

The causal effect of primary school reforms on women reproductive behaviors in Ethiopia. Is the expansion in education quantity the primary mechanism?

**Abstract** Several studies investigated the causal impacts of Africa's school reform programs on demographic outcomes. Many of the studies attributed the reform's causal effects to the post-reform expansions in the quantity of education. Nonetheless, the observed increases in school enrollment came at the expense of education quality needed to derive economic and social developments. The present study uses a formal mediation analysis framework to quantify and decompose the fertility effect of the 1994 Ethiopian school reform program into the impact through the most widely hypothesized mechanism (years of schooling) and the causal impact of the program net the effect of years of education. The results suggest a fertility-inducing effect of the reform mainly driven by mechanisms other than years of education. The fertility-reducing effects of increased school enrollment were too small to offset the positive impact of the unknown mechanisms. The result points to potential losses in the effectiveness of similar reform programs if adverse effects, such as deteriorated school quality, were not given as much attention.

**Keywords.** *School reform program. Reproductive behavior. Causal mechanisms. Matching. Mediation Analysis. Ethiopia. Years of education. Education quality.*

## 1. Introduction

Sub-Saharan Africa is the only major world region with a total fertility rate of above four children per woman, accompanied by the highest level of female illiteracy (WIC 2019; World Bank 2022). In an effort to promote education and improve other socio-economic outcomes, many sub-Saharan African countries underwent school reform programs in the 1990s (World Bank 2009). Studies indicate that the education reform programs encourage school enrollments and increase overall educational attainments in the post-reform periods (World Bank 2009; Moussa and Omoeva 2020; Majgaard and Mingat 2012). However, the overall impact of the universal primary education policies on the ultimate goal of 'improving the socio-economic well-being' is obscured. In addition, little is known about the causal effect of the hypothesized mediator (increased years of education) on the pathway between the reforms and intended socio-economic outcomes. For example, is the reform's impact (if any) on women's reproductive behavior conveyed through the post-reform expansions in the quantity of education? With such knowledge, policymakers can design similar programs that could mitigate mediators with adverse impacts and foster relevant mechanisms which promote desirable impacts.

Because educational attainment is a crucial determinant of women's reproductive decisions, policies that aim to promote education in sub-Saharan Africa were frequently presumed to have a fertility-reducing impact (Akinkoye 1979; May and Rotenberg 2020). However, improvement in the average years of schooling was only one of the immediate outcomes of the African school reform programs. Many African countries implemented the school reform programs with little or no planning, which caused a surge in primary school enrollments at the expense of education quality (Chimombo 2009; Grogan 2009; World Bank 2009; Zhang 2006). Further research confirmed that progress in school attainment made by many developing countries does not guarantee improvements in learning outcomes (Hanushek 2013; Filmer, Hasan, and Pritchett 2006; Pritchett 2013). These dual impacts of the reforms on education quality and quantity, for example, suggest competing mechanisms in the causal pathway between UPE policies and fertility outcomes.

The present analysis uses the potential outcome framework to address the following research questions. First, did exposures to the 1994 Ethiopian primary school reform have a causal impact on women's reproductive behavior? I employ a matching method that compares the fertility outcome of women below the primary school exit age (7-14) at policy implementation and exposed to the reform with those girls above the primary school exit age (15-22) in 1994. The matching methods select control units (age 15-22 in 1994) with similar confounding baseline characteristics as of the treated women. It ensures the identification of the treatment effect that the observed outcome differences are not due to confounders that may affect both the exposure to the reform and reproductive decisions. Second, what were the causal pathways through which the reform could have affected fertility outcomes? I use a two-stage formal mediation analysis developed by Flores and Flores-Lagunes (2009) to explore the contributions of possible mediators for the estimated impacts. More specifically, the method decomposes the estimated causal impact into the effects through the most widely hypothesized mechanism (years of schooling) and the causal impact of the program net the impact of years of education. The net treatment effect is the causal effect of the reform while 'blocking' the exposure's effect on completed years of schooling. No prior research made such an explicit decomposition while identifying the causal relationship between school reform exposures and fertility behaviors in sub-Saharan Africa.

The main contribution of this study is to disentangle the causal mechanisms of the UPE policy on women's reproductive behaviors. Therefore, it goes beyond prior studies which employ the school reform programs as quasi-natural experiments to exogenously identify the effect of years of schooling on child health and reproductive behaviors (Andriano and Monden 2019; Keats 2018; Behrman 2015; Ferre 2009). These Instrumental variable-based studies presume that reform exposure would affect outcomes of interest only through its effect on the supposedly primary mechanism (years of education) and ruled out the net mechanism (direct) effect of the UPE policies.

Using data from the Demographic and Health Surveys (DHS), my analysis demonstrates that exposure to the school reform program is causally associated with an early onset of childbearing and a larger number of children at age 25. The findings further suggested that the causal impact of the school reform was mainly driven by mechanisms other than years of education. Consistent with the literature, the causal effect of years of education is negative and statistically significant. However, it was too small to offset the primary school reform program's positive effects through unknown mechanisms. The unidentified mechanisms could include the adverse effects of the program through the deteriorated school quality. However, the present study could not further decompose the net-mechanism effects due to data limitations. In addition, the main inferences were robust to the possible effects of unmeasured confounder(s) which could determine both the exposure to the school reform and the potential mechanism/outcome.

### **Schooling, learning and fertility**

Numerous studies in sub-Saharan Africa attempted to establish a causal relationship between schooling and fertility outcomes. The quasi-experimental studies use instrumental variable and (fuzzy) regression discontinuity techniques that exploit exogenous variations in completed years of schooling following the introduction of universal primary education programs (Behrman 2015; Keats 2018; Makate and Makate 2016; Osili and Long 2008b; Zanin, Radice, and Marra 2015) or change in school systems (Ali and Gurmu 2018; Ferre 2009). It took advantage of the fact that school reforms are exogenous interventions that differently affect women's schooling. This is based on girls' age at the time of the intervention. The studies generally confirmed higher years of schooling of women directly exposed to an intervention, which led to delayed marriage and first birth, a smaller ideal family size, and lower overall fertility.

Expanding access to school could influence women's reproductive behavior in its own right without directly promoting family limitation behaviors and attitudes. Evidence showed that school attendance reduces girls' time and exposure to sexual activities and delays childbearing (Berthelon and Kruger 2011). Similarly, increased average years of schooling could negatively affect overall fertility by changing average group norms (Bicchieri et al. 1997) and social influence (Montgomery and Casterline 1993). However, sufficient fertility reduction needs reproductive decisions to be in the 'calculus of conscious choice', which would require empowering women with basic life-supporting skills. Literacy skills acquired in school may influence women's attitude and behavior with long-lasting impacts through the following broad pathways:

First, learning skills acquired in schools could improve efficient utilization of birth control methods (El-Ibiary and Youmans 2007), better use of health care services (Gakidou et al. 2010), improve overall health knowledge (Glewwe 1999), and ability to access, process and comprehend family planning information outside of school (R. A. LeVine et al. 2004; Islam and Hasan 2000).

Second, basic literacy skills enable women to conceptualize future fertility plans. Results from DHSs show that women with more schooling tend to report to questions of 'ideal family size' numerically. In contrast, women with no formal education are more likely to provide non-numerical answers, such as 'it is up to God' (Kebede, Striessnig, and Goujon 2021; Riley, Hermalin, and Rosero-Bixby 1993). The classical demographic transition theories posit that inability to assign numeric values to future childbearing plans reflect a "pre-transition" mindset that fertility is not in the 'calculus of conscious choice' (Caldwell 1976).

Third, skills acquired in school could enhance labor market earnings that would increase the opportunity cost of childbearing (Green and Riddell 2003; Galor 2011). Likewise, studies suggest that women's reading skill is the primary causal pathway between a mother's education and child health outcomes, contributing to the onset of fertility declines in high fertility settings (Smith-Greenaway 2015b; Notestein 1953).

Fourth, knowledge and skills gained through formal schooling and school-based reproductive health education led to higher exposure to different values and ways of thinking (R. LeVine, LeVine, and Schnell 2001; Ajuwon and Brieger 2007; Goldman and Collier-Harris 2012). In the same vein, schools provide an essential platform for disseminating ideas about alternative family norms, birth control methods, and sexual behaviors (Caldwell 1976; 1980).

Examining the effect of education on fertility outcomes for the sample of women included in the present analysis<sup>1</sup> revealed that studies based on only 'time served' in school could drastically underestimate the true impact of women's education. Table 1 compares the estimated effect of education on the cumulative number of children at age 25 from alternative samples. Column 1 shows results from the entire sample. The 'restricted' sample, column 2, excludes women who completed at least one year of primary education but had no reading skills. These exclude about one-third of women with some years of primary schooling but could not read a simple sentence written in their preferred language. The results from the 'full' sample revealed no statistically significant difference, in the number of children at age 25, between women with primary education and those with no formal education. In contrast, in the 'restricted' sample, primary education attainment is associated with a 14 percent reduction in the cumulative number of children at age 25.

Although the findings could not be interpreted as causal effects, it highlighted the importance of distinguishing the learning effect from the pure act of girls attending school. It is particularly essential in sub-Saharan Africa, where schooling has a weak correlation with learning (Smith-Greenaway 2015a). For example, the most recent DHSs showed that 70 percent of Ugandan women who completed third grade could not read a simple sentence written in their preferred

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<sup>1</sup> Includes women aged 7-22 at the implementation of the 1994 Ethiopian primary school reform.

language. In Ethiopia, while about 53 percent of women (15-49) have attended formal education, only one-fourth proved reading skills. In countries like Niger, more than one-fifth of women with six years of schooling have no essential reading skills.

