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Abolishing User Fees, Fertility Choice, and Educational Attainment1

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Abolishing User Fees, Fertility Choice, and Educational Attainment[1]

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Abstract

This study examines the effect of abolishing user fees from health 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 as an exogenous decline in price of quality investments, we document evidence consistent with the model that parents substitute fertility and increase educational investments. The absence of treatment effects among children not subject to the health policy eliminates channels through heterogeneous preexisting trends or unobserved concurrent changes. Overall, our findings highlight a health policy as a motivating force underlying the demographic transition and economic growth.

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

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I. Introduction

Economists hold long-standing interests in explaining the secular demographic transition characterized by the historical shift from high to low fertility rates. The extent, or even the existence, to which fertility can be portrayed as a matter of parental choice has also been at the heart of the development policy debates. The stylized fact that the demographic transition has almost always been coupled with human capital development and economic growth[1] 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, 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 the quality of children raises the shadow price (marginal cost) of the 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 induce parents to substitute quantity with quality. The mechanism we focus on in this study differs from the existing empirical studies in three distinct aspects. First, a number of economic theories have attributed the fertility decline to historical changes in factors raising 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); and elevated women's status (Mincer 1963; Galor and Weil 1996; Lagerlöf 2003). Empirical evidence on the interaction between quantity and quality is also predominantly concentrated on research contexts involving constraints on the quantity of children, i.e., using exogenous changes in the number of children due to unexpected incidences, such as twin births or sibling's compositions (Rosenzweig and Wolpin 1980; Angrist and Evans 1998; Angrist, Lavy, and Schlosser

[1] Most notably, rapid economic growth in China and India since the 1970s has been accompanied by intense population policies to curb fertility growth. See Ebenstein (2010) and Qian (2009) for the effect of China's One-Child Policy on educational attainment.

2005; Black, Devereux, and Salvanes 2005) or economic incentives for child-bearing, such as changes in relative female wages (Schultz 1985, 1986) or financial incentives (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.[2] Our research design takes advantage of the 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 returns to education during schooling, thereby effectively lowering the price of quality investments. We explore whether this shock in turn induces parents to invest more into human capital and to 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.[3]

[2] For example, Galor and Weil (2000) develop a unified endogenous growth model where increased returns to education due to technological progress is followed by reductions in fertility and increases in the 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 price change in human capital investment 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.

[3] For example, in models considering intergenerational effects from parents to children, the utility of children becomes an important component of parental utility function. Increased investments in child education, therefore, raises the marginal cost of a child, as constrained by budget constraint, leading to the negative associations between quantity and quality of children. Also in models in which intergenerational effects run from children to parents as parents seek care from children in their old age, parents

Third, we identify a health policy as a motivating force underlying the demographic transition. The policy intervention we investigate is the abolition of user fees from healthcare services in the aftermath of apartheid dismantlement in 1994 in South Africa. Assessing the benefits of free health services is important in its own right, as there are increasing interests among development policy makers whether user fees should be charged or abolished from basic health services.[4] While a growing 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 one hand, the estimated effects are likely to be overstated by unobserved time factors (Lagarde and Palmer 2008), while on the other hand, estimates would be substantially understated if the health improvement experienced during early childhood triggers the demographic transition and economic growth. We know of no other study that exploited a health policy as a motivating factor underlying the development and growth process.

In this sense, 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 status and socioeconomics status up to middle age.[5] The critical period programming is often referred as a channel link-

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maximize their future transfer from children in relation to the cost of rearing children, again producing the quantity-quality tradeoff.

[4] 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 enhance targeting individuals in need of a good and increasing usage among them. Recent evidence, primarily derived from randomized controlled trials, however, suggests that even charging a small price substantially reduces take-ups of health goods (i.e., insecticide-treated nets, deworming drugs, or water disinfectant), due in large part to crowding out the poor in need of these goods (Kremer and Miguel 2007; Holla and 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 enhancing equity in access to health good and services and to improving health status.

[5] The literature along with the fetal origins hypothesis is summarized in Almond and Currie (2011). Further evidence on 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).

ing early childhood health to later educational outcomes, but the precise mechanism remains unknown. We highlight the fertility reduction at the root of a linkage between the two.

To establish causality, the critical identification assumption is that the source of health improvements was plausibly exogenous. We exploit the unique history of South Africa under apartheid. For more than four decades prior to 1994, black Africans had limited political representation as well as ability to choose residential locations. Extreme domination by whites over the allocation of health resources unrelated to demand of black Africans 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 (2013) shows that the removal of user fees in 1994 led to substantial improvements in nutritional status among children in communities with a health facility relative to those without by 1998. This study investigates its long-term effect in 2004 on educational outcome and fertility decision as a test of the QQ model.

