, although the policy was implemented simultaneously at the national level, households in communities with existing health facilities had greater intensity of exposure because they gained immediate access to healthcare services, while those in communities without any health facility had to travel long distances to receive treatment or wait until facilities were built in their own communities.[10] Thus, we define the high-treatment region as communities where there was any medical institution, in particular a clinic, hospital, or dispensary, in 1993. We use the interaction of these two variations in a reduced-form equation of the difference-in-differences (DD) framework to measure the effect of free health services on outcomes of interests.

Specifically, we estimate;

$$Y_{ickt} = \alpha_0 + \alpha_1(High_c \times Post_t) + X'_{it}\boldsymbol{\alpha}_2 + (W'_c \times Post_t)\boldsymbol{\alpha}_3 + \gamma_k + \mu_c + \epsilon_{ickt} \quad (1)$$

in which i indexes individual, c denotes community, k denotes birth cohort, t denotes year, $High_c = 1$ for the high-treatment region, and $Post_t = 1$ for $t = 2004$. A vector of variables, X_{it}, controls for key individual and household characteristics.[11] The community characteristics (W_c)

[10] Indeed, the government reported that the construction of health facilities in Kwazulu-Natal province was extremely slow due to political instability and violence (Cameron 1996; Khan, Lootvoet, and Vawda 2006); even the first democratic election did not take place until 1996. Such evidence supports our assertion that communities that did not have health facilities in 1993 continued to have no health facilities for several years afterwards, creating variation in access to healthcare services.

[11] For the quality analysis, the individual level of variables control for a female dummy and first-born dummy, while the household level of variables include mother's education, mother's age, the average educational attainment among adult members, and the number of adult members. The definition of "adult members" should be considered with care.

control for the number of primary and secondary schools in 1993; distance to primary and secondary schools, market, and bank in 1993; (log of) population in 1993; and are interacted with the post dummy, addressing the concern that there may be differential trends in the outcome variable correlated with baseline community characteristics in 1993. Additionally, we include the community fixed effects, μ_c, which help purge any time-invariant community characteristics, and birth-year cohort fixed effects, γ_k, to account for year-specific shocks common across all individuals within a birth cohort. All standard errors are clustered at the community level, allowing possible correlations over time within communities.

The outcome of interests for the quality analysis is educational attainment by child i aged between 7 and 14. In calculating educational attainment, we focus on the number of years based on completed grades, rather than the actual number of years in school, as these should be most closely related to educational performance or grade progression in an environment where grade repetition is highly common.

The outcome of interests for the quantity analysis is the number of children aged 8 or less that woman i had given birth to for the sample of those aged 31 to 45 years old. Since the policy was reformed in 1994, these children from KIDS04 indicate post-reform fertility, whereas the same age groups from KIDS93 provide analogous information in the pre-reform period.[12]

To address the concern that the fertility variable might be biased by child deaths for these age groups, two pieces of evidence are important. First, Tanaka (2014) presents evidence that the relative increase in nutritional improvements in the high-treatment region due to increased access to health services is not concentrated on the lower tail of the distribution. Instead, the reductions in the proportion of children with extremely low nutritional status were similar between the two regions. This is indeed not surprising because the services children obtained were consultations or relatively simple treatments such as growth monitoring and curative services upon being sick. These services were likely to have affected the nutritional status among children while they are less likely to have resulted in substantial reductions in mortality (which typically responds only to

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If siblings of our sample are included as adult members, adult members' education may suffer from bias due to endogeneity, since there is a possibility that siblings' education level and our outcomes (children's quantity and quality) are determined simultaneously. To avoid this possibility, we define adult members as those over 30 years of age. For the quantity analysis, the individual-level variables include educational attainment and the household-level variables include the average educational attainment of adult household members as well as the number of adult household members.

[12] Ideally, a precise history of childbirths and pregnancies over the 10-year period would yield a more exact measurement of fertility. Because the pregnancy history questions in both KIDS93 and KIDS04 do not report the year of pregnancy and suffer from low response rates (about 21% of women did not answer the questions), we infer fertility using the number of children aged 8 or less from the family roster.

larger-scale interventions).[13] Second, we present further evidence in Section VI A that mortality reductions were similar using available data.

The parameter of interests, α_1, essentially measures the contribution of free health services to the long-term effects on child quality and quantity outcomes across communities with distinct intensity of access to health facilities (equivalently distinct improvements in child nutritional status). The estimated effects would be biased if there were unobserved heterogeneities across these communities that were correlated with the evolutional path of the outcomes. We discuss the validity of such identification assumptions in the following subsection.

D. Validity of the Identification Assumptions

The validity of our identification strategy hinges on two important observations. First, the internal validity of the econometric framework requires an assumption that the availability of health facilities is plausibly exogenously determined. This is less likely to hold in contexts where rich households/communities can exert greater political power to bring in more resources. South Africa under apartheid, however, provides a rare case where this assumption is plausible because whites allocated resources to black Africans' communities in a rather random manner, over which black Africans had no control (Case and Deaton 1999).[14] Table 3 provides testable implications to support this assertion. We conduct the balancing test to investigate the correlation between the treatment status and various individual, household, and community characteristics in the baseline sample from KIDS93. Significant differences in characteristics may lead to severe bias in our analysis, as they indicate that the two types of regions were distinct.

As it turns out, we find signal evidence that the treatment status balances almost all baseline characteristics. Namely, there is no statistical difference in: demographic characteristics among all individuals in the dataset (years of educational attainment, age, and the ratio of female in Panel A); quantity investments observed from parental characteristics among women aged 20s to 40s in their reproductive history (i.e., number of pregnancies, number of births, number of births still alive, number of their children dying before age 1, number of their children dying between aged 1 and 5 in Panel B); quality investments observed in educational attainment among children aged 7 to 15 for all children and subdivided by gender in Panel C; household characteristics using all households in the dataset (household size, dependency ration, and

[13] Also note that Tanaka (2014) shows some decrease in nutritional status among older children, i.e. those who were not entitled to free healthcare, due to declining quality of services and the low morale of health providers. This suggests that little in the way of technological advancements that could have affected all patients, regardless of age, took place.
[14] Case and Deaton (1999) present evidence that school quality, measured by pupil-teacher ratios, which was extensively dispersed across black districts immediately before the end of apartheid, is not associated with socioeconomic characteristics among black families.

monthly income in Panel D); and community characteristics (the number of primary schools, distance to the nearest market, and distance to the nearest bank, except the number of secondary schools and population in Panel E).

Taken all together, these observations are consistent with the historical fact that the existence of health facilities under apartheid was based on an unknown, rather random rule determined by the white minority and unrelated to local characteristics, including demand or need, among black Africans. Therefore, these results provide no indication that unobserved heterogeneities would threaten the internal validity of our econometric strategy.

Second, in order to assess improved nutritional status as the mechanism through which free health services affected child quality and quantity, improvements in health status in the post-reform period had to have been substantially larger in the high-treatment region relative to the low-treatment region and have taken place exogenously to various channels other than free health services. We draw extensive supporting evidence from Tanaka (2014), who, using the same research setting, finds that the health policy reform resulted in sharp and statistically significant increases in nutritional status, as measured by weight-for-age z-scores and weight-for-height z-scores, between 1993 and 1998 in the high- as compared to the low-treatment region.

In addition, two pieces of compelling evidence from his findings are illustrated in Figure 1. The first shows that health status was virtually identical across cohorts in 1993 between the two regions (Panel A). This is either because children did not receive adequate health services even if there was a facility (i.e., due to budget constraint), or because the health services they did receive failed to improve health status (i.e., due to low quality of services). In either case, this finding precludes the existence of differential pre-trends. Further, children not entitled to free health services (i.e., those who were 6 years old and above as of 1994) did not experience a similar treatment effect, although they were exposed to all other changes in society and government policies (Panel B).[15] On the other hand, a sharp increase in health status is illustrated at the eligibility cut-off age indicated by a dotted line. This evidence eliminates effects through other concurrent changes in society.

All of the above pieces of evidence constitute the key ingredients for our analysis; we interpret the variation in health status induced by the abolition of user fees between the high- and low-treatment regions as an exogenous source of changes in the prices of child quality investments, which are otherwise difficult to exploit.

