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Length of secondary schooling and risk of HIV infection in Botswana: evidence from a natural experiment

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Abstract

Background—An estimated 2·3 Million individuals are newly infected with HIV each year. Existing cross-sectional and longitudinal studies have found conflicting evidence on the association between education and HIV risk, and no randomized experiment to date has identified a causal effect of education on HIV incidence.

Methods—A 1996 policy reform changed the grade structure of secondary school in Botswana and increased educational attainment. We use this reform as a 'natural experiment' to identify the causal effect of schooling on HIV infection. Data on HIV biomarkers and demographics were obtained from the 2004 and 2008 Botswana AIDS Impact Surveys, nationally-representative household surveys (N = 7018). The association between years of schooling and HIV status was described using multivariate OLS regression models. Using exposure to the policy reform as an instrumental variable, we estimated the causal effect of years of schooling on the cumulative probability that an individual contracted HIV up to his or her age at the time of the survey. The cost-effectiveness of secondary schooling as an HIV prevention intervention was assessed in comparison to other established interventions.

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Contributors

JWDN and JB conceived and designed the study. JWDN conducted statistical analysis under the guidance of JB. JWDN and JB wrote the report. GF and SV suggested improvements to the statistical analysis. GF, SM, and SV contributed important revisions to the report. All authors approved the final submitted version of the report.

Declaration of Interests

We declare that we have no competing interests.

Findings—Each additional year of secondary schooling induced by the policy change led to an absolute reduction in the cumulative risk of HIV infection of 8·1% points (p = 0·008), relative to a baseline prevalence of 25·6%. Effects were particularly large among women (11·6% points, p = 0·046). Results were robust to a wide array of sensitivity analyses. Secondary school was cost-effective as an HIV prevention intervention by standard metrics.

Interpretation—Additional years of secondary schooling had a large protective effect against HIV risk, particularly for women, in Botswana. Increasing progression through secondary school may be a cost-effective HIV prevention measure in HIV-endemic settings, in addition to yielding other societal benefits.

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Introduction

HIV continues to be a major global health challenge with an estimated 2·3 Million new infections each year.[1] Formal education, particularly of girls, has been hailed as a 'social vaccine' to reduce the spread of HIV.[2] However, there is little causal evidence for this claim.[3] Existing cross-sectional and longitudinal studies have found conflicting evidence on the association between education and HIV risk. Early national surveillance surveys found higher rates of HIV among people with more education in a number of sub-Saharan Africa countries.[4,5] However, other studies have found a protective association between higher education and HIV infection, particularly as the epidemic has matured and information on prevention strategies has become more widely available.[6–8] The effect of education is theoretically ambiguous. Education may reduce HIV risk through: increased exposure to information about HIV and prevention methods[9,10]; improved cognitive skills to make complex decisions[11]; better financial security[12–15], reducing participation in transactional sex for women[16]; ability to match with lower risk sex partners[16–19]; and increased future orientation. On the other hand, education may increase the size of one's sexual network; prolong the period of pre-marital sex[20]; and increase transactional sex among men.[21] In addition to its contribution to HIV epidemiology, this study contributes to the broader debate about whether the relationship between education and health is causal.[22–25]

The challenge in determining the causal effect of schooling on HIV infection risk is that educational attainment is closely related to factors such as socioeconomic status, psychological traits, and preferences, which are difficult to control for fully in observational studies, and which may also affect HIV risk. Several randomized trials have sought to identify the impact of schooling on HIV risk, but they have been underpowered to look at HIV incidence and have been paired with other interventions that make it difficult to attribute any effects to schooling.[26–28] This study exploits variation in educational attainment generated by a policy reform in Botswana in 1996, which changed the grade structure of secondary school nationwide in such a way that it increased average years of schooling by approximately 0.8 years. The policy change affected specific birth cohorts – i.e., those who would have entered secondary school in 1996 or later – and was unlikely to have affected HIV risk through mechanisms other than schooling itself. Using multiple survey waves to disentangle age and cohort effects, we use the resulting variation in

exposure to the reform to identify the causal effect of education on the cumulative risk of HIV infection.

Methods

Study Population and Data Source

Study Population—Botswana has among the highest rates of HIV in the world, with 25·6% of adults aged 15–49 years infected in 2008 (BAIS 2008). The study population included all male and female citizens of Botswana residing in the country in 2004 or 2008. Respondents younger than 18 years were excluded because they would not have had the opportunity to complete secondary education. Respondents born prior to 1975 were excluded because previous school reforms led to rapid changes in education for these older cohorts.[29] Immigrants to Botswana were excluded because they would not have been exposed to the schooling intervention if they migrated in adulthood.