*Table 1 estimated effects of education on fertility outcomes when schooling is not learning*

	Cumulative number of children at age 25	
	<i>'Full' sample</i> (1)	<i>'restricted' sample</i> (2)
<b>Women's education</b>		
None (reference)		
Primary	-0.033 (0.026)	-0.140*** (0.039)
Secondary	-0.821*** (0.032)	-0.872*** (0.032)
Completed secondary or more	-1.20*** (0.034)	-1.230*** (0.034)
Constant	1.699*** (0.009)	1.685*** (0.010)
Observation	19,152	16,698
AIC	59,396.25	51,460.38

Note: The table reports the Poisson model regression estimate of the effect of women's education on the cumulative number of children at age 25. The estimates are reported as marginal effect. Standard errors are shown in parentheses. Data are from Ethiopian DHSs and include women aged 24 or above at the time of the interview. The 'full' sample includes all women who were age 7-22 during the implementation of the 1994 school reform. The restricted sample excludes women with at least one year of primary schooling but were not able to read a simple sentence. Both models include controls for age of women at interview date, urban residence, religion, province of residence, and number of siblings. \*p<0.1; \*\*p<0.05; \*\*\*p<0.01

This study uses the 1994 Ethiopian school reform program to show the relative causal contribution of time served in school on reproductive behaviors. It hypothesized that the program effect net of the causal effect of years of education mainly reflects the effects of the deteriorated school quality caused by the mass education expansion program. However, the available data does not allow us to test the hypothesis.

## 2. Primary school reforms in Ethiopia

In the initial post-independence years, many governments in Sub-Saharan Africa were engaging in an ambitious expansion of free primary education and other social services, which creates

optimism in the continent (Ezenwe 1993). However, the time of high expectation was soon replaced by an unfortunate period of economic stagnation and political instability related to the mounting government external debt, world oil price shocks, and declining terms of trade (Ezenwe 1993; Bates, Coatsworth, and Williamson 2007). In an effort to “restructure the economy”, in the 1980s, many governments of the region implemented austerity measures, and introduced user fees for education and other public services (World Bank 2009). The indirect and direct cost of education coupled with existing political and socio-economic miseries led to a declining enrollment rates, higher drop outs and stalls in women’s educational progress (Avenstrup, Liang, and Nellemann 2004; Kebede, Goujon, and Lutz 2019; Unicef 1987). In the last three decades, however, many African governments implemented school fee abolishing policies, as part of wider policy reforms (World Bank 2009).

In 1994, with little prior planning, Ethiopia introduced a radically new ‘Education and Training’ policy. School fees were abolished across all grade levels of primary school (1-8) and the first two years of secondary school (9-10). In addition, the government provides grants to schools (US\$ 1.20- US\$ 2.35 per pupil per year) to match the pre-existing school fees (World Bank 2009). The immediate impact of the school reform in Ethiopia was a surge in primary school enrollments. In the following year of the reform, 317,000 additional students entered first grade, a 28 percent annual growth (Ministry of Education 1996). Primary school gross enrollment rose from 26.2 percent in 1994 to 79.8 percent in 2004. The improvement was particularly remarkable among girls, rural schools, and disadvantaged regions (World Bank 2009).

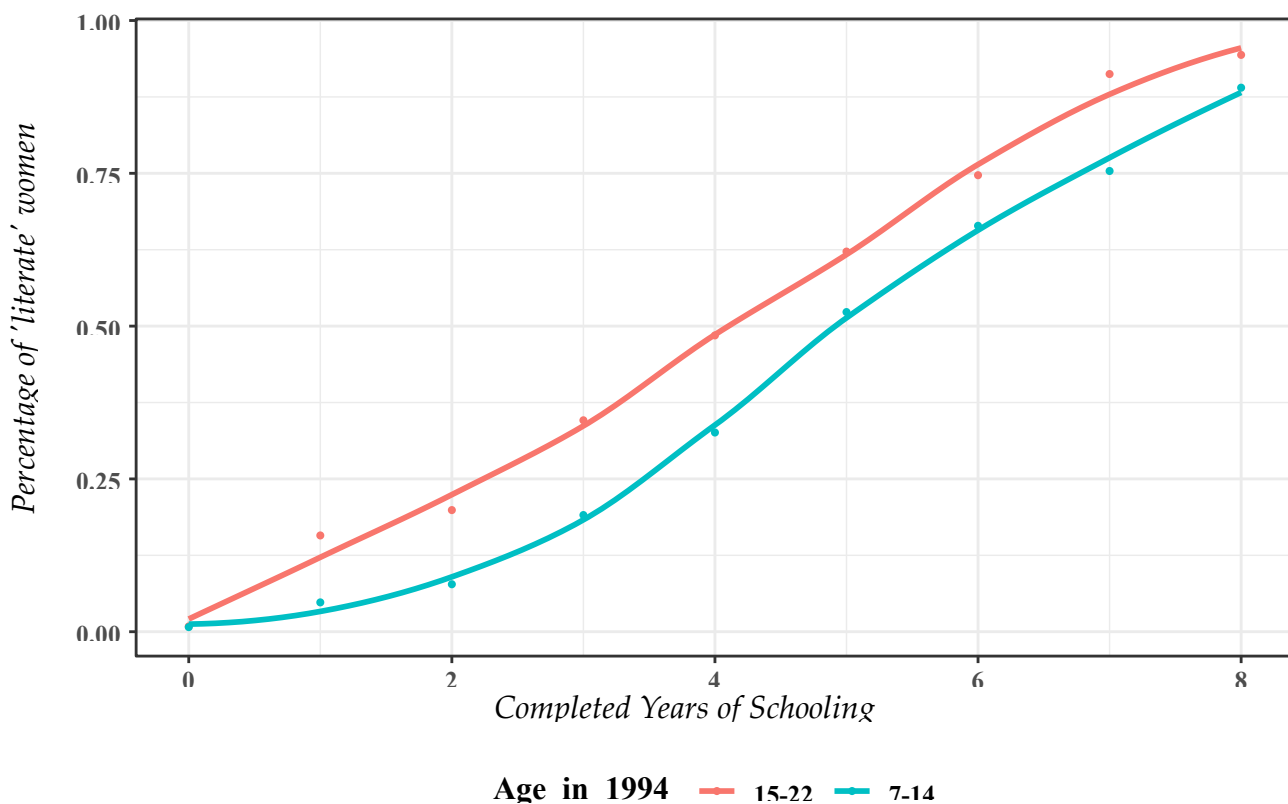
Elimination of school fees, though, was only one aspect of the reform. Curriculum reform, school feeding programs, decentralization, and alternative primary education in marginalized regions were instead the main components of the reform. The length of the primary school extends from 6 years to 8 years, with starting age of 7 remaining unchanged. Additionally, the decentralization policy allowed federal state governments to choose the language of instruction in primary schools and give discretion to local authorities in school administrations (Ministry of Education 1994). In the years prior to 1994, several regions decide to switch the language of instructions from Amharigna to local languages (Tigrigna in Tigray, Oromogna in Oromia, Somaligna in Somali region, Sidamigna , Wolayitigna , Hadiyigna , Gedeogna , and Kembatigna in SNNPR region)(Boothe and Walker 1997). Since primary schools were the main focus of the broader policy reform, I assumed primary-school-age women (7-14) in 1994 were exposed to the reform while women older than 14 during the reform were not affected by it.

## **Primary school reforms and school quality**

An insufficiently discussed aspect of the primary school reforms in sub-Saharan Africa was its adverse effect on school quality. Countries mainly adopt the so-called “big bang” approach and

implemented school reforms with little or no prior planning and negligible school infrastructural improvements (World Bank 2009). Though the sectoral budget in Ethiopia has increased with the reform, it could not match the surge in primary school enrollment. Thus, the primary mechanism to accommodate reform-triggered enrollment surges was by reducing resources available per primary school pupil. In Ethiopia, the pupil-teacher ratio in primary schools had declined since 1980 before it rose again with the reform. It increased from 26:1 in 1993 to 67:1 in the year 2000. Beside this, schools were forced to hire unqualified teachers to cope with explosions in enrollment. For example, by the year 2000, about 76 percent of upper primary school (5-8) teachers were not qualified (Education Management Information Systems 2000). Similarly, the mismatch between sectoral budget and enrollment expansions caused a more intensive use of classrooms, textbooks, and other school facilities. Between 1992 and 1996, the number of students per school increased by 90 percent, and an average classroom had to serve 20 more students in 2004 than it used to accommodate in 1994 (Ministry of Education 2000; World Bank 2009)

Figure 1: Reading skill vs MYS by women's age during the 1994 school reform



Author's calculation from DHSs

The deteriorated school quality in Africa was a challenge for cognitive improvement and learning outcomes (Pritchett 2013). The Ethiopian National Baseline Assessment in 1999 and 2004 clearly showed that expansions in enrollment were not translated into learning: the average test results

of Grade 4 and 8 students, in all subject areas, were below the minimum passing score of 50 percent. Comparing average reading skill of women in primary school age in 1994 with the preceding cohort, figure 1 revealed the deteriorated school quality following Ethiopia's primary school reform. At each completed year of primary school, the reading skill of women directly exposed to the 1994 school reform while of primary school age was significantly lower than those above school exit age at the reform implementation (15-22). For example, about 78 percent of women with four years of primary school and who were of primary school age in 1994 could not read a sentence written in their preferred language. In contrast, about 44 percent of their counterparts from the previous cohort were 'literate'.

### 3. Methods and Data

#### 3.1. Identification Strategy

The study seeks to quantify and decompose the impact of the 1994 Ethiopian school reform program on women's reproductive behavior (the outcome) into the effect through the most widely hypothesized mechanism (years of schooling) and the causal impact of the program net the impact of years of schooling. Exposure to the 1994 primary school reform implementation varies by women's age at the reform. While girls in primary school age (7-14) were likely to be affected during the reform implementation, girls above the primary school exit age were not exposed. In this analysis, I exploited the age-specific differences in the probability of exposure to the school reform to form the treated units (age 7-14 in 1994) and control units (age 15-22 in 1994).

To define these parameters of interest, I use the Neyman-Rubin potential outcome framework as suggested by Flores and Flores-Lagunes (2009). The expected causal effect of exposure to the school reform program ( $T$ ) for a randomly chosen woman ( $i$ ) that was actually exposed to the program, the average treatment effect on the treated ( $ATT$ ), can be defined as:

$$ATT = E[Y_i(1) - Y_i(0) / T_i = 1] - \dots - [E1]$$

Where,  $Y_i(1)$  and  $Y_i(0)$  are the potential outcomes for treated woman ( $i \in N$ ) that can be observed in the presence of the school reform program ( $T = 1$ ) and under the absence of the program ( $T = 0$ ), respectively. Thus, the  $ATT$  compares the observed outcomes for a treated woman with the counterfactual outcome that would be observed had they not been exposed to the reform.