[15] The point estimates are indeed negative for these cohorts, indicating that there was a negative treatment effect of health policy among non-affected cohorts, if any, possibly due to overcrowding or lowered staff morale. Also note that the author is aware of no other contemporaneous policy that specifically focused on children under 6 years old.

V. Empirical Results

A. Effect on Child Quality

We start by investigating the effect of the policy change on later educational attainment for children aged 7 to 14. Panel A of Table 4 reports the estimated impacts on educational attainment. All specifications include community and cohort fixed effects. Column (1) presents the basic framework without any additional covariates, while Column (2) controls for the individual level variables (birth year dummies, a female dummy and first-born dummy), and household level variables (mother's education, mother's age, the average educational attainment among adult members, and the number of adult members); and Column (3) additionally controls for initial community characteristics (the number of primary/secondary schools in the community in 1993; distance to primary/secondary schools, market, and bank in 1993; and (log of) population in 1993) interacted with the post dummy.

The preferred estimate in Column (3) suggests that children in the high-treatment region had completed 0.410 more years of schooling than the corresponding cohorts in the low-treatment region. The stability in the point estimates across extended control variables bolsters the view that the interaction term ("High × Post") is not correlated with changes in these variables. Although this is not a formal test of exclusion restriction, the absence of significant correlation with observable characteristics strongly suggests the absence of significant correlation with unobservable variables (Altonji, Elder, and Taber 2005). It is worth noting that the point estimate increases after controlling for the initial community characteristics, suggesting that the bias associated with initial community characteristics, if any, goes against our finding. This is indeed consistent with the fact that post-apartheid policies focused on ensuring equality by allocating more resources to under-resourced communities.

In order to highlight factors that potentially contributed to increased educational attainment, we examine changes in enrollment rate between 1993 and 2004. Table A1 shows that the mean enrollment rate in 1993 was already high; 86 % of individuals aged between 7 and 20 were enrolled in school. The difference-in-differences estimate in the second column using KIDS93 and KIDS04 shows that changes in enrollment rate was similar between the high and low treatment region. This is not surprising in places like South Africa where enrollment rate is already high. Rather, repetition is known to be the critical issue with attaining greater education among South African children (Lam, Ardington, and Leibbrandt 2011). We wished to compare changes in the number of grades repeated before and after the policy reform, but such information is available only in KIDS04. Thus, we compare the mean differences between the high and low

treatment region in grade repetition in 2004. The estimate suggests that children in the high-treatment region had a significantly lower grade repetition. The evidence suggests, given the similarity in other educational aspects in *ex-ante* characteristics, that decreases in the number of grades repetition contributed to increased educational attainment.

In what ways did parental investments in children's education increase? To answer this question, we explore changes in parental expenditures on education between 1993 and 2004. Table A2 suggests that expenditures on school fees increased significantly more in the high treatment region. The increase in expenditure was economically substantial; the amount is equivalent to the mean expenditures in 1993. Further, expenditures on stationary and books and uniform all increased significantly. Increases in food expenditure are substantial in the point estimate but not statistically significant. Interestingly, changes in transportation expenditure were subtle and negative, potentially reflecting the finding (shown later) that physical access to school did not change. Its important implication is that educational attainment improved not due to variation in availability of educational facilities, but rather due to, parental investments to opportunities for schooling and equipment to learn.

Overall, the finding is consistent with the predictions of the quantity-quality model. Parents in areas with greater access to free healthcare increased their human capital investments in children after realizing the increased returns to learning obtained through improved health.

B. Effect on Child Quantity

We now turn to the effect of the health policy reform on child quantity. Panel B of Table 4 reports the coefficients of interest, based on the specification in equation (1), using the number of children aged 8 or less as the dependent variable for mothers aged 31 to 45. This variable captures the overall fertility in the post-reform period between 1994 and 2004. All specifications include community and cohort fixed effects.

Column (1) provides the estimate based on a basic specification. It shows that the introduction of free health services had a statistically significant negative impact on the number of children, indicating that mothers in the high treatment regions had approximately 0.334 fewer births than mothers in the low treatment regions over the period. The estimate is robust in terms of controlling for variation across individual and household characteristics in Column (2) and initial community characteristics in Column (3). The preferred estimate in Column (3) suggests that there was about 43 percent reduction in fertility. The stable point estimates across specifications are again assuring the validity of our identification assumption that the treatment status is not associated with these key determinants of child quantity.

It is also worth noting that our estimates of the policy impact on both quality and quantity are likely to be understated if any, because our DD estimates measure changes in the outcomes in the high-treatment region relative to the low-treatment region, where children still had some exposure to the policy. Thus, the estimates would be greater if we could find a pure control group among the same cohorts without any exposure to the health policy.

C. Robustness Checks on Child Quality Effect

Studies intended to evaluate social policies often suffer from bias due to two identification issues: 1) that of inherent heterogeneities between the treated and controlled groups, leading the controlled group to provide a false counterfactual for what would have happened to the treatment group without an intervention; and/or 2) that of erroneously picking up other effects through concurrent changes in society. In this subsection, we conduct falsification tests and robustness checks with regard to the effects on child quality in an effort to explore these possibilities.

Columns (1) and (2) of Table 5 Panel A report the results from the falsification tests on child quality investments, focusing on the sample of children aged between 17 and 24 from KIDS93 and KIDS04. These cohorts from KIDS04 were more than 6 years old in 1994, rendering them ineligible for free health services. It turns out that none of the estimates is statistically different from zero. This finding has several important implications.

First, it suggests that parental behaviors with respect to investing in children's education would have been similar in the two regions without the health policy. Because even the observations from KIDS04 were not granted free access to health services due to the age eligibility rule, these estimates capture the evolution of human capital development among cohorts not affected by the health policy reform. This helps preclude bias arising from preexisting heterogeneities in education had there not been health improvements.

Second, it is important to keep in mind that the majority of these cohorts from KIDS04 were in school in 1994 and thus were exposed to various other changes, including educational reforms as described in Section II, in the post-reform period. Even though the health policy had an impact on fertility, it is still possible that the changes in educational outcomes were driven by these other concurrent changes. However, our findings do not support such an assertion. The finding rather addresses a bias arising from post-treatment heterogeneities in trends for cohorts affected by concurrent changes.

Lastly, the finding rules out the externality effect of parents having used some of the savings from reduced medical costs from their younger siblings to pay for older siblings' school fees. The absence of such evidence suggests that the income effect was negligible in our contexts,

but the price effect through improved child health status and increased returns to education had the first order effect.

As a further robustness check of accounting for pre- and post-treatment bias, if any, in the falsification tests in estimating the main child quality effect, we employ a difference-in-differences-in-differences (DDD) strategy, which essentially estimate the treatment effect of the policy reform by directly removing community-specific trends using counterfactual cohorts.[16] The young cohort consists of the sample used in the main analysis, and the old cohort, which provides counterfactual trends, is the sample used in the falsification tests. Column (3) uses the interaction between the high-treatment dummy and post dummy, whereas Column (4) uses the interaction between community dummies and the post dummy to capture more precise variation across communities. The parameter of interest is the coefficient of the triple interaction between the high-treatment dummy, post dummy, and young cohort, which is interpreted as the changes in the outcome variable among the young cohort after adjusting community-specific trends using the old cohort.

The point estimates remain in the range of the main results and statistically significant at 5 percent level in both specifications. Further, the negative and statistically insignificant coefficient of the interaction term between the high-treatment dummy and post dummy is consistent with the previous finding that there is no trend difference among cohorts without exposure to the health policy reform.

Overall, the findings on child quality in the main analysis are robust and remain significant even after adjusting for heterogeneous trends across communities. Our evidence finds no indication that differential pre-existing or post-treatment trends confound such results, but rather evidence shows that the control group used in the main analysis serves as a valid comparison group, providing strong support to our identification strategy.