We document evidence consistent with the theoretical predictions that abolishing user fees from health services induced parents to reduce fertility and to increase educational investment between 1993 and 2004. Furthermore, our finding of the increases in the educational attainment is limited to specific cohorts of children whose improvements in nutritional status have been realized, only after which the fertility fell. In addition, we conduct a number of falsification tests that show the absence of treatment effects in both the pre-reform cohorts and the post-reform children in schools not subject to the health policy, which eliminate other potential channels such as heterogeneities in preexisting trends or unobserved concurrent changes in educational reforms or income effects. Overall, our findings suggest that improved nutritional status affected child quantity and quality through its effect on the price of quality investments.

The reminder of the paper is structured as follows. Section II describes the health policy and educational background in South Africa, datasets used in the main analysis, and its summary statistics. Section III presents an econometric framework and discusses the validity of its identification assumptions. Section IV reports empirical results, and Section V concludes.

II. Background

A. Historical Context 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. The resulting society was characterized by racial segregation between black Africans, coloreds, and whites. Among these groups, black Africans were the most poor and underserved and suffered extreme domination by whites in all aspects of their lives. In particular, this included their residential choices and allocation of resources within their communities.

Among children, the overriding issue was the disparity and inequality in nutritional status and educational performance across races and geographical areas. With respect to health, black African children suffered from substantially low health status due to absence of health facilities. Even in cases where facilities existed, the high costs relative to income and low quality of services served as major impediments to the access of quality healthcare services.

The new democratic government established in 1994 took immediate efforts to ensure equal access to public services. The most notable policy in the health sector was the abolition of user fees from healthcare services to pregnant women and to children under 6 years old.[6] Various studies have found substantial improvements in access to and utilization of healthcare services among these groups of individuals (McCoy 1996; Department of Health 1998; Schneider and Gilson 1999; Wilkinson et al. 2001; Cooper et al. 2004; Morestin and Ridde 2009). More importantly for our research context, the policy led to immediate and substantial improvements in nutritional status among children by 1998 (Tanaka 2013).

In the educational sector, on the other hand, progress in eliminating racial disparities was limited, although much effort was made in equalizing allocation of government funds. Two facts are especially important for our research purpose. First, most Black Africans continued to have poor school performance due in large part to differences

[6] 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 from infants through adolescents, and micro-nutrient supplementation.

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 still have stayed 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 and quality of schools their children attend (Case and Deaton 1999).

Second, there remains a large degree of variation in student performance, 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), which Lam, Ardington, and Leibbrandt (2011) call "schooling as a lottery."[7] The weak explanatory power of school inputs and household attributes on educational performance leaves the identification of key factors as an open question.

B. Data Sources

We use longitudinal datasets from the KwaZulu-Natal Income Dynamics Study.[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 is characterized by attributes in common 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, Project for Statistics and Living Standards and Development, and thus it provides information prior to the new

[7] There are several possible explanations for such variation. On one hand, Hoadley (2007) points to the problems in a chaotic classroom environment as well as ineffective management, leading to poor governance of school functions, and on the other hand, van der Berg and Shepherd (2008) attribute to poor internal assessment of student performance. Lam, Ardington, and Leibbrandt (2011) establish a model with evidence to support it that a stochastic linkage between actual ability and measured performance led to high enrollment and high repetition rates among black African schools.
[8] For more detailed description of KIDS, see Tanaka (2013), May et. al. (2000), and May et. al. (2007).

health policy reform, and the subsequent waves in the post-reform period revisited only black Africans and Indians in KwaZulu-Natal province. Though the second wave was conducted in 1998, the long period of time between the first and the third waves enables us to trace children from early childhood to primary schooling ages. These datasets report detailed information on key variables, including years of education and the number of children as well as other important individual and household characteristics, such as age, gender, and education of all household members. In addition, 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]

We make two restrictions to the sample used in the main analysis in an effort to remove heterogeneities that may cause bias in our estimates. First, the sample is restricted to only black Africans. This is appropriate as the goal of the government has been to target disadvantaged groups, and hence black Africans have been the most affected. Second, to avoid impacts through relaxation of migration restrictions after 1994, we eliminated the households from KIDS04 who had moved their residential community since 1993.[10] One may be concerned that such sample selection may lead to a sample bias by keeping more households unconcerned about health access in the low-treatment region, leading the estimated impacts to be overstated. Evidence discussed below, however, suggests this is less likely; a number of individual and household characteristics are similar between the two regions. We also conduct a falsification test to explore the parallel trend assumption in Section IV C.