The fundamental problem in the causal estimate of  $ATT$  is that, for women exposed to  $T$ , we can observe only  $E[Y_i(1)]$ . In a random treatment allocation where  $T$  is assigned independent of potential outcomes, one can use the observed outcomes of the untreated units,  $E[Y_i(0) / T_i = 0]$  as a surrogate for the counterfactual outcomes of the treated women.<sup>2</sup> However, assignments to the

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<sup>2</sup> The treated and untreated units are just two random samples from the same population



school reform program were conditional on a set of observable pre-treatment characteristics; for example, women from disadvantaged regions and at primary school age in 1994 were more likely to be exposed to  $T$ . Therefore, an appropriate treatment comparison invokes the following conditional independence assumption:

**Assumption 1.**  $Y(1), Y(0) \perp T/X$

Which states that the potential outcomes are jointly independent of the treatment assignment conditional on the set of covariates  $X$ . Assumption 1 further implies that, once we condition on  $X$ , the mean observed outcome for the untreated units is equal the mean counterfactual outcomes for the treated women, had they been exposed to the reform;  $E[Y_i(0)/T_i = 0] = E[Y_i(0)/T_i = 1]$ . In combination with the overlap assumption specified below, assumption 1 is sufficient for identifying the ATT.

**Assumption 2.**  $0 < pr(T = 1/X = x) < 1$

Assumption 2 states that the conditional distribution of the exposed and control units overlap for the vector of covariates or no combination of covariates perfectly predict treatment assignment.

## The (net) mechanism effect

The primary purpose of the study involves decomposing the *ATT* in to the effect through years of schooling (the mechanism effect), and the causal effect of the treatment while 'blocking' the exposure's effect on completed years of schooling (the net treatment effect). Years of schooling can be viewed as an intermediate outcome or a post-treatment mechanism variable in the causal pathway between treatment exposure and the final outcome of interest. Since the exposure  $T$  affects the mechanism variable ( $S$ ), any observed posttreatment mechanism variable could represent two different potential values:  $S_i(1)$  for a treated woman and  $S_i(0)$  if not exposed to the treatment.

In the same vein, one can derive the potential outcome of interest  $Y$  as a function of both  $T$  and  $S$ , and, for each  $i$ , four composite potential outcomes can be considered: (i).  $Y_i(1, S_i(1))$ , the potential outcome when a woman  $i$  is exposed to the reform and the mechanisms variable is affected by the exposure. This is equivalent to the potential outcome  $Y_i(1)$ . (ii).  $Y_i(0, S_i(0))$ , the outcome when the woman is not exposed to the reform and the mechanism is not affected by the treatment, and is equivalent to  $Y_i(0)$ . (iii).  $Y_i(1, S_i(0))$ , the outcome when a woman is exposed to the reform but the mechanism is kept at the level in the absence of the exposure. (iv).  $Y_i(0, S_i(1))$ , is the potential outcome a woman who were not exposed to the reform obtains if she receives the mechanism value as if she were treated.

Using the above composite potential outcomes, the mechanism average treatment effect for the treated (*MATT*) can be defined as:

$$MATT = E[Y_i(1, S_i(1)) - Y_i(1, S_i(0)) / X_i = x, T_i = 1] \text{ --- [E2]}$$

and the Net Average Treatment Effect (*NATT*) can be defined as the average causal effect of school reform exposure on an outcome, blocking the effect of the exposure on the post treatment mechanism ( $S_i$ ):

$$NATT = E[Y_i(1, S_i(0)) - Y_i(0, S_i(0)) / X_i = x, T_i = 1] \text{ --- [E3]}$$

The above equations imply that to estimate the *MATT* and *NATT* from non-experimental data, one must estimate the potential outcome for a treated unit under treatment, but blocks the effect of T on Y;  $Y_i(1, S_i(0))$ . This require to fix the observed value of the mechanism variable for each  $i$  at  $S_i(0)$ . In principle, one could adjust the exposure's effect on the mechanism, by simply controlling the observed values of  $S$ , in standard analytical approaches such as multivariate regression or matching [with or without controlling for years of education]. However, without satisfying several strong assumptions, simple treatment comparisons 'adjusting' for the observed values of a posttreatment variable lacks causal interpretations (Rosenbaum 1984; Rubin 2005; Flores and Flores-Lagunes 2009).

## Principal Stratification

In order to causally interpret our parameters, treatment comparisons were rather made based on the concept of principal stratification developed by (Frangakis and Rubin 2002) [ I employed the framework and notations of Flores and Flores-Lagunes (2009)]. The basic idea is to stratify units based on the values of the potential mechanism outcome  $S(0), S(1)$ , irrespective of treatment status. Women corresponds to a "principal stratum" defined by the joint potential values of a posttreatment variable  $\{s(0) = s_0, s(1) = s_1\}$  are comparable. The advantage of principal stratification over the naive approaches of controlling observed values of the mechanism values is that membership to a principal stratum is not affected by treatment assignment and, hence, principal effects are causal effects.

Adopting the basic concept of principal stratification our estimands can be redefined as:

$$MATT = E\{E[Y_i(1, S_i(1)) - Y_i(1, S_i(0)) / s(0) = s_0, s(1) = s_1, X_i = x, T_i = 1]\} \text{ --- [E4]}$$

and

$$NATT = E\{E[Y_i(1, S_i(0)) - Y_i(0, S_i(0)) / s(0) = s_0, s(1) = s_1, X_i = x, T_i = 1]\} \text{ --- [E5]}$$

Note that, *MATT* and *NATT* sum to *ATT*.

The practical difficulty in the identification and estimation of our parameters as defined above arise from the fact that the principal strata  $\{s(0) = s_0, s(1) = s_1\}$  is not observable. For example, a treated woman with 4 completed years of schooling may belong to either a stratum  $\{s(1) = 4, s(0) = 4\}$  or  $\{s(1) = 4, s(0) = 13\}$ . Therefore, in the absence of a counterfactual experiment that would allow us to directly observe  $Y_i(1, S_i(0))$ , one need to invoke additional conditional independence assumption:

**Assumption 3.**  $E[S_i(1)/X_i, T_i = 1] = E[S_i(0)/X_i, T_i = 0]$

which states that given a rich set of covariates  $X$ , treatment is not assigned based on expectation that the posttreatment mechanism outcome will be different under treatment and control conditions. Assumption 3, therefore, would enable us to find suitable counterfactual mechanism values from the other arm of the treatment.

### 3.1. Estimation strategy

While estimating and decomposing the *ATT* into the mechanism effect (*MATT*) and the net treatment effect (*NATT*) under the potential outcome framework, I follow a two-stage estimation strategy [a similar strategy was employed by Ferraro and Hanauer (2014)]. In the first stage, I use matching to create comparable treated and control units in terms of observable pretreatment confounding variables. Matching ensures the covariate distribution in the two arms of the treatment assignment looks as they would be in a randomized experiment [hence, assumptions 1 and 3 are satisfied]. By creating balance across covariates, matching provides an estimate of the *ATT*. In addition, it provides estimates for the counterfactual mechanism values  $S_i(0)$  for each treated unit, using the observed mechanism values from the matched untreated units.

I use optimal full matching with a caliper which involves assigning women into a subclass that contains one treated unit and one or more control units or one control unit and one or more treated units. The full matching method provides better covariate balance than pair matching techniques such as 1:1 nearest neighbor propensity score matching and 1:1 Mahalanobis distance matching [Appendix table S.2-S.4 compares covariates balance improvements with alternative matching methods]. Furthermore, full matching presents the additional advantage that the matching discarded no treated units to estimate the *ATT* effectively. The matched data set consists of 12,398 treated women and 12,201 control women. After matching, I estimated the average treatment effect on the treated (*ATT*) with regression bias adjustment and matching weights.

In the second stage, I follow the approach suggested by Flores and Flores-Lagunes (2009) to estimate the counterfactual outcome for treated units had treatment not affected the posttreatment mechanism variable,  $Y_i(1, S_i(0))$ . It is a regression-based method based on a simple assumption about the way the mechanism and the covariates affect outcomes in the original and

counterfactual treatments. It assumes that the conditional expectation of  $Y(1, S(0))$  and  $Y(1, S(1))$  in terms of  $\{X, S(0)\}$  and  $\{X, S(1)\}$ , respectively, shares the same functional form.

**Assumption 4.** Suppose

$$E[Y(1, S(1))/S(1) = s_1, X = x, T = 1] = f_1(S(1), X)$$

Then

$$E[Y(1, S(0))/S(0) = s_0, X = x, T = 1] = f_1(S(0), X)$$

which implies that the covariates and the mechanism have similar effects on the potential outcomes both in the original and counterfactual treatments.

(Flores and Flores-Lagunes 2009) suggested the implementation of the above method by first running a regression of the outcome of interest on the mechanism variable and all of the covariates, using the treated matched units. We then evaluate the counterfactual  $Y_i(1, S_i(0))$  by plugging an estimated counterfactual mechanism value  $\hat{S}_i(0) = E[S_i(0)/T = 1]$  and covariates  $X$  into the estimated coefficients. With the above adjustment the NATT can be rewritten as:

$$NATT = E[f_1(S(0), X)] - E\{E[Y^{obs}/T = 0, S^{obs} = s_0, X = x]\} \text{ --- [E6]}$$

And the  $MATT = ATE - NATE$ .

The counterfactual mechanism value for each treated unit was estimated using the information from the observed mechanism value of the corresponding control unit(s)<sup>3</sup>. First, we estimate a model for

$$E[S^{obs}/S(0) = s_0, X = x, T = 0] = f_0(X, T = 0) \text{ --- [E7]}$$

The coefficients from the above estimation are, then, used to estimate

$$[\hat{S}_i(0)/T = 1] = f_0(X, T = 1) \text{ --- [E8]}$$

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<sup>3</sup> In cases where there are more than one control matches for a treated unit within common principal stratum, the weighted median mechanism value was taken.