D. Robustness Checks on Child Quantity Effect

In this subsection, we turn to addressing pre-treatment bias and post-treatment bias with respect to child quantity effect. Columns (1) and (2) of Table 5 Panel B investigate whether preexisting trends in the outcome variables across communities confound the estimated effects on child quantity. The dependent variable is the number of children aged 11 to 19 for the sample of women aged 42 to 56. Because these children were all born before 1994 (even the observations

[16] Note that as it is previously explained, the counterfactual cohorts considered here are children aged 17 to 24 from KIDS93 and KIDS04. These cohorts present counterfactual evidence in terms of pre-treatment bias in education for cohorts not affected by the health policy as well as post-treatment bias in education for cohorts affected by concurrent changes in social policies.

from KIDS04), any differences in the estimates capture differential patterns in childbearing in the pre-reform period between the two regions. Note that these women had children aged 0 to 8 when they were 31 to 45 years old, thereby allowing an appropriate comparison with women used in the main analysis. The results provide no evidence of statistical difference in fertility transition for these women. Rather, the point estimates are consistently positive, indicating that fertility was on a relatively *increasing* trend in the high-treatment region before the policy reform, and thus the bias, if any, goes against finding a reduction in fertility.

Columns (3) and (4) of Table 5 Panel B present robustness evidence after directly controlling for pre-existing trends in the main analysis based on the DDD strategy. We use children aged 0 to 8 for women aged 31 to 45 as the young cohort and children aged 11 to 19 for women aged 42 to 56 as the old cohort. The finding suggests that the point estimates are similar to the main results and remain statistically significant at 10 percent level. Consistent with the finding in the falsification test, the coefficient of the interaction between the high treatment dummy and post dummy suggests that there is no differential trend among the old cohort.

The mechanism that we focus on to explain the observed reduction in fertility is that improved health status among children entitled to free health services induced parents to lower subsequent fertility. While the DDD estimates presented above have substantial advantage in explicitly adjusting for pre-existing trend heterogeneity across communities, an omitted, and thus confounding, variation still remains at community-year-cohort level. For example, post-apartheid investments or social programs targeting only the high-treatment region may explain the observed fertility reductions. While we directly test and reject various alternative hypotheses related to such concerns in the next section, here we address the post-reform bias by refining treatment variation at the household level. In particular, we interact the difference-in-difference term (the interaction between the high treatment and post dummy) with the number children aged 9-15 or the number of children aged 16 or above. Essentially, we are estimating the difference-in-differences-in-difference effect by exploiting variation in the number of children who were benefited from the free health services, i.e., children aged 9-15 in KIDS04 were all benefited from health services, whereas those aged 16 or above even in KIDS04 were not.

Table 6 shows that the point estimate for the triple interaction term with the number of children aged 9-15 is large in magnitude and statistically significant at one percent level, suggesting that fertility reduction is larger for households with more children eligible to free health services (and equivalently more children who improved health status). Importantly, the point estimate for the triple interaction term with the number of children aged 16 or above is not only substantially lower and statistically indistinguishable from zero in itself, but it is also

statistically different from that for the triple interaction term with the number of children aged 9-15. The finding suggests that a sheer number of children in the household does not matter, but what contributed to fertility reduction is the number of children who improved health status, giving credence to the idea that improved health status among children is the primary contributor to the post-reform fertility reduction. We conduct a further robustness check by restricting our sample to women at the same age group who have three or less pre-policy fertility in Column (2) that essentially removes women with high pre-determined fertility. The finding is similar quantitatively and qualitatively.

Overall, the findings from the falsification tests and robustness checks leave little doubt that preexisting trends do not confound the main findings, and that unobserved changes in society do not play a significant role. Rather, it is clear that the significant effects we find in child quantity are indeed due to health improvements driven by increased access to free health.

VI. Alternative Hypotheses

Distinguishing causal pathways is an important part of our study, as causation substantially determines policy implications. In this study, we emphasize the causal linkage that predicts changes in the price of child quality investment, generated by health improvements, will induce parents to increase educational investments and lower fertility. In this section, we explore various alternative pathways that may account for the observed relationship between increased access to healthcare, reduced fertility, and increased educational attainment.

A. Do reductions in mortality account for changes in fertility?

One important mechanism linking reductions in fertility and increases in educational attainment is the effect of health policy on mortality reductions. Reductions in infant and child mortality lead to lower fertility when children are viewed simply as parental investment for their future consumption (Kalemli-Ozcan 2002, 2003; Tamura 2006).[17] The critical requirement to support this model is, by construction, variation in mortality reductions across the regions.

Contrary to the assumption that increased access to healthcare should lower infant/child mortality, however, evidence highlights that mortality reductions were similar between the high- and low-treatment regions. As mentioned earlier, our understanding of child mortality hinges on

[17] Note that existing theories offer ambiguous predictions about the direction of changes in fertility in response to mortality reduction. The model of Barro and Becker (1989) in fact predicts an increase in fertility when mortality reduction is portrayed as a lowered cost of having surviving children. Clearly, our finding of reduction in fertility is not supportive to this brand of theories. While it is theoretically plausible that mortality affects, whether it increases or decreases, fertility, Galor (2012) argues that such idea is not supported by historical evidence.

the observations in the previous work by Tanaka (2014) that free health services resulted in sharp improvements in nutritional status without referring to a specific group of children at low nutritional status. This suggests that the pattern of mortality reduction was not associated with the region type. To the best of our ability, without direct information about child mortality, we now explore this mechanism in a quantitative way.

KIDS04 includes a question as to how many children the woman has given birth to who were born alive but later died. The question was asked of all women between 15 and 49 years of age. The two limitations pertaining to this variable are the absence of an equivalent question in KIDS93 that would enable comparisons and the fact that the question does not ask exactly when child deaths occurred (meaning that the answers may include child deaths that took place before 1994 and thus had nothing to do with the health policy reform). However, we still think that the variable can provide meaningful observations for two reasons. First, we have already shown that many characteristics were similar between the two regions in KIDS93. Thus, the mortality rates can also be expected to be similar in the *ex-ante* observations, allowing us to examine differences in means rather than the difference-in-differences estimates. This addresses the first limitation. Second, we can examine differences in mortality across different ages of mothers. Since younger mothers are more likely to have given birth in recent years, namely after 1994, we would find greater mortality reductions in high treatment regions among younger mothers if the high treatment regions have had greater reductions in mortality. This addresses the second limitation.

We report the results in Table A3. The dependent variable measures mortality by dividing the number of child deaths by the number of all children born to the mother. The sample is women aged between 15 and 49 from KIDS04. Column (1) reveals that the child mortality in the low-treatment region is about 10.3 percent, while the high-treatment region has a 2.2 percentage point higher mortality rate. Importantly, the difference is not statistically significant, suggesting that there is no evidence of mean differences in child deaths between the two regions.

Child mortality increases with mother's age, as the children's age increases, giving a longer period in which to observe a death. However, the pattern of mother's age effect does not exhibit any differential trend between the two regions. If child mortality had in fact fallen more in the high-treatment region, the sign of the coefficient would be positive and significant. Our results suggest if anything that mortality declined more in the low-treatment region among younger mothers.

We conduct several robustness checks. In Column (2), we add non-linear impacts of mother's age by interacting its squared value with the high-treatment region dummy, yet the results are the same. In Column (3), we include community fixed effects in place of the high-

treatment region dummy, but we still obtain similar outcomes. Overall, we find no evidence that differences in mortality reduction could be the primary explanatory mechanism driving the main results.

B. Are family planning and contraception confounded?

Increased access to healthcare, particularly improved reproductive care, may also have led to expanded access to contraception among mothers who wanted to mitigate unwanted or high-risk pregnancies. Reduction in cost of contraception, which works as increasing the cost of quantity, will tend to reduce fertility.

The use of contraception itself, however, does not invalidate the mechanism we investigate in the main analysis. Suppose, for example, mothers may have chosen to adopt contraception as a strategy to achieve/maintain the optimum number of children in response to improved child health. Such a choice is still consistent with the argument that the price change in quality investment was causal to reductions in fertility. In other words, whether fertility reductions occurred through changes in family planning or the use of contraception does not matter as long as the underlying motivation is driven by parental responses to future quality investment.

That said, the conventional wisdom regarding fertility behavior is that fertility reflects desired births, and demand for children is determined by other factors than family planning (Becker 1991; Pritchett 1994). Indeed, costs of contraceptive use are substantially smaller than costs of childrearing, and thus the view that changes in the cost of contraception have substantial influence on fertility requires an assumption that people in high-fertility countries have more children than they actually desire or that people are myopic and make "wrong" long-term decisions.