C. Summary Statistics

The baseline information of community-level infrastructure as of 1993 is presented in Table 1. The first column shows the mean values of respective variable, and the second column shows its standard deviation. All variables for health facilities (clinics, dispensaries, hospitals, family planning clinics, and maternity homes) are dummy variables, being equal

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

[10] Note that the residential location as of 1993 was exogenously determined under the apartheid regime. The Bantu Authorities Act of 1951 forced black Africans to move and to live in underdeveloped and infertile areas known as "homelands." This was solely determined and controlled by white elites, and thus the areas black Africans lived in 1993 do not reflect self-selection.

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 were available on average. 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.

As discussed later, we define the high-treatment region as communities that had at least one medical center (clinic, hospital, or dispensary), and otherwise defined as the low-treatment region. The last row indicates that 41 percent of the total 56 communities (equivalent to 23 communities) constitutes the high-treatment region. The variables for primary and secondary schools are in numbers; the table shows that there existed 1.86 primary schools and 0.82 secondary schools on average. It is clear that these communities lacked in educational facilities as well as health facilities.

Table 2 presents summary statistics on children's quantity for the sample of black African women aged 31 to 45, decomposed by the treatment status, using observations from KIDS93 in Panel A and those from KIDS04 in Panel B. We use children aged less than 10 to measure the fertility in the post-reform period, and thus we report the corresponding figures in the pre-treatment period in Panel A.

A notable observation is that while the number of children less than 10 years old was slightly higher in the high-treatment region in the pre-reform period, it became lower in the high-treatment region in the post-reform period, while the numbers decreased in both regions. Consistent with the model predictions, this provides initial evidence that improved health status, or reduced price of quality investments, following abolishing user fees, led to a fall in fertility.

In Table 3, we present summary statistics on children's quality for the sample of black African children aged 7 to 15, decomposed by the treatment status, using observations from KIDS93 in Panel A and those from KIDS04 in Panel B. Note that those aged 15 in 2004 denotes the cut-off cohort of the age eligibility to free health services; individuals sixteen years of age or older in 2004 were six years of age or older in 1994 and thus were not entitled to free health services. We use years of education, computed from completed education, to measure the parental investments to child quality. It is worth noting that the use of completed education avoids measurement error arising from grade repetition, which is prevalent among these students. While the years of education increased in both regions, the magnitude was greater in the high-treatment region, where improvements in nutritional status were greater and declines in fertility were larger. These findings are consistent with the predictions posed by quantity-quality tradeoff models and precede the regression-adjusted results in the main analysis below.

III. Research Design

A. 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 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 for a long distance to receive treatments or wait until facilities were built.[11] Thus, we define the high-treatment region to form the treated 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 (DID) framework to measure the effect of free health services on outcomes of interests.

Specifically, we estimate;

$$(1) \quad Y_{ickt} = \alpha_0 + \alpha_1(High_c \times Post_t) + \alpha_2 Post_t + X_{it}'\boldsymbol{\alpha}_3 + (W_c' \times Post_t)\boldsymbol{\alpha}_4 + \gamma_k + \mu_c + \epsilon_{ickt}$$

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,

[11] 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.

X_{it} , controls for key individual and household characteristics.[12] The community characteristics (W_c) control for the number of primary and secondary schools, and distance to market in 1993, and are interacted with the post dummy, addressing a concern that there may be a differential trend in the outcome variable that is correlated with baseline community characteristics in 1993. Additionally, we include the community fixed effects, which help purge any time-invariant community characteristics, and birth-year cohort fixed effects to account for year-specific shocks common across all individuals. All standard errors are clustered at the community level, allowing possible correlations over time within communities.

The outcome of interests for the quantity analysis is the number of children aged 10 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 form pre-reform fertility rates. Ideally, precise history of childbirths or pregnancies over the 10-year period would be useful to exactly measure fertility. Unfortunately, however, the pregnancy history questions in both KIDS93 and KIDS04 do not report the year of pregnancy and suffer from low response rates (about 23% of women did not answer the questions). Thus, instead of using the pregnancy history, we infer the fertility using the number of children aged 10 or less from the family roster.