### 3.1. Data

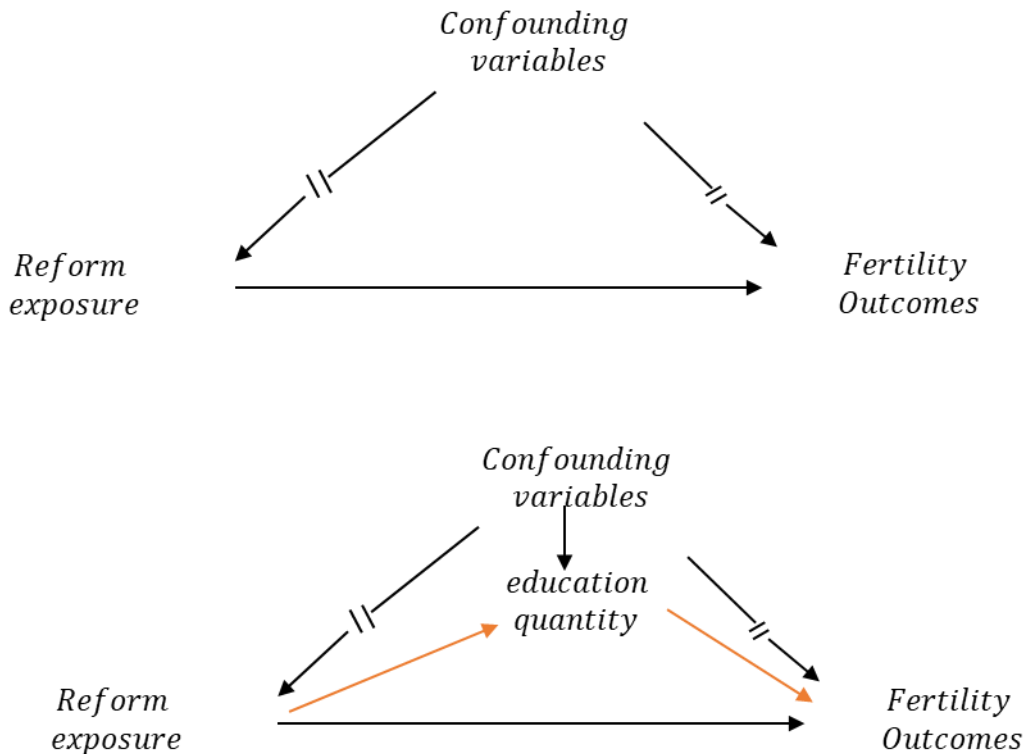
Data were mainly obtained from consecutive standard demographic and health surveys (DHS) conducted in 2000, 2005, 2011, and 2016. The DHS used a two-stage cluster sampling technique and standardized questionnaires to collect comparable and nationally representative data on population health, living conditions, and household demographics. Data from the four surveys were pooled to provide an adequate sample size for our analysis. The final sample consists of 12,398 women directly exposed to the 1994 primary school reform while of primary school age (7-14) and 12,767 women above primary school age (15-22) in 1994. I restricted the analysis to women who were 19 years old or above and possibly not attending school at the survey time. Table 2 provides summary statistics of key variables of the analysis

The Ethiopian DHSs provide multiple indicators of women's reproductive behavior (outcomes of interest) relevant to the demographic changes in sub-Saharan Africa. In this study, a binary outcome variable-whether a woman started childbearing before age 20-was used as an indicator of the timing of reproductive events. Additionally, to capture the quantum of fertility rather than differences in birth timing, I analyzed the cumulative number of children women had by age 25 y. The choice for the cut-off age of 25 was made due to data limitations. Table 2 revealed that women in the treated group tend to have larger number of children at age 25 and earlier onset of childbearing than their control group counterparts.

As discussed in the identification strategy section, I use the quasi-experimental design depicted in the directed acyclic graph (DAG) in Figure 2 to measure the effect of the school reform programs on fertility outcomes. Estimating the ATT is made difficult by confounding variables: factors that affect exposure to the school reform and also the potential outcomes. One can identify the effect of the education reform on fertility outcomes only by controlling for or blocking the effect of these confounding variables.

A set of pre-treatment confounding variables were identified in an effort to make the treatment assignment as good as randomized. Studies showed that rural residents benefited more from the school reforms than their urban counterparts (World Bank 2009). Thus, I control for the impact of urban residence, as it is defined in DHS (urban vs rural resident). Similarly, the decentralization policy associated with the school reforms allowed administrative regions to implement the reform differently. While some regions changed the language of instructions in primary schools to native languages, in others, even school fees were not eliminated until 1996. Therefore, a categorical variable for the administrative region was included as an exogenous covariate in the above model. Additionally, age at the time of the interview, the number of siblings, and a dummy for religion were controlled. The other key variable included in the estimation is the baseline Gross enrollment ratio (GER) by administrative zone and area of residence. The GER data was collected from the 1994 national census results. The distribution of these confounding variables considerably

varies by women’s exposure to the reform (see table 2). For example, exposed women were younger at an interview date, more likely to live in urban areas, and grew up with fewer siblings. Appendix table S.1 provide the list of variables, definitions and data sources.



*Figure 2: Directed acyclic graphs (DAGs) depict the empirical strategy for identifying the average causal and mechanism effects.*

Mechanism variable is an intermediate outcome in the causal pathway between the reform exposure and fertility outcomes (see figure 2). Access to schooling is the most widely hypothesized mechanism through which exposure to the school reforms could affect reproductive behaviors (Keats 2018; Pradhan and Canning 2016; Behrman 2015). Girls exposed to the reform program had an average of 0.37 more years of schooling than girls who were not at primary school age in 1994. Considering the country's overall low level of schooling (55 percent of women age 20-49 in 2016, for example, had no formal education), the observed difference is sizable. Therefore, we collect data on individual completed years of schooling from the DHSs.

Table 2: Summary statistics for key variables

	Control cohort (age in 1994: 15-22)	Exposed cohort (age in 1994: 7-14)	Full sample
	Mean (SD)	Mean (SD)	Mean (SD)
Years of school	2.62 (4.32)	2.99 (4.57)	2.80 (4.45)
No. Children at age 25*	1.66 (1.33)	1.75 (1.35)	1.70 (1.34)
1 <sup>st</sup> birth before age 20	.502 (.500)	.495 (.499)	.498 (.500)
Age at survey	30.6 (6.23)	26.86 (4.48)	28.75 (5.75)
Urban resident	.307 (.461)	.342 (.474)	.324 (.468)
Muslim	.346 (.475)	.370 (.482)	.358 (.479)
Number of Siblings			
[0,3]	.200 (.400)	.214 (.410)	.207 (.405)
[4,6]	.397 (.489)	.411 (.492)	.404 (.490)
More than 6	.401 (.490)	.374 (.483)	.388 (.487)
Region			
Tigray	.094 (.292)	.087 (.282)	.091 (.287)
Afar	.057 (.232)	.069 (.253)	.063 (.243)
Amhara	.125 (.331)	.115 (.319)	.120 (.325)
Oromia	.146 (.353)	.138 (.345)	.142 (.349)
Somali	.059 (.236)	.066 (.248)	.062 (.242)
Benishangul-Gumuz	.063 (.243)	.072 (.260)	.068 (.251)
SNNPR	.141 (.348)	.125 (.331)	.133 (.340)
Gambella	.055 (.229)	.065 (.246)	.060 (.238)
Harari	.063 (.244)	.062 (.242)	.063 (.243)
Addis Ababa	.121 (.327)	.129 (.335)	.125 (.331)
Dire Dawa	.070 (.255)	.067 (.250)	.068 (.252)
GER	.043 (.422)	.453 (.434)	.441 (.428)
Sample Size	12,767	12,398	25,165

Data were from DHSs including women aged 20 or above at the time of the interview; \*consider only women age 25 or more at interview date

## 4. Results

### 4.1. Effect of exposure to primary school reforms

In an attempt to identify the causal effects of exposure to primary school reforms on reproductive behaviors, table 3 presents the estimates from the naïve logistic and Poisson regression models. Column (1) and (2) compares the effects of women’s exposure to the reform program on the timing of first birth without and with controlling for relevant exogenous background characteristics, respectively. In a model that does not adjust for confounders, treatment status has no statistically significant association with first birth timing. However, accounting for confounding variables, reform exposure has a strong positive effect on reproductive behaviors; the odds of having a first child before age 20 were about 13 percent higher among those directly exposed to the reform while of primary school age than in the control group.

*Table 3. Pre-matching results: Estimated effect of exposure to primary school reforms on women’s reproductive behavior*

	<i>Reproductive Behavior</i>			
	<i>Odds Ratio</i>		<i>Rate Ratio</i>	
	(1)	(2)	(3)	(4)
Reform Exposure	0.97 (0.024)	1.131*** (0.031)	1.049*** (0.011)	1.074*** (0.012)
Constant	1.008*** (0.017)	0.561*** (0.052)	1.662*** (0.013)	1.530*** (0.071)
Adjusted for baseline differences	No	Yes	No	Yes
Obs.	25,165	25,165	19,096	19,096

Note: The table reports the conventional logistic and Poisson estimates of the effect of exposure to the 1994 Ethiopian school reform on indicators of reproductive behaviors. Data are from DHSs and includes women aged 20 or above at the time of the interview. Results in columns 2 and 4 controls for age of women at interview date, urban residence, religion, province of residence, number of siblings.

\*\*\*, \*\*, and \* represent significance at 1%,5% and 10% levels, respectively. terms in parentheses are robust standard errors

Similarly, women in the treated group tend to have more children at age 25; compared to older women, the birth rate of a woman of primary school age during the school reform increases by a factor of 1.05-1.07. The treatment effect is again more substantial in a model that controls baseline



characteristics such as community level baseline GER, religion, number of siblings, urban and region of residence, and age at the interview date.

The above comparisons revealed that the distributions of treatment characteristics of the treated and untreated women are different, which calls for matching methods to eliminate the influence of observable confounders. Table 4 displays impact estimates based on the 'full matching' method with post-matching regression bias adjustments. The results indicate that treated women had a differently larger number of children at age 25 and were more likely to have first birth before age 20 than they would have been in the absence of the program. The causal estimate, column 1, shows that treatment exposure increases the likelihood of early childbearing for those exposed to the reform by about four percentage points (or about 10 percent higher than the probability amongst the control group). Similarly, the average number of children at age 25 for a treated woman would have been ~0.11 smaller had she not been in primary school in 1994. This mean difference is equivalent to ~8.5 percent of the average number of ever-born children of control women at age 25.