On the other hand, increased contraceptive use may point to an alternative view when it is causal to reductions in fertility regardless of considerations of quality investment, and subsequent increases in educational attainment are demonstrated by the standard quantity-quality tradeoff model, where changes in quantity are a driving force for improved quality. To investigate whether access to family planning may be confounded (note that our treatment status in the main analysis does not take into account of access to family planning), we present the changes in access to family planning clinics and family planning workers before and after 1994 in Table A4. The first row indicates that the mean number of family planning clinics in community was 0.42 in 1993 (and note that because every community had either zero or at most one family planning clinic, it also indicates that 42 percent of communities had a family planning clinic). The difference-in-

differences estimate between 1993 and 1998 suggests that the high treatment region built a fewer number of clinic. This is consistent with the government goal to eradicate inequality in healthcare access across regions. Also, the finding is consistent with the previous finding that more new health clinics were built in communities in the low-treatment region. We also use an alternative dependent variable, a dummy for having any family planning workers in the community, but the finding is essentially the same.[18]

Further, we explore changes in contraceptive uses or practices in two ways. First, we focus on breastfeeding practice, one of the most common contraceptive methods to avoid post-natal pregnancy, particularly in developing countries, where access to artificial birth control methods (i.e., condoms or sterilization) is limited. We compare the changes separately between 1993 and 1998, and between 1993 and 2004. Both coefficients in Table A4 are small compared to the mean in 1993 and statistically insignificant. Second, we examine changes in delivery intervals, the number of years since the last birth, for mothers with multiple children under 15 years old (those born after the policy reform). A contraceptive use, such as breastfeeding, often intends to maintain or prolong birth intervals. We find that there is a little and statistically insignificant change from the mean interval of 3.4 years in 1993.

All together, we find no evidence that increased access to family planning and contraception are confounded. If anything, access increased more in the low-treatment region, making the bias go against our main finding.

C. Were there any policies that directly affected price of child quantity?

Changes in the price of child quantity have been widely shown to underlie demographic changes in various other settings. For example, increases in relative female wages are shown to lower fertility through an increase in the marginal costs of additional children. In this study, we emphasize the changes in child quantity only through changes in price of quality investments, and in this subsection, we explore and reject alternative policies that may have direct impact on the price of child quantity.

First, we consider a case where the new health policy itself affected the price of child quantity. Because the free health services included pre-natal and post-natal care, and possibly the costs of newborn delivery, as well as safer delivery with more skilled attendance at birth, there is no doubt that the costs of pregnancy were affected. However, this should have caused a decline in price, not an increase, and consequently fertility should have increased. Our results, however,

[18] Note that information on the number of family planning workers is available only in KIDS98 but not in KIDS93, and thus it was not feasible to use the variable.

show the opposite response, a reduction in child quantity. Therefore, we can discard the effect of health policy on fertility that operated via changes in the price of quantity.

There were two welfare programs operant during this period--the old-age pension program in the early 1990s and the South African Child Support Grant starting in 1998--that may have changed the costs of child-bearing during the period of study and are also likely to have led to increased fertility. Even if these resource transfers were to be used for child education, methodologically speaking, in order for these programs to bias our estimates, their treatments would have had to be correlated with the availability of health facilities, which was not the case (Aguero, Carter, and Woolard 2010; and Duflo 2003). To the best of our knowledge, there is no other policy that meets such a criterion and that potentially affects the price of quantity.

D. Is an educational reform confounded?

In identifying the impacts of social policies, estimations of the treatment effect are often biased by a correlation between the treatment status and an error term. In this study, on the other hand, we have provided both quantitative and qualitative evidence that the treatment status is orthogonal to household and community characteristics in the pre-reform period, one of the salient features we can exploit to circumvent the endogeneity issue. This, however, still does not rule out omitted variable bias in the post-reform period. For example, the high-treatment region may have received more investment in education in the post-reform period, if these places were seen as more politically important or based on some other unknown factor that led to the establishment of health facilities in the first place.

The literature does not support substantial improvements in educational system even after 1994 (Lam, Ardington, and Leibbrandt 2011). Further, we have already provided three pieces of evidence that such post-reform bias is unlikely. First, the quantitative estimates from the subsection VC show that educational attainment was similar among children who were in school at the time of the policy reform yet not affected by the health policy. If any sort of educational reform exclusive to the high-treatment region had a major impact on educational attainment, educational outcomes should have increased for these cohorts. Second, we explicitly control for differential trends based on pre-reform community characteristics, addressing differential patterns of development with regard to whether primary and secondary schools existed in 1993. Lastly, a goal of post-apartheid policies was to disproportionately target hitherto deprived and underserved areas, and thus if anything, the high-treatment regions were less likely to receive further investments.

We provide an additional quantitative test of the last assertion in two ways. First, we compare changes in school infrastructure between KIDS93 and KIDS98.[19] Table A5 shows the results for three different variables (i.e., number of schools, distance to schools, and number of classrooms), for two levels of schooling (i.e., primary and secondary). All six estimates are statistically insignificant, while four of them are negative, indicating that physical infrastructure increased less in the high-treatment region. Again, this is consistent with other finding in health infrastructure that the high-treatment region had disadvantage in receiving post-reform investments.

Second, we explore quality improvements in service delivery. Ideally, we wished to observe changes in quality of school infrastructure or other types of facilities and services between 1993 and 2004. However, the community questionnaires for KIDS93 and KIDS04 contain no relevant questions that would afford direct comparison. Given that community characteristics were broadly similar between the two regions in 1993, we compare the differences in average perceptions/evaluations of service delivery in 2004. Perceptions are valued from 1 to 5, with 1 being "very unhappy" to 5 being "very happy." The results in Table A6 show that almost all community services, including primary school, health facilities, various types of physical infrastructure, water/electricity supply, and social welfare, were evaluated more negatively in the high treatment region. At most, there is no difference in the perceived quality of public services (which should reflect both quantity and quality of services) between the high- and low-treatment regions. If anything, people in the high-treatment region were less satisfied, which is consistent with the assertion that the high-treatment region was disadvantaged by post-reform investment.

E. Does migration cause selection bias?

Migration was strictly regulated under apartheid, which provides one of the salient features in our research setting that highlights similar observed characteristics across communities in 1993. This does not, however, preclude a possibility that people migrated during the post-apartheid period. This might bias the main finding if households who sought for better access to healthcare services also had preferences toward low fertility and high child education.

We address bias arising from such self-selection via migration by focusing only on the sample who resided in the same community in 1998 as they did in 1993. This essentially eliminates those who migrated immediately after the fall of apartheid.[20] In particular, for the analysis

[19] Note that such information is not available in KIDS04. By using KIDS93 and KIDS98, our intention is rather to highlight immediate changes in educational infrastructure after 1994.

[20] Note that there are two cases that migration can be observed. One is the case that individuals are observed in a different community in KIDS98 from KIDS93. Another is the case that individuals are observed only in KIDS04, whose residential communities in 1993 are not observed. Both of these cases are eliminated from this exercise, leaving only

of child quality we restrict the sample to children whose parents live in the same community in 1998 as they did in 1993, and for the analysis of child quantity we restrict the sample to women who themselves lived in the same community in 1998 as they did in 1993. Column (1) of Table A7 presents the DD estimates for educational attainment in Panel A and for fertility in Panel B, which are analogous to Table 4. Column 2 presents the DDD estimates for educational attainment in Panel A and for fertility in Panel B, which are analogous to Table 5 and 6, respectively.

It shows that more than 96 percent of parents for children stayed in the same community, and more than 80 percent of women at respective ages stayed in the same community.[21] The DDD estimates are quantitatively similar as in the main analysis. The point estimate is slightly lower for child quality yet remains statistically significant at 5 percent level, whereas the point estimate for child quantity becomes larger in magnitude and statistically significant at lower level. Such evidence provides support that migration does not bias the main finding.

F. Do parents respond to contemporaneous or future incentives?

Most literature that examines the effect on fertility and/or educational attainment focuses on a mechanism in which contemporaneous adjustments to current shocks explain quantity-quality tradeoff. In this study, the underlying motive is the price changes in child quality/quantity in the future that induce parents to make contemporaneous adjustments. It remains an open question as to whether parents in developing countries adjust behavior to future economic incentives.