Data Source—Data were obtained from the Botswana AIDS Impact Surveys (BAIS) II (2004) and III (2008), nationally-representative household surveys with HIV biomarker collection. For each survey, approximately 8,300 households were selected; all members aged 10–64 were eligible to be interviewed. Household and individual participation rates were, respectively, 92% and 93% for survey year 2004, and 87% and 82% for survey year 2008, yielding a total sample of N=29,606 individuals. HIV test participation rates were 61% for survey year 2004, and 67% for survey year 2008.[30,31] Data on age, sex, and years of schooling were available for 99·7% of respondents with a valid HIV test result. Figure S1 displays the participant flow diagram.

Study Design

Individuals at risk for HIV may self-select for higher (or lower) educational attainment based on unobserved characteristics (confounders). Thus, bivariate and covariate-adjusted associations between years of schooling and HIV status may not reflect a causal relationship.[6,8,10,32] To obtain causal effects, we exploited exogenous variation in educational attainment resulting from a 1996 policy reform that changed the grade structure of secondary school in Botswana. In January 1996, Botswana shifted the tenth year of education from senior secondary to junior secondary school, with the goal of increasing access to grade ten.[29] (For a description of the reform, see Appendix.) This ‘natural experiment’ provides an opportunity to estimate the causal impact of schooling on risk of HIV infection, by comparing birth cohorts exposed to the reform vs. those unexposed.

Procedures

The key exposure in our analysis was the “total years of schooling by the time of the survey”. Our outcome of interest was HIV status at the time of the survey. HIV status reflects a binary stochastic realization of an underlying probability: the cumulative probability of HIV infection up to a respondent’s age at the time of the survey. We defined an indicator - “reform cohort” – taking the value one if the respondent was born in a cohort exposed to the 1996 education policy reform and zero otherwise. Given that children are expected to start primary school at age 7, children are expected to enter junior secondary

school at age 15. Therefore, individuals born in 1981 or later would have entered junior secondary school in 1996 or later, and were thus classified as "exposed".

Statistical Analyses

As a benchmark, we assessed the naïve association between years of schooling and HIV status. We assessed the crude relationship graphically and then adjusted for covariates in descriptive multivariate OLS (linear probability) regression models.[33] We estimated several specifications, modeling years of schooling as a continuous covariate; allowing for different slopes for 0 – 9 years and 10 – 13+ years of schooling; and with separate indicators for each additional year of schooling completed. We present linear probability models (i.e. as opposed to logistic models) to facilitate comparison with the 2SLS instrumental variable models used to analyze the policy reform.[34]

Our analysis of the policy reform proceeded in three steps. First, we assessed whether birth cohorts exposed to the reform ("reform cohorts") had higher educational attainment than birth cohorts not exposed to the reform. We estimated the effect of exposure to the reform on total years of schooling completed in multivariate OLS regression models ("first stage"). We also assessed the effects of the reform on the probabilities of completing at least 7, 8, 9, 10, 11, 12, and 13+ years of schooling and show graphically how this distribution changed across birth cohorts. Second, we assessed the "intention-to-treat" (ITT) effect of being in a reform cohort on HIV status in multivariate linear probability models. Third, we estimated two-stage least squares (2SLS) regression models, using exposure to the reform as an instrument for total years of schooling while adjusting for covariates. Natural experiments that change the probability of an exposure can be analyzed like RCT's with non-compliance.[35] Under plausible assumptions, the treatment effect among so-called "compliers" is the ratio of the ITT and the difference in the probability of receiving treatment (ITT / First Stage = IV). We interpret our IV estimates as "local" to the subpopulation who "complied" with their treatment assignment – i.e. persons who increased their years of schooling because of the reform.[36]

In all models, we controlled flexibly for age with a full set of single-year age indicators, to account for the non-monotonic pattern of HIV infection across ages in Botswana and lower expected years of schooling for persons at younger ages.[37] We also included indicators for district of birth. Finally, we adjusted for a continuous linear term in year of birth, to account for continuous trends in HIV infection risk across birth cohorts. Exposure to the reform was modeled as an intercept shift for cohorts born in or after 1981. We estimated all models first for women and men separately, and then on the pooled sample. When pooling sexes, we included indicators for sex and the interactions of sex with all other covariates; however, we did not interact sex with the main exposure, so that the coefficient of interest reflects a weighted average of effects for men and women.