*Table 4. Post-matching results: the average causal effects of exposure to the school reform program on reproductive behavior*

	<i>Reproductive Behavior</i>			
	First birth before age 20		Children at Age 25	
	(1)	(2) +	(3)	(4)+
ATT	0.041** (0.011)	0.052*** (0.010)	0.105*** (0.032)	0.170*** (0.032)
Constant	0.453*** (0.010)	0.326*** (0.041)	1.250*** (0.016)	1.091*** (0.149)
Matched Obs.	24,599	24,679	18,530	18,610
Matched treated-units	12,398	12,398	8,590	8,590

Note: The table reports the estimated impacts of the 1994 Ethiopian school reform program on those women exposed to the reform (the average treatment effect on the treated-ATT). The ATT was estimated using 'Full matching' method. Matching weights with robust standard errors and covariate adjustments were used to estimate the ATT. Covariate balances were made based on the following pre-treatment variables: community level baseline GER, age of women at an interview date, urban residence, religion, province of residence, and number of siblings. In Columns 2 and 4, however, years of schooling was included as an additional matching covariate.

Data are from DHSs and include women aged 20/25 or above at the time of the interview.

\*\*\*, \*\*, and \* represent significance at 1%, 5% and 10% levels, respectively. terms in parentheses are robust standard errors

+ the mechanism variable (years of schooling) was included as an additional matching covariate

## 4.2. Mechanism effects

To get an insight into the contribution of the mechanism variable to the estimated *ATT*, I calculated the net treatment difference (*NTD*) using the ‘full matching’ method that matches units based on pre-treatment variables and the mechanism (years of education). As shown in columns 2 and 4 of Table 4, the impact estimates significantly increased when adjusting for years of education (the mechanism); for example, the average treatment impact on the likelihood of early childbearing has substantially improved from about 4.1 percent to 5.2 percent after the adjustment. The estimated differences between the *NTD* (columns 2 and 4) and the *ATT* (columns 1 and 3) are suggestive of a fertility reducing mechanism effect (through years of schooling) and a substantial positive net mechanism effect (through unknown mechanisms). However, the estimation by simply adjusting for the posttreatment variable, without further assumptions, lacks causal interpretations as an average treatment effect (Rosenbaum 1984; Flores and Flores-Lagunes 2009).

Table 5. Mechanism effects: Estimated mechanism effects of primary school exposure on the treated women

Outcome	<i>observed mechanism</i>	counterfactual mechanism	<i>ATT</i>	<i>NATT</i>	<i>MATT</i>
	(1)	(2)	(3)	(4)	(5)
First birth before age 20	2.98	2.62	0.041** (0.011)	0.052*** (0.004)	-0.007 (0.005)
No. children at age 25	2.73	2.10	0.105*** (0.032)	0.154*** (0.012)	-0.036* (0.0187)
Matched treated- units			12,398	12,398	12,398

Note: The table reports decompositions of the *ATT* in to the *MATT* and *NATT*. Data are from DHSs and include women aged 20 or above at the time of the interview. All models include controls for age of women at interview date, urban residence, religion, province of residence, community level baseline GER and number of siblings.

\*\*\*, \*\*, and \* represent significance at 1%, 5% and 10% levels, respectively. terms in parentheses are robust standard errors

To have a causal interpretation of the estimates, I adopt the concept of principal stratification and estimation strategies discussed in the method section and evaluate the causal mechanisms underlying the average effects of the reform on the treated women. Table 5 summarizes how much of the estimated effect of the reform exposure was accounted for by the mechanism effect (through increased years of education) and through the net mechanism effects. The reform program increases the average years of schooling for those exposed. For example, for exposed women older

than 24 at the interview date, the reform exposure induced 0.63 additional years of schooling. Considering Ethiopia's low level of education, the 30 percent increase relative to the 2.1 average years of schooling in the control group is sizeable. The result further suggested that exposure to the school reform program has a fertility reducing effect through the supposedly primary mechanism (years of education) and opposite fertility inducing effect through the unknown mechanisms. Had the treatment not affected the mechanism, the estimated average number of children at age 25 and the likelihood of early childbearing for the treated women would have increased by about 46 percent and 27 percent, respectively. However, the fertility reducing impacts of the mechanism variable were too small to offset the positive net mechanism effects. Thus, the fertility effects of the program were predominantly driven by factors other than years of education.

### **4.3. Robustness checks**

In the following, I explore the sensitivity of the above results to changes in methodology bandwidth selection and intensity of exposure to the reform. Table 6 displays the exposure effect and the net mechanism effect on the treated women using three alternative matching methods for optimal full matching. The alternative matching methods produce positive and statistically significant fertility effects of the reform on the exposed women. The magnitude of the estimated effects on the cumulative number of children at age 25 is nearly identical to the estimates from the full matching method. Furthermore, the substantial positive net mechanism treatment effect remains robust to change in matching methods.

To investigate the sensitivity of the estimates to bandwidth selection, I re-estimate the specified models for different sizes of birth cohorts on each side of the cut-off (the reform year). The fertility-inducing treatment effect and the main result that the reform's effect was mainly driven by mechanisms other than years of education are robust to varying the bandwidth sizes [see appendix figure S.1].

Another concern is the potential differential impacts of the reform on the treated women based on the intensity of exposure to the program. For example, women closer to primary school entry age during the reform year were exposed longer than those at school exist age in 1994. Appendix Table S.5 compares the reform's effect on women of lower primary school age in 1994 with those of upper primary school age in the same year. The sensitivity check confirms the more substantial fertility-inducing reform effect on women exposed longer to the program while of primary school age in 1994.

Similarly, several regions made additional reforms by switching primary school languages from Amharigna to local languages. Women in these regions were, therefore, exposed differently than women of primary school age in regions with no language changes. A heterogeneity analysis

based on the introduction of local languages in primary schools confirms a stronger effect of the reform on women from the language reform regions.

*Table 6. Sensitivity checks: Estimated impacts of the reform exposure using alternative matching methods*

	<i>Reproductive Behavior</i>				No. matched treated units
	First birth before age 20		<i>Children at Age 25</i>		
	ATT	NATT	ATT	NATT	
1:1 Mahalanobis distance	0.028*** (0.006)	0.034*** (0.004)	0.115*** (0.019)	0.126*** (0.014)	12,398
1:1 Propensity score	0.028*** (0.006)	0.035*** (0.004)	0.118*** (0.019)	0.130*** (0.014)	12,398
1:1 Propensity score with caliper	0.024*** (0.006)	0.034*** (0.005)	0.093*** (0.020)	0.113*** (0.014)	9,952
Full matching	0.041** (0.011)	0.052*** (0.004)	0.105*** (0.033)	0.154*** (0.012)	12,398

Note: \*\*\*, \*\*, and \* represent significance at 1%,5% and 10% levels, respectively. terms in parentheses are robust standard errors

Finally, I assess how men's exposure to the reform would affect the reform's effect on women's reproductive behavior. About two-thirds of women in the sample are currently married, with an average of eight years age difference between husbands and wives. The considerable age difference between married couples implies that treated women were likely married to husbands who did not expose to the reform themselves. To glimpse the role of men's exposure to the reform, I compared the treatment effect on treated women married to treated men (vs. untreated women) and the treatment effect on treated women married to untreated men (vs. untreated women). The findings revealed a more substantial positive effect of the treatment in the transition to

motherhood in the latter group. However, we found no difference in the treatment effect on the number of children at age 25 between the two groups [see appendix table S.5].

Though the findings provide an insight into the role of men in fertility decisions, they should be interpreted with caution: First, potential sample selection and collider problems could emanate from restricting the sample to only currently married women; Women's treatment status could influence the marital status and partner selection. Second, it is unclear whether the differential impact of the reform based on the partner's exposure reflects the fertility reducing impact of the husbands' exposure or the fertility inducing impact of age differences (e.g., through child marriage with older men). Future work can expand on these findings by decomposing the relative contributions of the two possible factors. Third: the analysis did not consider the pre-treatment characteristics of men.

#### **4.4. Identification based on Sensitivity checks**

##### **4.4.1. Sensitivity analysis on the ATT**

As discussed in the method section, the causal identification of the effect of reform exposure on women's reproductive behavior (ATT) invokes a strong assumption that the education reform is as good as a random experiment conditional on a set of pre-treatment covariates. The plausibility of this assumption requires a rich set of observed confounding variables that jointly affect the treatment assignment and the potential outcome (Huber 2020). On the other hand, failure to control for an essential confounding variable could lead to a different conclusion about the reform's effect on fertility outcomes. Therefore, I try to identify the ATT controlling for pre-treatment covariates included in the matching exercise; community level baseline gross enrolment ratio, urban residence, region of residence, number of siblings, religious affiliation, and age of women.

However, despite the effort to control for observable sources of biases, one cannot rule out the possible effects of unobserved confounders jointly affecting the treatment assignment on the one hand and the potential outcomes on the other hand. For example, the estimated ATT is biased if one believes that unmeasured household characteristics, such as household wealth during childhood, would positively affect both exposures to the reform and fertility behaviors during adulthood. Therefore, practitioners proposed various sensitivity analysis methods that allow us to investigate the robustness of our estimates to the departure from the identification assumptions. i.e., How strong would the unobserved confounders have to be substantially altered our conclusion about the ATT?

Frank (2000) proposed a sensitivity index to quantify how strong the impact of an unmeasured confounder must be in our matching estimate to invalidate the above-discussed inferences. The method is based on the notion that for an observed confounder to affect the results, it must be strongly correlated with the treatment and potential outcome variables. I use the R package

konfound developed by Rosenberg, Xu, and Frank (2019). As shown in Table 7, to invalidate our causal inference of the strong and positive effect of exposure to school reform on the number of children at age 25, 66 percent of the estimate would have to be due to bias. It implies that the estimated impact would be reduced to zero only if we could replace about 66 percent of the observations (RIR=12,279) with hypothetical observations for which reform exposure has no effect on fertility outcomes. Similarly, to invalidate the inference of an effect of reform exposure on the likelihood of early childbearing, we would have to replace 72 percent of the observations with cases where there is zero effect of the reform.