While it is often infeasible and meaningless to disentangle the degree of contributions to school performance made by such improved health status while in school, as health status during early childhood is often correlated with later health status, or as health and educational investments and fertility reduction are often inter-related and jointed determined, the findings in Table A8 rather show that health status appeared to be similar among school-aged children in 2004. In particular, children in the low-treatment region were no less likely to be sick in the past 2 weeks or no less likely to be missing school due to illness. Such evidence suggests that children in low-treatment region have caught up in health status by the primary school ages, as they gain better access to health services (note that health infrastructure also expanded more in the low-treatment region, and thus these children are likely to gain better access after 1998). Such non-decreasing marginal policy effect on pre-primary children is likely and consistent with the

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the sample that stayed in the same community in 1998 as they lived in 1993. We present the estimates for the two migration cases separately in the Online Appendix II. We find consistent estimates across various ways to eliminating migrants.

[21] Note that sample size lowers more for women than for parents because we keep children whose parents are not identified. As Table 2 indicates, about 20 percent of children cannot be identified with mothers (and even more for fathers). They are preserved in our sample, while all women who moved are removed from our sample.

previous finding that the health policy similarly improved health status among children who were already born at the time of the policy reform (Tanaka 2014). The similarity in health status at the age of primary school discards differential parental investments as a response to health status for children in school.

In addition, we explore the timing of fertility decline among children born in the post-reform period. The findings in Table 7 has already shown that fertility among children aged 8 or less declined the most for those who have eligible siblings, i.e., those aged 9 to 15. If health status among these siblings during schooling ages was the primary determinant of fertility decline among younger siblings, we would not expect to observe fertility decline among cohorts close to those aged 9 to 15, namely those aged 6 to 8, as they were born when these primary school children as of 2004 were still in the pre-primary school ages. Table A9 instead highlights that the fertility decline was the largest among those aged 6 to 8, suggesting a decline in fertility immediately after health improvements were realized among eligible children.[22]

Both pieces of evidence suggest that the observed differences in parental investments in education must be derived from other differences that have been already incurred, namely improved health status during early childhood.

VII. Conclusion

In this paper, we examine the effect of abolishing user fees from health services on fertility and educational outcomes as a test of child quantity-quality tradeoff when the price of quality investment is reduced. We take advantage of the unique history of South Africa to study a set of communities that provide exogenous variation in health improvements for individuals whose *ex-ante* characteristics are otherwise similar.

By investigating the evolution of fertility and educational outcomes among children who were entitled to free health services, we find evidence in support of the QQ model: educational investments rose as a consequence of improved health status, and fertility fell. Our findings are robust across various specifications. In addition, we apply falsification tests and explore alternative hypotheses to preclude the possibility that preexisting trends in fertility and

[22] Note that fertility also declined among cohorts aged 0 to 2 as of 2004. This does not contradict with the previous finding that health status is similar between the high- and low-treatment regions among schooling-age children in Table A8, because such fertility decline can be explained not by differences in health status among older siblings but by increased educational investments into children in school in the high-treatment region. As the model indicates, child quantity-quality tradeoff continues and is enhanced over generations, as parents jointly determine both child quantity and quality. Our study highlights health improvements during early childhood as an initial trigger of such dynamics.

educational outcomes, educational reforms or other social policies, and mortality reductions could have confounded our results.

These results pose several important policy implications for other developing countries contemplating the abolition of user fees. First, the impacts of increased access to health services are not limited to improved short-term health status described in previous work by Tanaka (2014), but also extend to the long-term effect on educational attainment. Reduced fertility and increased education are considered to be two engines of economic growth, yet their causation--either one of them affecting the other or any third factor generating both--remains an important question in the empirics of economic growth and demographic transition. Our study highlights improved health status during early childhood as a mechanism linking them.

Second, health policy appears to be an effective mechanism for curbing population growth, as framed in the model, in the sense that parents, even in developing countries, make rational fertility choices based on future expected returns to human capital investments. This finding may discourage coercive population policies such as China's One Child Policy, which can lead to adverse societal side effects. Our findings suggest an alternative policy direction based on the fact that parents in developing countries successfully alter and adjust their fertility behavior in response to changes in economic incentives.

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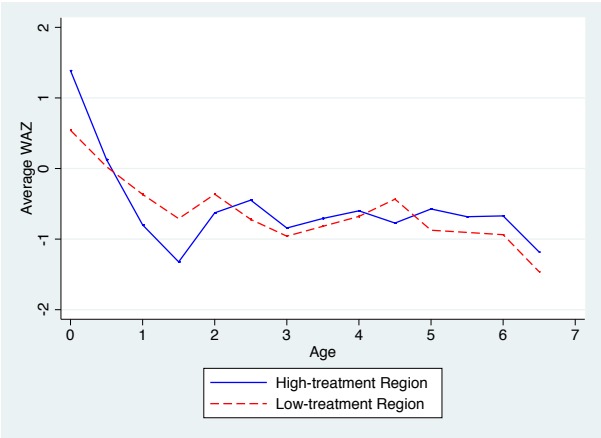
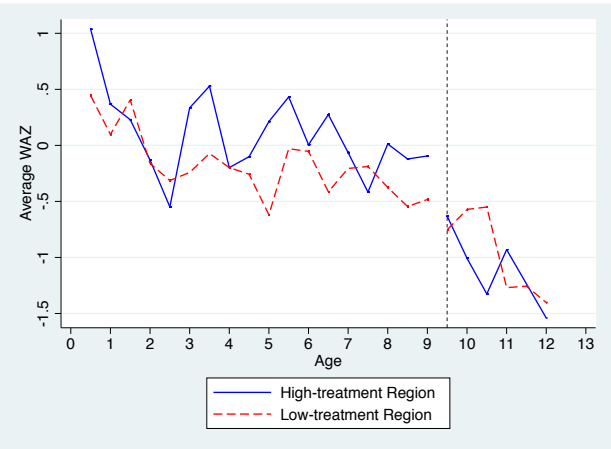
Figure 1: Average WAZ by Age and Region

Panel A. KIDS93



Panel B. KIDS98



Notes: The figure plots the mean value of weight-for-age z-scores (WAZ) by age and region. WAZ below -6 and above 5 are removed as outliers, as these numbers are seen as biologically implausible. The sample contains children aged 0 to 7 in KIDS93 in Panel A, and children aged 0 to 12 in KIDS98 in Panel B. Ages are calculated from ages in month, from which we denote age 0 month to 6 months as 0 year old, 6 months to 12 months as 0.5 year old, and so forth. The dashed line in Panel B indicates the timing of policy change; cohorts to the right are not affected by the policy, whereas cohorts to the left are partially or fully affected by the policy. The dashed line is drawn at age 9.5 years old since the policy started in June 1994, and most of the samples in KIDS98 were surveyed from March to May in 1998. Then, according to our calculation of ages, children at exactly 6 years old at the time of policy change are 9.5 years old in KIDS98.

Source: Author's calculation from the KIDS93 and KIDS98

Table 1: Infrastructure Information

	Mean	Std. dev.
Health Facilities		
Clinic	0.482	0.504
Dispensary	0.089	0.288
Hospital	0.036	0.187
Family planning clinic	0.411	0.496
Maternity home	0.107	0.312
Educational Facilities		
Primary school	1.857	1.920
Secondary school	0.821	0.606
Population	49,758	98,442
High-treatment region	0.411	0.496

Notes: This table provides information on infrastructure at the community level from KIDS93. The number of observations is 56. All variables under the category of health facilities are dummy variables, being equal to one if the respective type of facility existed in the community. The variables for primary school, secondary school, and population are in numbers. The high-treatment region indicates the share of communities that had one of clinics, dispensaries, or hospitals as of 1993.