One may be concerned that the fertility variable may be biased by child deaths under 10 years old. However, Tanaka (2013) presents evidence that the relative increase in nutritional improvements in the high-treatment region due to increased access to health services do not concentrate on the lower tail of the distribution; instead the reductions in share of children with extremely low nutritional status were similar between the two

[12] For the quantity analysis, the individual level of variables controlled for include educational attainment and the household level of variables include the highest and average educational attainment of household adult members and the number of household adult members. The definition of "adult members" should be considered with care. 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 members aged over 30. For the quality analysis, the individual level of variables control for a female dummy, while the household level of variables include mother's education, mother's age, the highest and average educational attainment among adult members, and the number of household adult members.

regions. This is indeed not surprising because the services children obtained were consultations or relatively simple treatments like growth monitoring and curative services when sick. These were still likely to have affected nutritional status among children but less likely to result in substantial reductions in mortality.[13] We present further evidence in Appendix I that mortality reductions were similar using available data.

The outcome of interests for the quality analysis is educational attainment by a child *i* aged between 7 and 15. In calculating educational attainment, we focus on the number of years computed from the completed grades given age, rather than the 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 parameter of interests, α_1, essentially measures the contribution of free health services to the long-term effects in child quantity and quality outcomes across communities with distinct intensity of access to health facilities. The estimated effects would be biased if there were unobserved heterogeneities across these communities that were correlated with the evolutional path of outcomes. We discuss the validity of such identification assumptions in the following subsection.

B. 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 opportunity that makes the assumption 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). Table 4 provides evidence 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.

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[13] Also note that Tanaka (2013) shows somewhat decreases in nutritional status among older children who were not entitled to free healthcare due to declining quality of services and the low morale of health providers. This is also suggestive that little technological advancement that should have affected all patients regardless of age took place.

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 through 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 education attainment among children aged 7 to 15 (years of educational attainment by gender in Panel C), household characteristics among all households in the dataset (household size, dependency ration, and 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 school in Panel E).[14]

Taking 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 minorities unrelated to local demand from 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 quantity and quality, improvements in health status in the post-reform period must have been substantially larger in the high-treatment region relative to low-treatment region and have taken place exogenously to various channels other than free health services. We draw sufficient supporting evidence from Tanaka (2013). Using the similar research setting, he finds that the health policy reform resulted in sharp and statistically significant increases in nutritional status, as measured by

[14] The finding is consistent with historical facts. Under apartheid, South Africa was characterized by extreme domination by the white minority over the black majority; most of the resources were allocated for white communities, and little was distributed to black communities. For educational facilities, 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.

weight-for-age z-scores and weight-for-height z-scores, between 1993 and 1998 in the high-treatment region compared to the low-treatment region.

In addition, two pieces of compelling evidence from his falsification tests are illustrated in Figure 1 (reproduced from Tanaka 2013). He first shows that health status was virtually identical across ages 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 health services they received did not lead to improved health status (i.e., due to low quality of services). In either case, this finding rejects a possible existence of differential pre-trends. Further, children not entitled to free health services (i.e., children who were 6 years old and above as of 1994) did not receive a similar treatment effect, although they have been exposed to any other changes in society and government policies.[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 these pieces of evidence are the key ingredients for our analysis; we interpret the variation in health status induced by abolition of user fees between the high- and low-treatment regions as an exogenous source of changes in prices of child quality investments, which otherwise is difficult to exploit.

IV. Estimation Results

A. Effect on Child Quality

We start by investigating the effect of the policy change on educational attainment. Panel A of Table 5 reports the estimated impacts on the years of completed education. All specifications include community fixed effects, while column 1 is based on a basic framework with only controlling for a post-period dummy, while column 2 controls for cohort fixed effects, and column 3 additionally controls for the individual level variables (birth year dummies and educational attainment) and household level variables (the highest

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[15] The point estimates are indeed negative for these cohorts, indicating that there was 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.

and average educational attainment of household adult members, and the number of household adult members).

The preferred estimate in column 3 suggests that children in the high-treatment region had completed 0.416 more years of schooling compared to the corresponding cohorts in the low-treatment region. The stability in point estimates across extended control variables provide support 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 is likely to suggest the absence of significant correlation with unobservable variables (Altonji, Elder, and Taber 2005). The comparison between boys (column 4) and girls (column 5) shows that the effect was similar in magnitude.

The finding is consistent with the predictions of the quantity-quality model. Parents in areas with greater access to free healthcare increased the human capital investments to children after realizing their increased returns to school through health improvements.

B. Effect on Child Quantity

We now turn to the effect of the health policy reform on child quantity. Panel B of Table 5 reports the coefficients of interest, based on the specification in equation (1). Columns 1 to 3 report the results using the number of children aged 10 or less as a dependent variable. This variable captures the overall fertility in the post-reform period between 1994 and 2004. All specifications include community fixed effects to control for time-invariant heterogeneities across communities.