Table 7. *Quantifying the Robustness of Inferences from the ATT Estimates using Frank's (2000) Indices*

Outcome	ATT	Impact threshold at 5% sign. Level	Bias (%)	RIR	Matched Obs.
<i>Children at Age 25</i>	0.105*** (0.032)	0.036	66.265	12,279	18,530
First birth before age 20	0.041** (0.011)	0.012	72.083	17,732	24,599

Though the degree of bias presented in table 7 are too high to invalidate our main conclusions, we also need to compare the impact of the unobserved covariates relative to the importance of the observed covariates. therefore, I use the sensitivity analysis recently proposed by Cinelli and Hazlett (2020) , which extends the familiar omitted variable bias (OVB) framework. The proposed method assesses the robustness of the estimated effect (the ATT) to the presence of an unobserved covariate of a different strength measured relative to the explanatory power of observed covariates. In addition, it provides bounds on the partial R2 of the unobserved confounder k times as strong as the observed covariate. (For this sensitivity analysis, I use the R package *sensemakr* developed by Cinelli, et al. ( 2020).

The bivariate sensitivity contour plot, displayed in Figure 3, presented the average treatment effects adjusted for the presence of a potential confounder parameterized by its relation to the treatment assignment, and the outcome. The horizontal axis displays the hypothetical partial R2 of the confounder with the treatment  $R2_{T\sim Z/X}$ , and the vertical axis depicts the hypothetical share of outcome variations,  $R2_{Y\sim Z/X}$ , explained by the unobserved confounder. Panel A in figure 3 shows the average causal effect of the treatment assignment on the number of children at age 25 adjusting for a confounder of different strengths relative to the partial R2 of the observed

covariates, *age*. The benchmark covariate, *age*, is the most important observed covariate in explaining the treatment assignment, as exposure to the school reform program was based on the birth cohort of women. A confounder as strong as the observed variable, *age*, would have partial  $R^2$  with the treatment  $R^2_{T \sim Z/X} = 9.8\%$ , and with the outcome  $R^2_{Y \sim Z/X} = 0.45\%$ . The test result revealed that, at 5 percent significance level, the null hypothesis of zero effect would still be rejected given unobserved confounder as strong as *age*. Instead, it requires an extremely powerful confounder, at least twice as strong as *age*, to radically change our conclusion about the estimated ATT. In addition, as *age* is directly related to the criteria we use for the treatment assignment, an unobserved confounder stronger than *age* in explaining the treatment is unlikely.

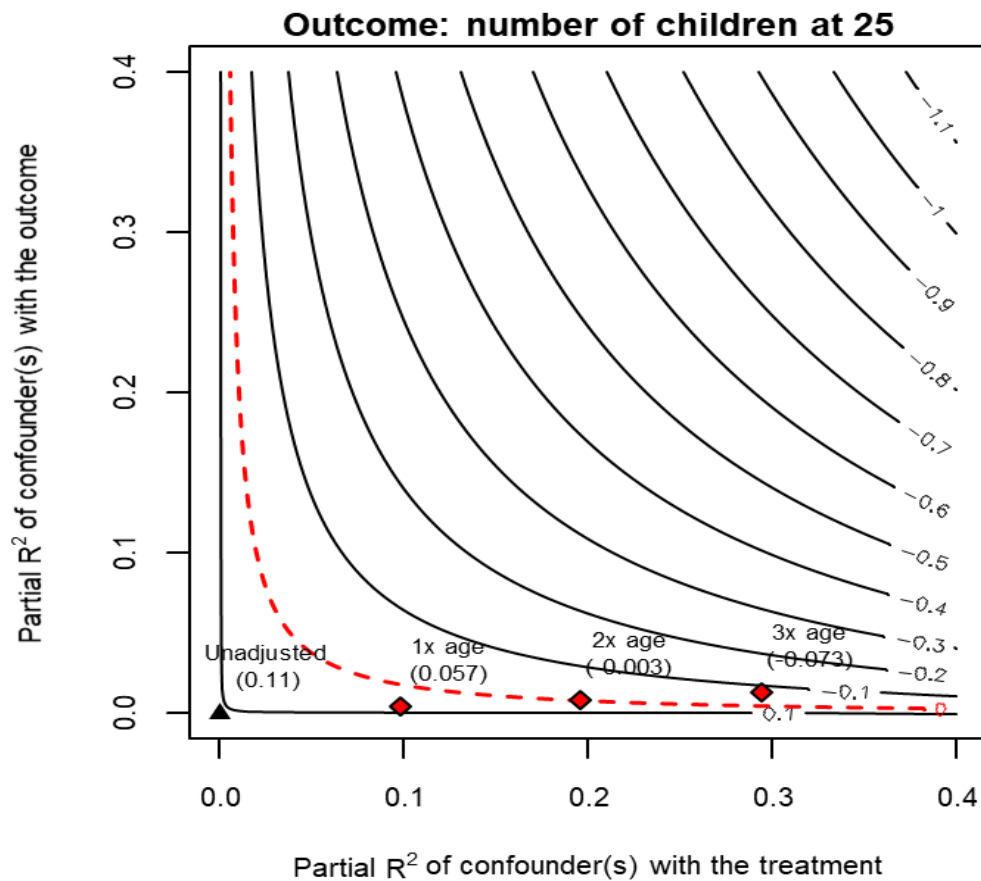


Figure 3. Sensitivity contour plots of point estimates for the causal effect of school reform programs on the number of children at age 25, using the approach proposed by Cinelli and Hazlett (2020).

I have also checked, in Appendix Figure S.3, the sensitivity of the estimated treatment effect for the presence of unobserved confounder measured relative to other observed covariates, urban residence, and religion. These are essential covariates, but less important than age, in explaining the variations in treatment assignment and the number of children at age 25. The contour plot shows that the unmeasured confounder, three times as strong as religion and urban residence, would significantly change neither the size nor the direction of the estimated effects. A similar sensitivity test for the outcome, birth before age 20, revealed the robustness of the estimated treatment effects for the hypothesized unmeasured confounder. In conclusion, the primary finding that exposure to the 1994 school reform program induces fertility could change only in the presence of exceptionally powerful unobserved confounders.

#### 4.4.2. Sensitivity analysis on the average (neat) mechanism effects

The other key identification assumptions in the main analysis was the conditional independence of the mechanism variable, also called the sequential Ignorability assumption. It rules out sets of possible unobserved or unmeasured confounders jointly affecting the mechanism and the potential outcomes given the randomization of the treatment assignment and the rich set of pretreatment covariates (section 4.4.1 discussed the randomization of the treatment assignment).

Imai et al. (2010) developed a widely used sensitivity analysis method for the partial identification of direct and indirect effects evaluated based on the linear structural estimation model (LSEM) in the spirit of Barron and Kenny (1986). Under the LSEM, the causal effects are estimated from the following regressions:

$$S_i = \alpha_2 + \beta_2(T_i) + \partial_2 X_i + \varepsilon_{i2} \text{ --- (4.1)}$$

$$Y_i = \alpha_3 + \beta_3(T_i) + \gamma(S_i) + 3X_i + \varepsilon_{i3} \text{ --- (4.2)}$$

Where  $Y_i$ ,  $S_i$ , and  $T_i$  stands for the observed values of the outcomes, the mechanism variable and the treatment, respectively. Both equations include the observed covariates  $X$ , included in the main analysis. Baron and Kenny (1986) proposed the direct effect (akin the NATT) be assessed by estimating  $\beta_3$ , and the total effect (comparable to the MATT) by estimating  $\beta_2 \cdot \gamma$ .

Employing the Imai et al. (2010a) simulation-based estimation strategy, the results of the LSEM estimation are shown in table 8 (I use the mediation package to implement it in R). The estimates are comparable with the results from the primary analysis. The total effect, which is analogous to the ATT, indicates that women exposed to the reform were more likely to give birth before age 20 and have a higher number of children at age 25. Similarly, consistent with our primary results,



the treatment effects were mainly channeled through mediators other than the hypothesized mechanism. The direct effect of exposure to the reform caused a 3.5 percentage point increase in the likelihood of adolescent birth and a significantly higher number of children at age 25. On the other hand, the indirect effect conveyed through education constitutes only one-third of the total treatment effect on the likelihood of giving birth at an early age. The similarity of the above estimates with our primary findings demonstrates the robustness of our results to alternative estimation strategies.

*Table 8. Estimated direct and indirect effects of exposure to primary school reforms on women's reproductive behavior using the Linear Structural Estimation Models (LSEM)*