Table 2: Summary Statistics

	KIDS93			KIDS04		
	Obs.	Mean	Std. Dev.	Obs.	Mean	Std. Dev.
Panel A: Variables for Quality Analysis						
Education attainment	1,798	3.032	2.116	1,818	3.827	2.361
Age	1,798	10.318	2.354	1,818	10.622	2.286
Female dummy	1,798	0.485	0.500	1,818	0.503	0.500
First-born dummy	1,798	0.212	0.409	1,818	0.314	0.464
Birth order: Missing	1,798	0.147	0.355	1,818	0.212	0.409
Mother's education	1,385	5.323	3.725	1,338	7.101	4.239
Mother's education: Missing	1,798	0.230	0.421	1,818	0.264	0.441
Mother's age	1,452	37.229	8.451	1,188	36.428	9.331
Mother's age: Missing	1,798	0.192	0.394	1,818	0.347	0.476
Ave. yrs. of adult members' educ.	1,798	4.235	3.343	1,818	5.888	3.342
# of adult members	1,798	2.763	1.496	1,818	2.634	1.546
Panel B: Variables for Quantity Analysis						
# of children aged 8 or less	659	0.953	1.115	740	0.526	0.765
# of children aged 11 to 19	659	1.253	1.265	740	0.966	1.057
Age	659	37.209	4.223	740	37.396	4.267
Yrs. of education	659	6.149	3.887	730	8.005	4.344
Yrs. of education: Missing	659	0.000	0.000	740	0.014	0.116
Ave. yrs. of adult members' education	659	5.054	3.253	740	6.769	3.280
# of adult members	659	3.129	1.610	740	2.965	1.716

Notes: This table reports summary statistics of variable means and standard deviations with regard to children's quality analysis (Panel A) and quality analysis (Panel B). Observations are at the individual level. The sample in the quality analysis consists of children aged 7 to 14, and the sample in the quantity analysis consists of women aged 31 to 45.

Table 3: Balancing Test of Baseline Characteristics

	Low Treatment Region			High Treatment Region			(2)-(5)
	Obs.	Mean	Std. Dev.	Obs.	Mean	Std. Dev.	Diff.
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: Demographic characteristics (all individuals in the dataset)							
Educational attainment	5,097	4.655	4.055	2,743	4.794	4.060	-0.139
Age	5,099	24.122	18.281	2,743	23.991	18.591	0.131
Female	5,099	0.522	0.500	2,743	0.537	0.499	-0.015
Panel B: Quantity investments (women in 20s to 40s)							
Missing info. on pregnancy history	1,080	0.209	0.407	579	0.200	0.401	0.009
# of pregnancies	856	2.954	2.662	463	2.806	2.591	0.149
# of births	856	2.850	2.626	463	2.698	2.515	0.153
# of births still alive	856	2.451	2.234	463	2.309	2.134	0.142
Missing info. on children's deaths	856	0.163	0.369	463	0.158	0.365	0.005
# died before 1 year	715	0.231	0.623	390	0.233	0.649	-0.003
# died between age 1 and 5	715	0.151	0.539	389	0.111	0.381	0.041
Panel C: Quality investments (children aged 7 to 15)							
Educational attainment (all)	1,283	3.387	2.351	697	3.257	2.303	0.130
Educational attainment (boys)	664	3.200	2.241	346	3.052	2.135	0.148
Educational attainment (girls)	619	3.586	2.449	351	3.459	2.443	0.128
Panel D: Household characteristics (all households)							
Household size	648	6.657	3.752	384	6.292	3.224	0.366
Dependency ratio	648	0.344	0.205	384	0.341	0.201	0.003
Total monthly income	648	1,003	1,017	384	1,011	1,070	-8.051
Panel E: Community characteristics (all communities)							
# of primary schools	33	1.515	1.395	23	2.348	2.442	-0.833
# of secondary schools	33	0.636	0.549	23	1.087	0.596	-0.451***
Population / 1000	33	26.329	70.038	23	83.373	122.831	-57.044**
Distance to the nearest market	33	20.667	20.174	23	18.304	19.382	2.362
Distance to the nearest bank	33	23.121	19.342	23	21.000	18.986	2.121

Notes: This table reports the means of observable characteristics in the low-treatment region, in the high-treatment region, and the respective number of observations and standard deviation, using information from KIDS93. Column (7) presents the difference in means between the two types of the region with statistical significance indicated by the asterisk. The sample is all individuals in Panel A, women aged 20s to 40s in Panel B, children aged 7 to 15 in Panel C, and all households and communities in Panel D and E, respectively.

***Significant at the 1 percent level.

**Significant at the 5 percent level.

Table 4: Effect of Health Access on Child Quantity and Quality

	(1)	(2)	(3)
Panel A: Child quality	Dep. var: Educational attainment		
High × Post	0.288	0.316	0.410
	(0.141)**	(0.143)**	(0.190)**
Community and cohort FE	Y	Y	Y
Individual and HH level variables	N	Y	Y
Initial community characteristics	N	N	Y
Sample	Children aged 7-14		
Observations	3,616	3,616	3,616
R-squared	0.635	0.653	0.655
Panel B: Child quantity	Dep. var: Number of children		
High × Post	-0.334	-0.307	-0.411
	(0.124)***	(0.122)**	(0.132)***
Community and cohort FE	Y	Y	Y
Individual and HH level variables	N	Y	Y
Initial community characteristics	N	N	Y
Sample	Women aged 31 to 45		
Observations	1,399	1,399	1,399
R-squared	0.157	0.167	0.174

Notes: This table reports only the coefficients of interest based on equation (1), using both KIDS93 and KIDS04. In Panel A, we report the estimates from the quality analysis. The dependent variable is educational attainment measured by the number of years of completed education for the sample of children aged 7 to 14, whose age cohorts in KIDS04 were fully exposed to the new health policy. In Panel B, we report the estimates from the quantity analysis. The dependent variable is the number of children aged 8 or less, whose age cohorts in KIDS04 represent post-reform fertility, for the sample of women aged 31 to 45. All standard errors in the parentheses are clustered at the community level.

***Significant at the 1 percent level.

**Significant at the 5 percent level.

Table 5: Falsification tests and Robustness Checks on Child Quality and Quantity Effect

	Falsification tests (DD)		Robustness checks (DDD)	
	(1)	(2)	(3)	(4)
Panel A: Child quality	Dep. var.: Educational attainment			
High × Post	-0.072	0.028	-0.173	-
	(0.304)	(0.331)	(0.311)	-
High × Post × Young	-	-	0.716	0.792
	-	-	(0.313)**	(0.310)**
Community and cohort FE	Y	Y	Y	Y
Individual and HH level variables	Y	Y	Y	Y
Initial community characteristics	N	Y	Y	Y
Community FE × Post	-	-	N	Y
Sample	Children aged 17-24 from KIDS93 and KIDS04		Young: Children aged 7-14 Old: Children aged 17-24	
Observations	2,660	2,660	6,276	6,276
R-squared	0.283	0.288	0.731	0.737
Panel B: Child quantity	Dep. var.: Number of children			
High × Post	0.114	0.187	0.058	-
	(0.219)	(0.183)	(0.184)	-
High × Post × Young	-	-	-0.375	-0.365
	-	-	(0.195)*	(0.200)*
Community and cohort FE	Y	Y	Y	Y
Individual and HH level variables	Y	Y	Y	Y
Initial community characteristics	N	Y	Y	Y
Community FE × Post	-	-	N	Y
Sample	Women aged 42-56 (Children aged 11-19)		Young: Women aged 31-45 (Children aged 0-8) Old: Women aged 42-56 (Children aged 11-19)	
Observations	870	870	2,269	2,269
R-squared	0.207	0.231	0.207	0.227

Notes: This table reports only the coefficient of interests, the double-interaction between the high-treatment and the post dummies for the falsification tests based on the DD estimates in Columns (1) and (2), and the robustness checks based on the DDD estimates additionally report the triple interactions between the high-treatment, post, and the young cohort dummies in Columns (3) and (4). The dependent variable is educational attainment measured by the number of years of completed education for the sample of children aged 17 to 24 in Panel A, and fertility measured by the number of children aged 11 to 19 for women aged 42 to 56, or children aged 0 to 8 for women aged 31 to 45 in Panel B. The young cohorts refer to the sample used in the main analysis, those who were affected by the health policy. The old cohorts refer to the sample used in the falsification tests. For example, in the case of child quality analysis, the old cohorts are children aged 17-24, and these observations even from KIDS04 were not exposed to the new health policy, as they were more than 6 years old in 1994 (note that whereas they were affected by post-apartheid educational or other social policies while in school on the other hand). In the case of child quantity analysis, the old cohorts are women aged 42 to 56 with children aged 11-19, those who had children aged 0 to 8 when they were 31 to 45, while these children were all born before the health policy reform. All standard errors in the parentheses are clustered at the community level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

Table 6: Addressing Post-treatment Bias on Child Quantity Effect

Dep. var.: Number of children	(1)	(2)
High × Post	-0.248	-0.317
	(0.134)*	(0.159)*
(C1) High × Post × N(9-15)	-0.205	-0.181
	(0.059)***	(0.073)**
(C2) High × Post × N(16-)	-0.036	-0.022
	(0.042)	(0.067)
Community and cohort FE	Y	Y
Individual and HH level variables	Y	Y
Initial community characteristics	Y	Y
Sample	Women aged 31-45	Women aged 31-45 with N≤3
F test: (C1) = (C2)	4.27**	3.37*
	[0.044]	[0.072]
Observations	1,399	1,205
R-squared	0.230	0.242

Notes: This table reports the coefficients of the double interaction term between the high-treatment region and the post dummy, and its interactions with the number of children aged 9-15, denoted as N(9-15), or the number of children aged 16 or above, denoted as N(16-). The dependent variable is post-reform fertility, as measured by the number of children aged 8 or less. The sample includes women aged 31-45 in Column (1) and its subset whose total number of children is less than or equal to 3 in Column (2). All specifications include community and cohort fixed effects, individual and household level controls, and initial community characteristics. All standard errors reported in the parentheses are clustered at the community level.