For our effect estimates to have a causal interpretation, four assumptions must be satisfied (Figure 1). First, the instrument (Z) must have had an effect on schooling (E); this is testable and we find large effects. Second, the instrument (Z) must be independent of unobserved confounders (U), conditional on observed covariates (X); in our application this implies that people born before and after 1981 were similar, after controlling flexibly for age, district of

birth, and a linear trend in HIV risk across birth cohorts. The availability of two survey years enables us to identify these cohort effects, while controlling flexibly for age and period effects. Our models control for period effects implicitly, by simultaneously adjusting for age and a continuous term in year of birth. To allow for potential non-linearities in underlying cohort trends, we conducted robustness checks including quadratic terms for year of birth, reducing the window of observation to a narrower set of birth cohorts, and allowing the slope of the trend across birth cohorts to differ before and after 1981. Identification comes from the fact that the policy reform led to a discontinuous change in schooling across cohorts; our identifying assumption is that no other unobserved factors led to a discontinuous change in HIV risk for precisely the same cohorts. To generate added confidence in this assumption, we conduct a placebo test, assessing the impact of the reform on persons with less than nine years of schooling - a population that was not affected by the reform. We also estimated difference-indifferences models, exploiting the fact that the policy reform had a larger impact in some districts than others, based on the share of respondents with exactly nine years of schooling pre-reform.

Third, we assume that exposure to the policy reform (Z) affected HIV risk (Y) only through changes in schooling (E) ("exclusion restriction"); this is highly plausible given that the reform was a supply-side intervention that would not have specifically affected the reform cohorts except through their increased access to grade ten. Fourth, to interpret our results as complier causal effects (a.k.a. local average treatment effects), we assume monotonicity; i.e. that exposure to the reform (Z) only caused individuals to obtain more schooling or to have no change in schooling, and did not lead some individuals to obtain less schooling. Violations of this assumption are possible but unlikely (e.g. a person with a very strong preference for small class size might have continued to grade ten pre-reform but dropped out after grade nine post-reform).[36,38]

In addition to the robustness checks described above, we conducted a range of sensitivity analyses: including sampling weights, using alternate functional forms for age, modeling the outcome using a Probit link function, and imputing HIV status for respondents who did not consent to biomarker collection using two different methods.

This study was reviewed by the Harvard School of Public Health Institutional Review Board and considered exempt from full review as it was based on an anonymized dataset.

Cost-Effectiveness

To compare the cost-effectiveness of secondary schooling vis-à-vis other proven HIV prevention interventions, we calculated the costs per HIV infection averted and per disability-adjusted life year (DALY) averted using estimates of the per-pupil costs of secondary education published by the UNESCO Institute for Statistics. Details of cost-effectiveness calculations are presented in the Appendix.

Role of Funding Source

The sponsors of the study had no role in study design, data collection, analysis, interpretation of data, writing of the report, or in the decision to submit for publication.

JWDN and JB had full access to all of the data in the study and take responsibility for the decision to submit for publication.

Results

Descriptive Tables and Figures

The 2004 and 2008 BAIS surveys included 3,965 women and 3,053 men with valid HIV biomarkers, for a total of 7,018 respondents (Figure S1). Table 1 displays summary statistics.

Descriptive Analysis: Association between Education and HIV Infection Risk

The crude association between HIV infection risk and schooling was non-monotonic, peaking for persons completing 8 – 9 years of education and declining sharply thereafter (Figure 2). The strong association between schooling and HIV risk at higher grade levels persisted in multivariate regression (Table S1). Each additional year of schooling above nine years was associated with a −3·6% point lower risk of HIV infection (se = 0·4% points). In contrast, there was no association between schooling and HIV risk in lower grades (coefficient = 0·3 % points; se = 0·2% points). Though suggestive, these associations – like those previously reported in the literature – may be confounded by unobserved characteristics. In what follows, we use a natural experiment to estimate causal effects.

Effects of the 1996 Grade Reform on Educational Attainment

The reform increased average years of schooling completed by 0·79 years ($p < 0·0001$), with the effect largely driven by gains in grade-ten completion (Tables 2 and S2). Figure 3 shows the proportion of respondents who completed at least 7, 8, 9, 10, 11, or 12 years of schooling and how this distribution changed across birth cohorts. The fraction of students completing at least 7, 8, or 9 years of schooling rose gradually and continuously across birth cohorts. However, the share of students with at least ten years of schooling was much higher for cohorts born in 1981 or later. Due to grade repetition and/or late entry into school, some respondents born in 1979 and 1980 likely were also affected by the reform. Modest increases in completion of years 11 and 12 were also observed for reform cohorts (Table S2).

The Causal Effect of Education on HIV Infection Risk

Table 2 presents “intention-to-treat” results, in which HIV status was regressed directly on the instrument and covariates. Women who were exposed to the reform were 7% points less likely to be HIV positive ($p = 0·017$); men were 5% points less likely to be HIV positive ($p = 0·052$). The pooled coefficient was 6% points ($p = 0·002$). Observed HIV prevalence closely matched the model predictions (Figure S2). In 2SLS (instrumental variables) models, each additional year of schooling induced by the reform reduced infection risk by 8% points ($p = 0·008$) and 12% points for women ($p = 0·046$). We were not able to reject the hypothesis that schooling had zero effect on HIV infection for men ($p = 0·085$) nor that the effect for men differed from the effect for women ($p = 0·556$).