Column 1 provides the estimate based on a basic specification with additional controls of a post period dummy. It shows that the introduction of free health services has a statistically significant negative impact on the number of children, indicating that mothers in the high treatment regions had approximately 0.382 fewer births than mothers in the low treatment regions over the period. The estimate is robust to controlling for variation across birth-year cohorts (Column 2) and additional individual, household and community characteristics (Column 3). The finding is robust to the inclusion of cohort fixed effects as well as other individual, household and community attributes, reassuring again the validity of our identification assumption that the treatment status is not associated with other key determinants of child quantity.

It is also worth noting that our estimates of the policy impact on fertility are likely to be understated, because our DID estimates measure the fertility decline 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 same cohorts without any exposure to the health policy.

To further identify the mechanism, we disaggregate children into two groups: those aged less than 5 and those aged 6 to 10. If (realized) improved health status was a key determinant of fertility choice, the magnitude of effect is expected to be larger among younger cohorts, as parents first need to observe health improvements among older children before altering their own fertility behaviors.[16] As predicted, the magnitude of the reduction in fertility turns out to be greater among children aged less than 5 (column 4) relative to those aged 6 to 10 (column 5). The estimate indicates the number of children aged less than 5 per woman in the high treatment region is approximately 0.204 fewer than that in the low treatment region, which accounts for about 57 percent of the total reduction due to the introduction of free health services. On the other hand, the effect on older children between 6 and 10 years old is smaller and is not statistically different from zero. These findings indicate that the linkage between health policy and fertility may not be direct; rather the health improvements achieved during the first several years among the older children are likely to have offered a channel between the two.

C. Falsification Tests

Studies intended to evaluate social policies often suffer from bias due to one of 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 to explore these possibilities. The basic idea is that we repeat the main analysis on child quantity/quality using the samples not exposed to the policy change.

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[16] Note that observing the fertility effect among older cohorts does not necessarily invalidate our identification strategy. For example, if parents update their fertility behavior based on expected improvements in child health due to greater access to health services, it is possible to have immediate shifts in fertility in 1994. On the other hand, greater impacts among younger cohorts indicate a strong signal that fertility choice was influenced by realized, not expected, improvements in nutritional status.

This should formulate valid counterfactual evidence in quantity/quality transitions, had there not been health policy reform.

Panel A of Table 6 reports the results from the falsification tests on child quality investments, focusing on the sample of children aged between 17 and 25. Again, these cohorts were more than 6 years old in 1994, making them ineligible to free health services. As it turns out, none of the estimates are statistically different from zero. This finding has several important implications:

First, it suggests that parental behaviors in investing children's education would have been similar 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 policy reform. This helps preclude bias arising from preexisting heterogeneities.

Second, it is important to keep in mind that the majority of these cohorts from KIDS04 were in school in 1994, when various other changes including educational reforms as described in Section 2 were implemented. 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 finding provides no support to such a mechanism. Furthermore, the finding rules out an externality effect such that parents could have 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.

Panel B of Table 6 investigates whether preexisting trends in the outcome variables across communities confound the estimated effects on child quantity. It presents the results on child quantity using children among those aged greater than 11 between the high and low treatment region for women aged 31 to 45. Note that because these children were all born before 1994 (even the observations from KIDS04), any differences in estimates capture differential patterns in childbearing among women in 20's or early 30's in the pre-reform period between the two regions. The result in column 1 provides no evidence of statistical difference in fertility transition for these women. In addition, disaggregating children between younger cohorts (ages 11 to 15 in column 2) and older cohorts (ages 16 to 20 in column 3) reveals that bias from preexisting trends, if any, goes against our finding; the point estimate for children aged 11 to 15 is positive and greater than that for children aged 16 to 20, indicating that the high-treatment region was on an *increasing* trend of fertility before the policy change. The finding leaves little scope for preexisting trends to differ.

As is clear, we cannot formulate non-affected fertility in the post-reform period, as any newborns after 1994 were essentially affected by the health policy, leaving a concern of unaccounted changes unaddressed. Instead, we resort to qualitative evidence that there is no specific policy that may have changed costs of child bearing. Note that the South African Child Support Grant (CSG), which was first rolled out in 1998, is likely to have reduced costs of child bearing. Methodologically speaking, however, in order for CSG to bias our estimates, the treatment by CSG has to be correlated with the existence of health facilities, which was not the case (Aguero, Carter, and Woolard 2010). To be the best of our knowledge, there is no other policy that meets such a criterion.