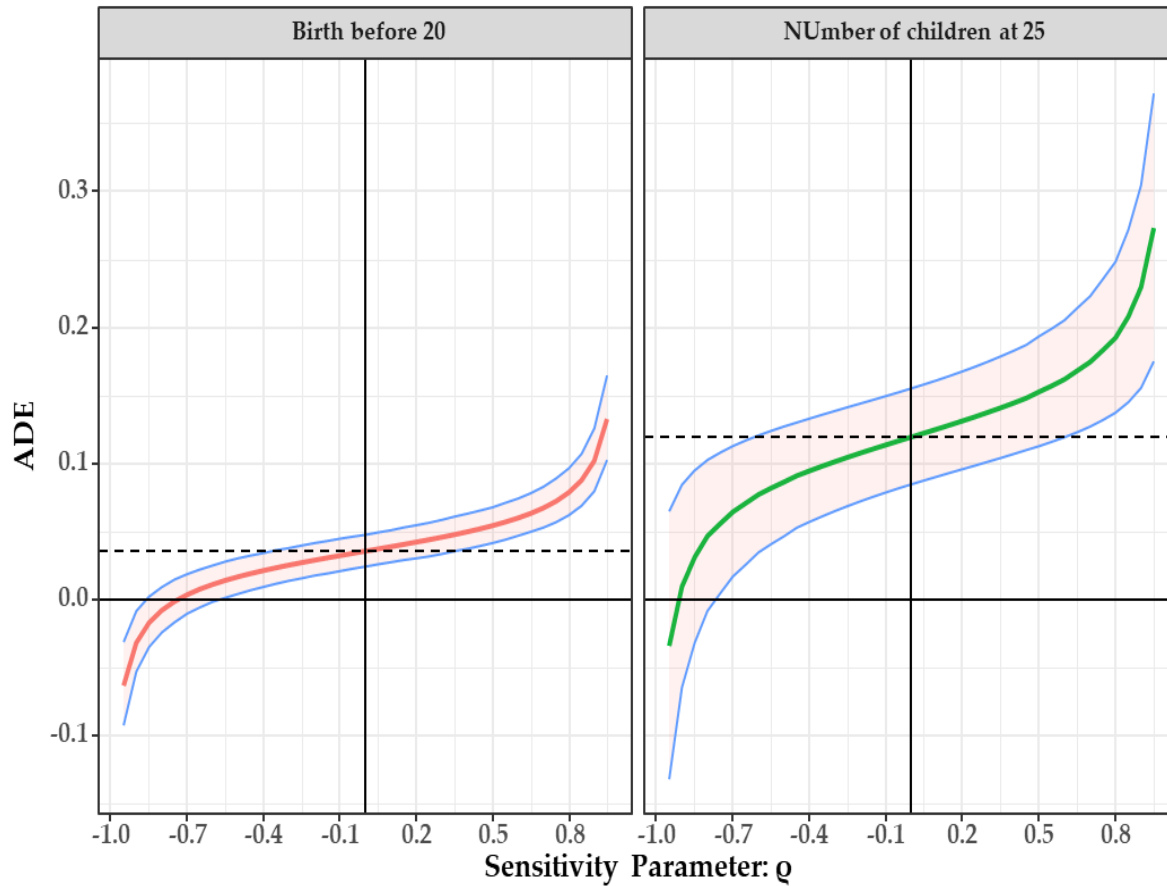
	<i>Reproductive Behavior</i>			
	First birth before age 20		Children at Age 25	
	Estimate	95% CI	Estimate	95% CI
Total Effect	0.028***	[0.015, 0.04]	0.1092***	[0.0717, 0.150]
ADE (treated)	0.035***	[0.023, 0.051]	0.1194***	[0.0831, 0.160]
Proportion mediated (treated)	-0.291***	[-0.547, -0.17]	-0.0904***	[-0.1849, -0.040]
Obs.	24,517	24,517	18,448	18,448

Note: All models include controls for age of women at interview date, urban residence, religion, province of residence, community level baseline GER and number of siblings.

\*\*\*, \*\*, and \* represent significance at 1%, 5% and 10% levels, respectively

*How robust is our conclusion that the treatment effect was mainly channeled through the mediators other than education (the NATT effect) to the departure from the sequential ignobility assumptions?* Using the method suggested by Imai, Keele, and Yamamoto (2010), it was judged by evaluating the correlations  $\varepsilon_{i2}$  and  $\varepsilon_{i3}$  in equations (4.1 -4.2). The correlation coefficient, denoted by  $\rho$ , is called the sensitivity parameter. For the sequential ignobility assumption to hold, the two residuals should be uncorrelated ( $\rho = 0$ ). On the other hand, the non-zero correlation of  $\varepsilon_{i2}$  and  $\varepsilon_{i3}$  indicate the violation of the conditional independence of the mechanism. The basic idea of the sensitivity check is to assess how big the unobserved confounder effect, measured by  $\rho$ , must be in order to invalidate the above estimates. Estimates that would be negated only at relatively large values are robust to the presence of potential unobserved confounders.

Figure 4 : Sensitivity analysis for the Average Direct Effects (ADE)



The monotonic curves in Figure 4 display the estimated direct effect (the NATT) for values varying between -1 and 1. The horizontal dotted lines represent the estimated direct effect under the assumption  $\rho = 0$ . The point where the curve meets the  $Y = 0$  line represents the threshold  $\rho^*$  at which the average direct effect (ADE) would be zero. The smaller the  $\rho^*$ , the more susceptible the estimated effect is to confounding by unmeasured covariates. In our sensitivity exercise, the  $\rho^*$  values are  $-0.75$  and  $-0.9$  for the outcome indicators birth before age 20 and the number of children at age 25, respectively. These relatively big values indicate that the estimated direct effect could be minimized to zero only if there were a substantial violation of the identification assumption. Therefore, the positive and significant net average treatment effect on fertility indicators are robust to deviations from the basic identification assumption of no unmeasured confounding factor jointly affecting the mechanism and the potential outcomes.

## 5. Discussions and conclusions

Many studies have examined the relationship between school reform programs and demographic outcomes in sub-Saharan Africa. These studies exploit exogenous variations in female schooling caused by education reforms to show the causal links between education and desired number of children (Behrman 2015), teenage fertility (Pradhan and Canning 2016; Osili and Long 2008a), and child mortality (Andriano and Monden 2019; Makate and Makate 2016). However, the possible effects of mechanisms other than years of education have been ruled out through the exclusion restrictions that reform exposure could influence outcomes of interest only through its effect on years of education.

This study investigated the causal mechanisms through which exposure to the 1994 Ethiopian school reform program affects women's reproductive behavior. In particular, it considers the reform's effect through mechanisms other than years of schooling (unknown mechanisms). The estimation results revealed positive and sizable reform effects on timing to first birth and the number of children at age 25. The findings further implied that the causal impact of the school reform was mainly driven by mechanisms other than years of education. Consistent with the literature, the causal effect of years of education is negative and statistically significant. However, it was too small to offset the positive effects of the primary school reform program through the unidentified mechanisms.

Consistent with our findings, previous studies have shown lower than expected socio-economic impacts of primary school expansions in sub-Saharan Africa (Liu and Raftery 2020; Grant 2015; Mensch, Singh, and Casterline 2005; Zanin, Radice, and Marra 2015). In Ethiopia, for example, the proportion of women with at least one year of primary education doubled between 2000 and 2016. In contrast, the median age at first birth remained at about 18 years, and the median age at first marriage shows a modest increase from about 15 to 16 years. These seemingly weak associations between primary educational attainment and fertility indicators have led some scholars to call for an extension of the threshold level of compulsory schooling beyond primary education (Maïga 2013), or claim factors other than education for the speed of Africa's fertility decline (Garenne 2012).

Nonetheless, the above results should not be misinterpreted as evidence of the irrelevance of education on fertility in the region. It only reflects the inefficiency of the school expansion programs. The unidentified mechanism effects could include the adverse effects of the school reform programs through deteriorated school qualities. Empirical evidence showed that expansions in the quantity of education in many developing countries came at the expense of the quality needed to derive economic growth and social developments (World Bank 2009; Pritchett 2013). Future studies with richer measures of quality of education (not just years of education)

could better quantify the mechanisms through which school reforms affect socioeconomic outcomes. The data from the demographic and health surveys capture only one aspect of women's literacy status- reading skills. However, cognitive differences that alter perception and abstract skills require functional literacy, such as writing, arithmetic, oral communication skills, or abstract thinking.

Our findings should not also be interpreted as evidence against rapid education expansion programs, particularly in sub-Saharan Africa, where one-fifth of girls are still out of school. While the gap between schooling and literacy has recently widened, mass education expansion programs improved education opportunities and average literacy levels. This overall progress in the two education indicators could contribute to fertility declines in sub-Saharan Africa in two ways. First, school participation could delay girls' marriage and the onset of childbearing through the pure "incapacitation" effect. Staying in school delays the onset of childbearing, risk of early marriage, and pregnancy (Lloyd and Mensch 2008). Second, overall improvements in literacy and average years of schooling could influence reproductive behaviors through social interaction effects (Bongaarts and Watkins 1996; Montgomery and Casterline 1996).

To conclude, development efforts in accelerating fertility declines in sub-Saharan Africa must look beyond expanding primary education for all. If not adequately addressed, the increasing learning deficit in the region will continue to undermine the role of education in supporting socio-economic support progresses.

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## **Declarations**

**Conflict of interest:** The author declares no competing interests that are relevant to the content of this article.

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## Appendix

Table S.1: variable definition and data sources

Variable	Definition	Source
<b>Outcome variables</b>		
1 <sup>st</sup> birth before age 20	A binary indicator of whether the woman give birth before age 20 or not	DHS
Number of children at age 25	The cumulative number of live births occurred to a woman before age 25	DHS
<b>Confounding variables</b>		
Age	Age of women at the time of the survey in years	DHS
Area of residence	Area of residence (urban or rural) of sampled women	DHS
Religion	A binary indicator of whether the sampled woman is a Muslim or not	DHS
Number of siblings	A factor variable for number of siblings of a sampled woman ([0,3], [4,6], more than6)	DHS
Region	A factor variable indicating the administrative region where the woman resides	DHS
GER	The Gross enrollment ratio in 1994 by rural -urban and administrative zone	Central Statistics Authority

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*Table S.2: summary of covariate balance before and after optimal full matching*

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Covariate	Description	Status	Mean treated units	Mean control units	Absolute Std. mean differences
Age	Age of women at interview date	Pre-matching	26.86	30.89	0.830
		matched	26.86	26.90	0.008
Urban	The unit resides in an urban area	Pre-matching	0.342	0.301	0.074
		matched	0.342	0.330	0.024
siblingNo3	has 3+ siblings	Pre-matching	0.374	0.401	0.049
		matched	0.374	0.364	0.021
Muslim	Religious affiliation is Islam	Pre-matching	0.370	0.346	0.056
		matched	0.370	0.366	0.007
Region	Province of residence*				
GER	The gross enrollment ratio in 1994 by administrative zone and by rural and urban residence	Pre-matching	0.453	0.427	0.058
		matched	0.453	0.471	0.041

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\*See table 2 for the list of provinces

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*Table S.3: summary of covariate balance before and after 1:1 nearest neighbor matching*

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Covariate	Description	Status	Mean treated units	Mean control units	Absolute Std. mean differences
Age	Age of women at interview date	Pre-matching	26.86	30.89	0.830
		matched	26.86	30.23	0.750
Urban	The unit resides in an urban area	Pre-matching	0.342	0.301	0.074
		matched	0.342	0.310	0.068
siblingNo3	has 3+ siblings	Pre-matching	0.374	0.401	0.049
		matched	0.374	0.399	0.038
Muslim	Religious affiliation is Islam	Pre-matching	0.370	0.346	0.056
		matched	0.370	0.351	0.052
Region	Province of residence*				
GER	The gross enrollment ratio in 1994 by administrative zone and by rural and urban residence	Pre-matching	0.453	0.427	0.058
		matched	0.453	0.471	0.041