***Significant at the 1 percent level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

Appendices

Table A1: Factors Contributing to Educational Attainment

Dependent variable	Mean	Coeff.
Enrollment	0.86	-0.013
	[0.35]	(0.029)
Num. of grades repeated	0.747	-0.118***
	[1.00]	(0.038)

Notes: The first column represents the dependent variable. The second column reports the mean value of the respective dependent variable in 1993 for enrollment and in 2004 for number of grades repeated. The third column reports the coefficient of the interaction term between high-treatment region and post dummy using KIDS93 and KIDS04 (the regression also includes a post dummy and community fixed effects) for enrollment, whereas the sample is KIDS04 for the number of grades repeated and the reported coefficient is the mean difference between the high- and low-treatment region.

***Significant at the 1 percent level.

Table A2: Parental Expenditures on Education

Dependent variable	Mean in 1993	DD
School Fees	175.29	178.12*
	[758.97]	(101.80)
Stationary and books	38.91	23.55**
	[120.77]	(11.16)
Uniform	231.8	49.00**
	[191.47]	(23.38)
Food	134.88	70.61
	[239.67]	(54.13)
Transportation	492.32	-3.47
	[620.88]	(116.29)

Notes: The first column represents the dependent variable for each separate regression, the second column reports the mean value of respective dependent variable in 1993, and the third column reports the coefficient of the interaction term between the high treatment region and a post dummy for KIDS04. All regressions include a post dummy and community fixed effects. The sample includes households from KIDS93 and KIDS04. Standard deviations are reported in the square brackets, while the robust standard error clustered at the community level are reported in the parentheses.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

Table A3: Effect on Mortality

	(1)	(2)	(3)
High	0.022	0.022	
	(0.015)	(0.015)	
Age	0.004***	0.009	0.004***
	(0.001)	(0.008)	(0.001)
High × Age	-0.0005	-0.012	-0.0003
	(0.001)	(0.014)	(0.002)
Age^2		-0.0001	
		(0.0001)	
High × Age^2		0.0002	
		(0.0002)	
Constant	0.103***	0.103***	0.035***
	(0.010)	(0.010)	(0.002)
Obs.	1,103	1,103	1,103

Notes: The dependent variable is the number of child deaths divided by the number of all children born to the mother, using the sample from KIDS04. High is a dummy variable for the high-treatment region, and Age is mother's age (zero at the mean age). Column (3) includes community fixed effects in place of the high-treatment region dummy.

***Significant at the 1 percent level.

Table A4: Family Planning Access and Practices

Dependent variable	Mean in 1993	Diff.-in-diff.
Num. of family planning clinic	0.42	-0.885***
	[0.50]	(0.244)
Dummy for having family planning workers	0.39	-0.329
	[0.49]	(0.226)
Breastfeeding practice (1993/1998)	0.158	0.023
	[0.36]	(0.030)
Breastfeeding practice (1993/2004)	0.158	0.035
	[0.36]	(0.047)
Delivery interval	3.442	0.178
	[1.968]	(0.431)

Notes: The first column represents the dependent variable for each separate regression, the second column reports the mean value of respective dependent variable in 1993, and the third column reports he coefficient of the interaction term between the high treatment region and a post dummy for KIDS04. All regressions include a post dummy and community fixed effects. The sample includes communities from KIDS93 and KIDS98 for the first three dependent variables, and those from KIDS93 and KIDS04 in the last dependent variable. Standard deviations are reported in the square brackets, while the robust standard error clustered at the community level are reported in the parentheses.

***Significant at the 1 percent level.

Table A5: Changes in School Infrastructure

	School Level	
Dependent variable	Primary	Secondary
Number of schools	-0.953	-0.509
	(0.865)	(0.401)
Distance to schools	2.00	-5.87
	(10.38)	(9.40)
Number of classrooms	5.02	-5.47
	(9.16)	(10.01)

Notes: The coefficients represent difference-in-differences estimates between high- and low-treatment region before and after 1994, using KIDS93 and KIDS98. The regressions additionally control for a dummy for the post period and community fixed effects. Robust standard errors in the parentheses are clustered at the community level.

Table A6: Differences in Service Delivery

Variables	Coefficient
Primary school	-0.304
	(0.31)
Secondary school	0.098
	(0.36)
Hospitals and clinics	-1.481***
	(0.46)
Physical Infrastructure	-0.388
	(0.52)
Local market	-0.287
	(0.53)
Roads	-0.681
	(0.43)
Electricity supply	-0.689
	(0.43)
Water supply	-0.252
	(0.42)
Social security and social welfare	-0.153
	(0.36)

Notes: This table reports the differences in average perception of service delivery about various variables. Each coefficient reflects the coefficient of treatment dummy using KIDS04. The perception is valued from 1 being very unhappy to 5 being very happy. The number of observations is 59 communities.

***Significant at the 1 percent level.

Table A7: Addressing Migration

	(1)	(2)
	DD	DDD
Panel A: Child quality	Dep. var: Educational attainment	
High × Post	0.463	
	(0.177)**	
High × Post × Young		0.745
		(0.305)**
Community and cohort FE	Y	Y
Individual and HH level variables	Y	Y
Initial community characteristics	Y	Y
Community FE * Post	-	Y
Sample	Children aged 7-14	Young: Children aged 7-14 Old: Children aged 17-24
Observations	3,481	6,048
R-squared	0.654	0.736
Panel B: Child quantity	Dep. var.: Number of children	
High × Post	-0.412	
	(0.143)***	
High × Post × Young		-0.587
		(0.247)**
Community and cohort FE	Y	Y
Individual and HH level variables	Y	Y
Initial community characteristics	Y	Y
Community FE * Post	-	Y
Sample	Women aged 31 to 45	Young: Women aged 31-45 Old: Women aged 42-56
Observations	1,121	1,835
R-squared	0.172	0.249

Notes: The sample is restricted to those who resided in the same community in 1998 as they did in 1993. This essentially eliminates those who migrated immediately after the fall of apartheid. In particular, for the analysis of child quality we restrict the sample to children whose parents live in the same community in 1998 as they did in 1993, and for the analysis of child quantity we restrict the sample to women who themselves lived in the same community in 1998 as they did in 1993. Column (1) of Table A7 presents the DD estimates for educational attainment in Panel A and for fertility in Panel B, which are analogous to Table 4. Column (2) presents the DDD estimates for educational attainment in Panel A and for fertility in Panel B, which are analogous to Table 5 and 6 respectively.

***Significant at the 1 percent level.

**Significant at the 5 percent level.

Table A8: Differences in Health Status in 2004

Dependent variable	Mean in 1993	Diff.-in-diff.
Sick in the past 2 weeks	0.05	-0.004
	[0.22]	(0.016)
Not enrolled in school due to illness	0.015	0.003
	[0.12]	(0.005)

Notes: The first column represents the dependent variable for each separate regression, the second column reports the mean value of respective dependent variable in 1993, and the third column reports the coefficient of the interaction term between the high treatment region and a post dummy for KIDS04. All regressions include a post dummy and community fixed effects. The sample includes households from KIDS93 and KIDS04. Standard deviations are reported in the square brackets, while the robust standard error clustered at the community level are reported in the parentheses. Both dependent variables are dummy variables.