Overall, the findings from the falsification tests leave little room for preexisting trends to confound the main findings, or for unobserved changes in society to play a role. Rather, the significant effects we find in child quantity/quality are due to health improvements driven by increased access to free health services.

V. 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 price of quality investment is reduced. We take advantage of unique history in South Africa that provides exogenous variation in health improvements across communities for individuals whose *ex-ante* characteristics are similar.

By investigating the evolutions 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 have risen as a consequence of improved health status, while fertility fell. Our findings are robust across various specifications. In addition, evidence from falsification tests limits the scope of preexisting trends or omitted variables to confound the effects.

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 (2013), but also extend to the long-term effect on educational attainment. Reduced fertility and increased education are considered as the engine of economic growth, yet what causes them-- either one of them affecting the other or any third factor generating both-- remains a big question in the empirics of economic growth and demographic transition. Our study highlights improved health status as a mechanism linking them.

Second, a health policy stands out to be effective in curbing population growth, as is framed in the model, in a sense that parents make fertility choice based on future expected returns to human capital investments. This may lead to discouraging drastic population policies like One Child Policy in China, which is known to have many adverse effects. Our findings indicate that parents in developing countries successfully update their fertility behavior in response to changes in economic incentives.

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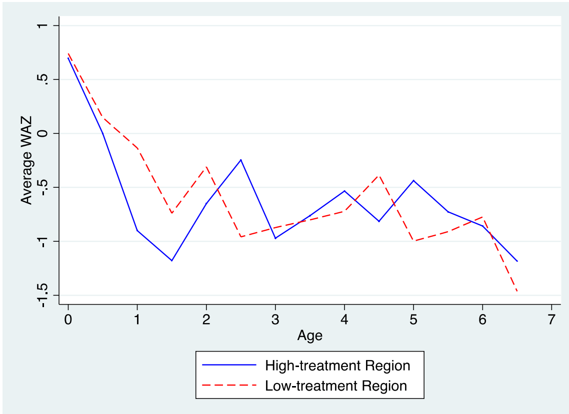
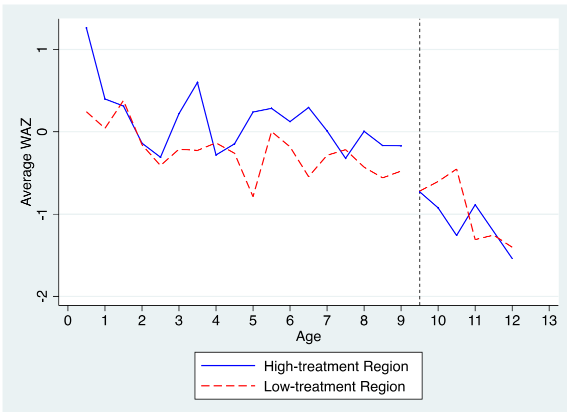
Figure 1: Average WAZ by Age and Region

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. High-treatment region indicates the share of communities that had one of clinics, dispensaries, or hospitals as of 1993.

Table 2: Summary Statistics on Children's Quantity by Treatment Status

	Low Treatment Region			High Treatment Region		
	Obs.	Mean	Std. Dev.	Obs.	Mean	Std. Dev.
Panel A: KIDS93						
# of children aged less than 10	445	1.231	1.360	208	1.365	1.263
# of children aged 11 to 20	445	1.292	1.319	208	1.385	1.382
Age	445	37.283	4.251	208	37.087	4.195
Yrs. of education	445	6.081	3.879	208	6.120	3.833
Max. yrs. of adult members' education	445	7.153	3.559	208	6.837	3.628
Ave. yrs. of adult members' education	445	5.106	3.188	208	5.087	3.330
# of adult members	445	3.337	1.720	208	2.716	1.252
Panel B: KIDS04						
# of children aged less than 10	472	0.968	1.093	267	0.768	0.921
# of children aged 11 to 20	472	1.108	1.229	267	1.026	0.979
Age	472	37.411	4.290	267	37.382	4.237
Yrs. of education	465	7.903	4.469	265	8.185	4.118
Yrs. of education: Missing	472	0.015	0.121	267	0.007	0.086
Max. yrs. of adult members' education	472	10.532	3.643	267	9.985	3.644
Ave. yrs. of adult members' education	472	6.693	3.156	267	6.928	3.472
# of adult members	472	3.030	1.867	267	2.768	1.496

Notes: This table provides summary statistics of variable means and standard deviations with regard to children's quantity from KIDS93 in Panel A and from KIDS04 in Panel B. Observations are at the individual level. The sample consists of black African women aged 31 to 45.