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\*See table 2 for the list of provinces

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*Table S.4: summary of covariate balance before and after 1:1 Mahalanobis distance matching*

Covariate	Description	Status	Mean treated units	Mean control units	Absolute Std. mean differences
Age	Age of women at interview date	Pre-matching	26.86	30.89	0.830
		matched	26.86	30.30	0.767
Urban	The unit resides in an urban area	Pre-matching	0.342	0.301	0.074
		matched	0.342	0.312	0.062
siblingNo3	has 3+ siblings	Pre-matching	0.374	0.401	0.049
		matched	0.374	0.388	0.028
Muslim	Religious affiliation is Islam	Pre-matching	0.370	0.346	0.056
		matched	0.370	0.348	0.044
Region	Province of residence*				
GER	The gross enrollment ratio in 1994 by administrative zone and by rural and urban residence	Pre-matching	0.453	0.427	0.058
		matched	0.453	0.471	0.041

\*See table 2 for the list of provinces

Table S.5: the impact of length of exposure

	<i>(a). women who were 7-10 y in 1994</i>		<i>(b). women who were 11-14 y in 1994</i>	
	First birth before age 20	Children at Age 25	First birth before age 20	Children at Age 25
ATT	0.050*** (0.013)	0.141*** (0.039)	0.034*** (0.011)	0.084*** (0.033)
NATT	0.063*** (0.011)	0.202*** (0.033)	0.040*** (0.010)	0.119*** (0.027)
Obs.	5,830	3,892	6,568	4,698

*Note: the control group in both cases were married women who were not exposed to the reform.*

*\*\*\*, \*\*, and \* represent significance at 1%,5% and 10% levels, respectively. terms in parentheses are robust standard errors*

*All models include controls for age of women at interview date, urban residence, religion, province of residence, and number of siblings*

Table S.6: Estimated effect of exposure to primary school reforms on reproductive behavior

	<i>(a). Married women who were exposed to the reform and married to treated men</i>		<i>(b). Married women who were exposed to the reform but married to untreated men</i>	
	First birth before age 20	Children at Age 25	First birth before age 20	Children at Age 25
Net Treatment Difference	0.030** (0.014)	0.172*** (0.040)	0.047** (0.009)	0.173*** (0.027)
Constant	0.507*** (0.011)	1.640*** (0.033)	0.579*** (0.007)	1.889*** (0.020)
Partners' age difference	2.99	2.79	10.53	1.055
Obs.	2,809	2,101	6,168	4,689

Note: the control group in all cases were married women who were not exposed to the reform.

\*\*\*, \*\*, and \* represent significance at 1%,5% and 10% levels, respectively. terms in parentheses are robust standard errors

All models include controls for age of women at interview date, urban residence, religion, province of residence, number of siblings, and husband's current age

Figure S.1: Treatment effects on the likelihood of early childbearing for varying bandwidths

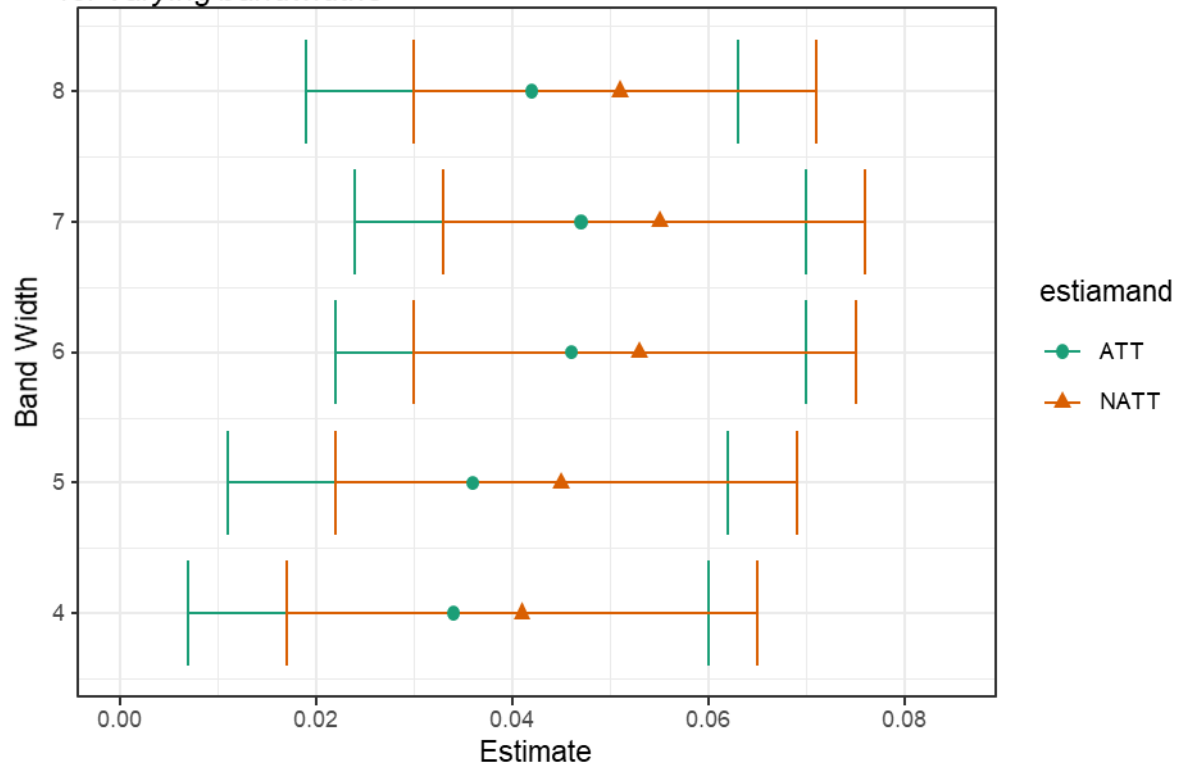


Figure 3. Sensitivity contour plots of point estimates for the causal effect of school reform programs on the number of children at age 25, using the approach proposed by Cinelli and Hazlett (2020).

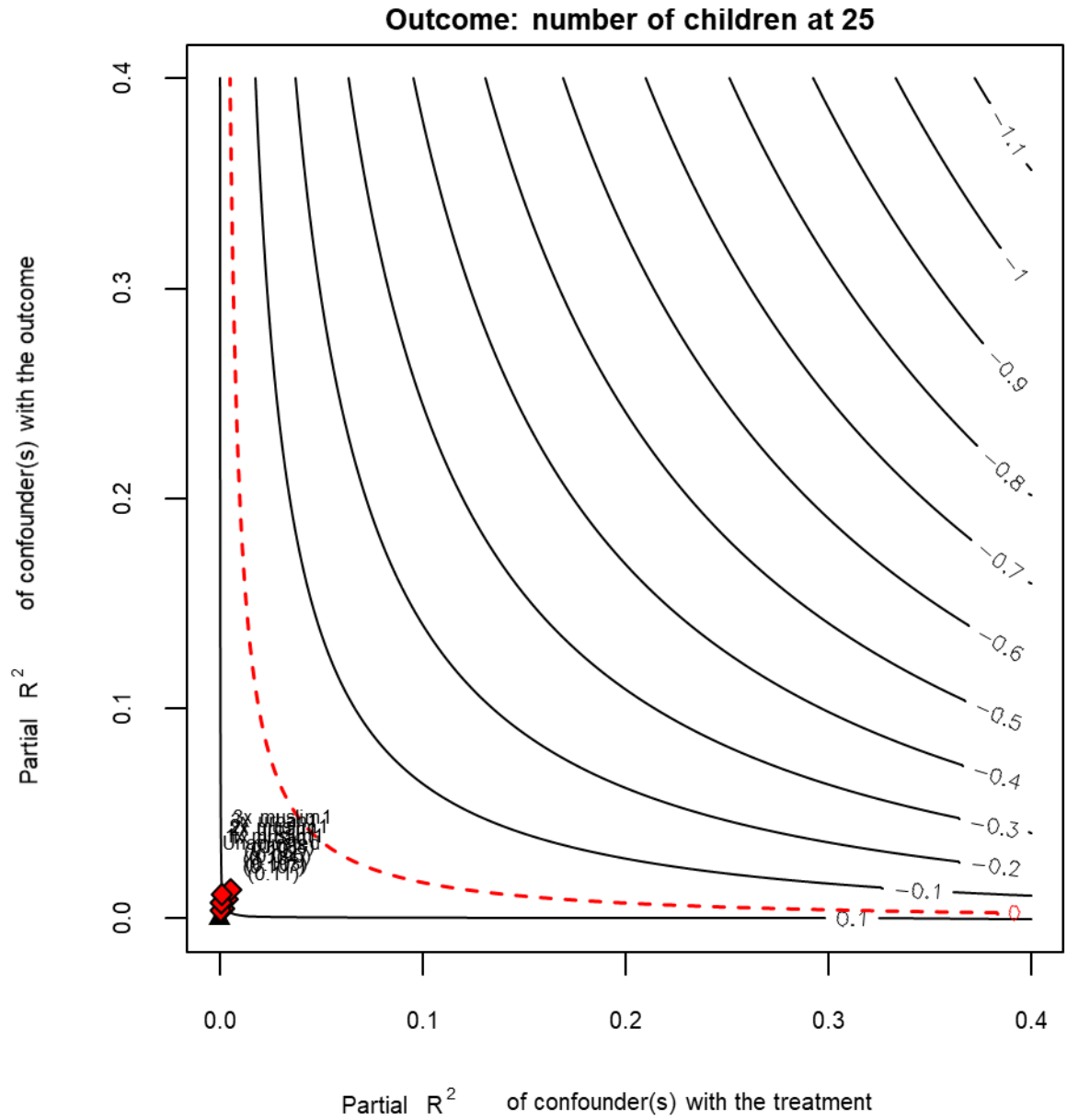


Figure S.2. Sensitivity contour plots of point estimates for the causal effect of school reform programs on the number of children at age 25, using the approach proposed by Cinelli and Hazlett (2020). Religious affiliation was used as a bench mark covariate.