Table A9: Changes in Fertility by Ages

Age:	Dep. var: Number of children				
	0-2	3-5	6-8	9-11	12-14
	(1)	(2)	(3)	(4)	(5)
High × Post	-0.111	-0.135	-0.165	0.067	0.038
	(0.043)**	(0.069)*	(0.082)**	(0.081)	(0.068)
Community and cohort FE	Y	Y	Y	Y	Y
Individual and HH level variables	Y	Y	Y	Y	Y
Initial community characteristics	Y	Y	Y	Y	Y
Sample	Women aged 31 to 45				
Observations	1,399	1,399	1,399	1,399	1,399
R-squared	0.102	0.135	0.129	0.121	0.117

Notes: This table reports the coefficients of interest using various age groups to construct the dependent variable.
**Significant at the 5 percent level.
*Significant at the 10 percent level.

Not For Publication

Abolishing User Fees, Fertility Choice, and Educational Attainment

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November 2015

Online Appendices

Online Appendix I

In the main text, we presented the falsification evidence on the fertility effect by investigating the fertility reductions among children aged 11 to 19 for the sample of women aged 42 to 56. Because these children were born before the policy reform, the difference-in-differences using the relevant control group, i.e., the same age groups from KIDS03, address pre-existing heterogeneous trends across the high- and low-treatment regions. In addressing the post-reform bias arising from other social policies that may have targeted only the high-treatment region for reducing fertility, we examined fertility reduction using variation in the treatment status at the household level, namely the number of children within the household who were benefited from the free health services. In this Online Appendix, we present further robustness evidence based on the similar idea.

In particular, variation in the treatment status, specifically defined as improved child health, comes from various types of child composition at the household level. The first group of women (denoted as G1 in Table 7) has no children aged 9 or above, which constitute children born before the policy reform in KIDS04. Among women who have children aged 9 or above, or *pre-determined* fertility for women in KIDS04, G2a refers to households in which all children are aged 9 to 15, the cohorts eligible to the free health services for those observed in KIDS04); G2c refers to households with children only aged 16 or above, indicating that none of the children even in KIDS04 received free health services; and G2b refers to households having children aged both in 9-15 and in 16 or above.

Interacting these types of households with the difference-in-differences term (an interaction term between the high-treatment region and post dummy), we conduct the DDD analysis for the sample of women aged 31 to 45, using the number of children aged 8 or less, post-reform fertility for those in KIDS04, as the dependent variable, both of which are the same as in the main analysis. If the observed fertility reduction in the main analysis were driven by an unobserved post-apartheid policy that targeted only the high-treatment region, we would expect to observe fertility reductions even for households in which no child health improvements occurred.

Column (1) of Table OA1 shows that there is a substantial and the largest reduction in post-reform fertility for households in which all pre-reform children were benefited from the free health services (i.e., among G2a). The magnitude of the point estimate for G2b is lower than that for G2a, if not statistically significantly different from each other. Notably, the magnitude of the point estimate for G2c is not only substantially lower and statistically indistinguishable from zero in itself, but it is also statistically significantly different from that for G2a. We conduct a further robustness check by restricting our sample to women at the same age group who have three or

less pre-policy fertility in Column (2) that essentially removes women with high pre-determined fertility. The finding is similar quantitatively and qualitatively.

The evidence above suggests that fertility did not fall for households in which no child improved health status. The findings in these robustness checks give credence to the idea that improved health status among children is the primary contributor to the post-reform fertility reduction.

Table OA1: Addressing Post-treatment Bias on Child Quantity Effect

Dep. var.: Number of children	(1)	(2)
High × Post × G1	-0.290	-0.308
	(0.112)**	(0.127)**
High × Post × G2a	-0.395	-0.519
	(0.109)***	(0.126)***
High × Post × G2b	-0.341	-0.444
	(0.185)*	(0.181)**
High × Post × G2c	-0.011	-0.197
	(0.138)	(0.143)
Community and cohort FE	Y	Y
Individual and HH level variables	Y	Y
Initial community characteristics	Y	Y
Sample	Women aged 31-45	Women aged 31-45 with N≤3
F test: (G2a) = (G2b) = (G2c)	3.47** [0.038]	2.31 [0.109]
F test: (G2a) = (G2b)	0.13 [0.721]	0.27 [0.606]
F test: (G2a) = (G2c)	6.66** [0.013]	4.59** [0.037]
Observations	1,399	1,205
R-squared	0.187	0.218

Notes: This table reports the coefficients of the triple interaction terms, an interaction of the difference-in-difference interaction between the high-treatment region and the post dummy with various types of child composition at the household level. G1 refers to households with no children aged 9 or above, which constitute pre-reform children in KIDS04. G2a refers to households in which all children are aged 9 to 15, the cohorts eligible to the free health services for those observed in KDIS04, G2c refers to households with children only aged 16 or above, indicating that none of the children even in KIDS04 received free health services. G2b refers to households having children aged both in 9-15 and in 16 or above. The outcome is the number of children aged 8 or less, post-reform fertility for those in KIDS04. All specifications include community and cohort fixed effects, individual and household level controls, and initial community characteristics. The same is women aged 31 to 45 in column 1 and its subset who has three or less pre-reform children in column 2. Standard errors reported in the parentheses are clustered at the community level.

***Significant at the 1 percent level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

Online Appendix II

In the main text, we addressed bias arising from migration by focusing only on the sample who resided in the same community in 1998 as they did in 1993. In conducting this sample selection, we considered two cases of migrations. The first case is that individuals are observed in a difference community in KIDS98 from KIDS93. We call this migration-out. The second case in that individuals are observed only in KIDS04, and we do not know where they lived in 1993, as they are not observed in KIDS93. We call this case migration-in. In the main analysis, we eliminated both cases. In this Online Appendix, we present the estimates for individual cases. In the table below, Columns (1) and (4) use sample that eliminate migration-out, and Columns (2) and (5) use sample that eliminate migration-in. We also present the estimates that eliminated both migration in and out, the estimates presented in the main text, in Columns (3) and (6) for the comparison purpose. The estimates in Columns (1) through (3) are based on the difference-in-differences approach, and those in Columns (4) through (6) are based on the difference-in-differences-in-differences approach. Panel A uses educational attainment as the dependent variable, while Panel B uses number of children as the dependent variable. The findings are essentially unchanged across various ways to eliminating migrants.

Table OA2: Addressing Migration Out and In

	(1)	(2)	(3)	(4)	(5)	(6)
Excluding those who migrated:	Out	In	In & Out	Out	In	In & Out
	DD			DDD		
Panel A: Child quality	Dep. Var.: Educational attainment					
High × Post	0.424	0.447	0.463			
	(0.183)**	(0.184)**	(0.177)**			
High × Post × Young				0.744	0.792	0.745
				(0.307)**	(0.307)**	(0.305)**
Community and cohort FE	Y	Y	Y	Y	Y	Y
Individual and HH level variables	Y	Y	Y	Y	Y	Y
Initial community characteristics	Y	Y	Y	Y	Y	Y
Community FE × Post	-	-	-	Y	Y	Y
Sample	Children aged 7-14			Young: Children aged 7-14 Old: Children aged 17-24		
Observations	3,562	3,535	3,481	6,193	6,131	6,048
R-squared	0.657	0.652	0.654	0.738	0.735	0.736
Panel B: Child quantity	Dep. var.: Number of children					
High × Post	-0.439	-0.400	-0.412			
	(0.139)***	(0.135)***	(0.143)***			
High × Post × Young				-0.417	-0.516	-0.587
				(0.202)**	(0.242)**	(0.247)**
Community and cohort FE	Y	Y	Y	Y	Y	Y
Individual and HH level variables	Y	Y	Y	Y	Y	Y
Initial community characteristics	Y	Y	Y	Y	Y	Y
Community FE × Post	-	-	-	Y	Y	Y
Sample	Women aged 31 to 45			Young: Women aged 31-45 Old: Women aged 42-56		
Observations	1,368	1,148	1,121	2,224	1,873	1,833
R-squared	0.163	0.173	0.172	0.224	0.247	0.249

Notes: The samples for migration-out are the respective sample who stayed in the same community as in 1993. The sample for migration-in removes individuals who migrated into a new community immediately after the fall of apartheid. In particular, for child quality analysis we eliminate respective children whose parent, at least one of the two, newly appeared in KIDS98, and for child quantity analysis we eliminate respective women who themselves newly appeared in KIDS98.

***Significant at the 1 percent level.

**Significant at the 5 percent level.