Table 3: Summary Statistics on Children's Quality by Treatment Status

	Low Treatment Region			High Treatment Region		
	Obs.	Mean	Std. Dev.	Obs.	Mean	Std. Dev.
Panel A: KIDS93						
Yrs. of education	1,283	3.387	2.351	697	3.257	2.303
Age	1,283	10.783	2.584	697	10.684	2.683
Female dummy	1,283	0.482	0.500	697	0.504	0.500
Birth order	1,090	2.980	1.798	598	2.803	1.591
Birth order: Missing	1,283	0.150	0.358	697	0.142	0.349
Mother's education	983	5.112	3.663	547	5.439	3.798
Mother's education: Missing	1,283	0.234	0.423	697	0.215	0.411
Mother's age	1,038	37.811	8.291	561	37.759	9.299
Mother's age: Missing	1,283	0.191	0.393	697	0.195	0.397
Max. yrs. of adult members' edu.	1,283	5.873	3.829	697	5.623	3.946
Ave. yrs. of adult members' edu.	1,283	4.212	3.238	697	4.282	3.507
# of adult members	1,283	2.945	1.606	697	2.353	1.118
Panel B: KIDS04						
Yrs. of education	1,252	4.104	2.556	762	4.295	2.503
Age	1,252	11.028	2.544	762	11.081	2.510
Female dummy	1,252	0.502	0.500	762	0.505	0.500
Birth order	970	2.202	1.296	601	2.043	1.231
Birth order: Missing	1,252	0.225	0.418	762	0.211	0.408
Mother's education	899	7.109	4.336	561	6.995	4.138
Mother's education: Missing	1,252	0.282	0.450	762	0.264	0.441
Mother's age	791	36.910	9.233	504	36.680	9.750
Mother's age: Missing	1,252	0.368	0.483	762	0.339	0.474
Max. yrs. of adult members' edu.	1,252	9.581	4.251	762	9.109	4.142
Ave. yrs. of adult members' edu.	1,252	5.926	3.265	762	5.894	3.466
# of adult members	1,252	2.615	1.657	762	2.677	1.363

Notes: This table reports summary statistics of variable means and standard deviations with regard to children's quality from KIDS93 in Panel A and from KIDS04 from Panel B. Observations are at the individual level. The sample consists of black African children aged 7 to 15. Note that these cohorts in KIDS04 are the children who were eligible to free health services starting in 1994.

Table 4: Balancing Test of Baseline Characteristics

	Low Treatment Region		High Treatment Region			
	Obs.	Mean	Obs.	Mean	Diff.	Std. Err.
Panel A: Demographic characteristics (all individuals in the dataset)						
Years of education	5,097	4.655	2,743	4.794	-0.139	0.096
Age	5,099	24.122	2,743	23.991	0.131	0.435
Female	5,099	0.522	2,743	0.537	-0.015	0.012
Panel B: Quantity investments (women in 20s to 40s)						
Missing info. on pregnancy history	1,080	0.209	579	0.2	0.009	0.021
# of pregnancies	856	2.954	463	2.806	0.149	0.152
# of births	856	2.85	463	2.698	0.153	0.149
# of births still alive	856	2.451	463	2.309	0.142	0.127
Missing info. on children's deaths	856	0.163	463	0.158	0.005	0.021
# died before 1 year	715	0.231	390	0.233	-0.003	0.04
# died between age 1 and 5	715	0.151	389	0.111	0.041	0.031
Panel C: Quality investments (children aged 7 to 15)						
Years of education (all)	1,283	3.387	697	3.257	0.13	0.11
Years of education (boys)	664	3.2	346	3.052	0.148	0.146
Years of education (girls)	619	3.586	351	3.459	0.128	0.163
Panel D: Household characteristics (all households)						
Household size	648	6.657	384	6.292	0.366	0.23
Dependency ratio	648	0.344	384	0.341	0.003	0.013
Total monthly income	648	1,003.30	384	1,011.4 0	-8.051	66.81
Panel E: Community characteristics (all communities)						
# of primary schools	33	1.515	23	2.348	-0.833	0.514
# of secondary schools	33	0.636	23	1.087	-0.451	-0.155***
Distance to the nearest market	33	20.667	23	18.304	2.362	5.393
Distance to the nearest bank	33	23.121	23	21	2.121	5.215

Notes: This table reports the means of observable characteristics in the low treatment region, in the high treatment region, and the difference in means between the two and its standard error, using information from KIDS93. 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.
*Significant at the 10 percent level.

Table 5: Effect of Health Access on Child Quantity and Quality

Panel A:	Dep. var.: Schooling years				
Child Quality	(1)	(2)	(3)	(4)	(5)
High × Post	0.388	0.342	0.416	0.419	0.413
	(0.153)**	(0.145)**	(0.150)***	(0.161)**	(0.197)**
Community FE	Y	Y	Y	Y	Y
Cohort FE	N	Y	Y	Y	Y
Control vars.	N	N	Y	Y	Y
Sample	All aged 7-15			Boys aged 7-15	Girls aged 7-15
Observations	3,994	3,994	3,994	2,010	1,984
R-squared	0.082	0.685	0.685	0.653	0.729

Panel B:	Dep. var.: # of children aged				
Child quantity	Less than 10			Less than 5	6 to 10
	(1)	(2)	(3)	(4)	(5)
High × Post	-0.382	-0.369	-0.359	-0.204	-0.155
	(0.152)**	(0.150)**	(0.149)**	(0.087)**	(0.108)
Community FE	Y	Y	Y	Y	Y
Cohort FE	N	Y	Y	Y	Y
Control vars.	N	N	Y	Y	Y
Sample	Women aged 31 to 45				
Observations	1,399	1,399	1,392	1,392	1,392
R-squared	0.104	0.130	0.146	0.096	0.130

Notes: This table reports only the coefficient of interests based on equation (1). All specifications include community fixed effects and a post period dummy. All standard errors in the parentheses are clustered as the community level.
***Significant at the 1 percent level.
**Significant at the 5 percent level.
*Significant at the 10 percent level.

Table 6: Falsification Tests on Effect of Health Access

Panel A: Child quality	Dep. var.: Schooling years		
	(1)	(2)	(3)
High × Post	0.032	0.034	-0.002
	(0.304)	(0.339)	(0.391)
Cohort FE	Y	Y	Y
Community FE	Y	Y	Y
Control var.	Y	Y	Y
Sample	All aged 17-25	Male aged 17-25	Female aged 17-25
Observations	3,136	1,501	1,635
R-squared	0.300	0.304	0.330

Panel B: Child quantity	Dep. var.: # of children aged		
	11 to 20	11 to 15	16 to 20
	(1)	(2)	(3)
High × Post	-0.112	0.003	-0.115
	(0.145)	(0.102)	(0.093)
Cohort FE	Y	Y	Y
Community FE	Y	Y	Y
Control var.	Y	Y	Y
Sample	Women aged 31 to 45		
Observations	1,392	1,392	1,392
R-squared	0.176	0.122	0.213

Notes: See the notes in Table 5.

Appendix I

In this Appendix, we present evidence that mortality reductions were similar between the high and low treatment regions. As mentioned in the main text, our understanding on child mortality hinges on the observations in the previous work by Tanaka (2013) that the free health services resulted in sharp improvements in nutritional status only among affected children and without referring to a specific group of children at low nutritional status. This is suggestive that the pattern of mortality reduction was not associated with the type of the region. To the best of our ability without direct information about child mortality, we now explore this mechanism in more detail.

KIDS04 includes the question inquiring how many children the person has given birth to who were born alive but later died. The question was asked to all women between 15 years old and 49 years old. Limitations with this variable are that there is no equivalent question in the KIDS93 that we can compare with and that the question does not ask when child deaths occurred (thus it may include child deaths that took place before 1994 that 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 an extended number of 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 concern. Second, we can explore any differences in mortality across different ages of mothers. Since younger mothers are likely to have given birth in recent years, namely after 1994, we would find greater mortality reductions in high treatment region among younger mothers if the high treatment region have had greater reductions in mortality. This addresses the second concern.

We summarize the results in Table 1A. The dependent variable measures mortality constructed from dividing the number of child deaths by the number of all children born to the mother. The sample is women aged between fifteen and forty-nine from KIDS04. Column (1) reveals that the child mortality in the low-treatment region is about 10.3 percent, and the high-treatment region had 2.2 percentage point higher mortality rate. Importantly, the difference is not statistically significant, suggesting that there is no evidence highlighting differential patterns of child deaths between the two regions.

Child mortality increases with mother's age, as their children become older and there is a longer period 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 fallen more in the high-treatment region, the sign of the coefficient would be positive and significant. Our results suggest if anything the 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 adding its squared value and the interaction with the high-treatment region dummy, yet the results are the same. In column (3), we included community fixed effects in replace of the high-treatment region dummy, but we still get similar outcomes.

Overall, we find no evidence that differences in mortality reductions could explain the primary channel driving the main results.

Table 1A: 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 specifications are described in the text above. High is a dummy variable for the high-treatment region, and Age is mother's age (zero at the mean age).