Sensitivity Analyses and Placebo Tests

Tables 3 and S5 – S7 display the results of robustness checks for the 2SLS results. In general, our results were not sensitive to sampling weights, imputation for HIV non-consent, different specifications of the outcome, alternate specifications of the continuous trend across birth cohorts, nor to different modeling strategies for age (Table 3, models 1 – 5; see Tables S4 – S7 for sex-specific and ITT results). Our difference-in-differences analysis returned similar effect estimates as our main results, though standard errors were larger (Table 3, model 6). In a placebo test, the effect of the reform on HIV risk was driven entirely by respondents with at least nine years of schooling, with no effect among respondents with less than nine years (Table 3, models 7 and 8).

Cost-Effectiveness of Secondary Education as an HIV Prevention Strategy

The annual per-pupil cost of secondary education was $2,248 in Botswana, using the average of 2005 and 2007 UNESCO estimates.[39] Since Batswana who stayed in school for an additional year had an 8·1% point lower risk of HIV infection, the cost per HIV infection averted was $27,753 USD. By standard cost-effectiveness benchmarks, an intervention is "very cost-effective" if it costs less than 1 × per capita GDP for each DALY averted. Based on calculations presented in the Appendix, we estimate that an HIV infection at age 20 would lead to 16·3 lifetime DALY's for someone who did not initiate antiretroviral therapy (ART); and 3·5 lifetime DALY's for someone who initiated ART, with a lifetime treatment cost of $12,400. All costs and DALY's were discounted at 3%. These calculations imply cost-effectiveness ratios (CER) of $4,387/DALY with ART and $1,703/DALY without ART; each of these CER's is less than Botswana's $5,178 per capita GDP (2009), implying that secondary school is very cost-effective as an HIV prevention intervention. Table 4 compares the cost-effectiveness of secondary school with other proven HIV prevention interventions in terms of HIV infections averted. Secondary schooling is more expensive than circumcision and treatment as prevention, but of similar cost-effectiveness to pre-exposure prophylaxis.[40–43] Importantly, unlike these other interventions, secondary schooling has large benefits beyond the reduction of HIV transmission – benefits that have been excluded from the above calculations.

Discussion

Using an education policy reform as a natural experiment, we find that secondary schooling has a large protective effect against risk of HIV infection in Botswana. Effects are particularly large among women and were consistent across a wide array of robustness checks. Our IV estimates are somewhat larger, but generally consistent with the strong negative associations we found between secondary schooling and HIV risk in multivariate OLS regression. We interpret our IV estimates as "causal" because they are not vulnerable to the types of unobserved factors – e.g. psychological traits, unmeasured socioeconomic status – that may confound previous studies of the association between education and HIV.

The effects of schooling on HIV risk are likely heterogeneous, and our effect estimates are "local" in several important ways. First, our estimates are local to the specific grades affected by the policy change (grades 10 – 12); these grades may be a "critical exposure

period"[44] in determining lifetime HIV risk since this is a period when sexual behavior patterns and labor market opportunities are formed; effects of schooling may be qualitatively different in primary school, and indeed in our descriptive analysis, we found the association between schooling and HIV risk to be non-monotonic. Second, the causal effects that we estimate are "local" to the subpopulation of compliers – i.e. those induced to increase schooling because of the reform. This subpopulation consists of persons who, in the absence of the reform, would have dropped out after ninth grade – a group likely to be at particularly high risk for HIV. Third, the results are local to an epidemiological context in which HIV is hyper-endemic with very high incidence for people ages 20 – 29 years; effects of this magnitude might not be observed in lower prevalence settings. Fourth, the effects are local to the years of risk exposure under study – the 1990's through early 2000's. Previous studies have reported changing associations between education and HIV risk over time, and we caution against generalizing to earlier cohorts who formed sexual behavior patterns before HIV emerged as an epidemic in Botswana;[20,45] however, we do suspect that our effect estimates are likely informative of current and future benefits of education in a society where HIV is endemic. In addition to treatment effect heterogeneity, it is also possible that the 2SLS results are larger than the OLS result because unobserved factors, such as personal charisma, may be positively associated with both educational attainment and HIV risk, thereby leading to downward bias in the OLS coefficient.

Our study has some limitations. First, consent rates were imperfect, and migration or mortality could have influenced the composition of the study sample. However, neither consent rates nor birth cohort sizes varied systematically with exposure to the reform, and our results were robust to imputation. Second, it is possible that some respondents acquired HIV prior to the age when they would have entered grade ten. Infection rates are very low prior to grade ten. More importantly, since our analysis was conducted on a risk difference scale, our approach is robust to the existence of prevalent HIV by grade ten, so long as prevalence was smooth across birth cohorts. Third, we only observe people through age 32 years. We cannot know whether we are measuring HIV infections truly averted or delayed. However, this is a common limitation of prevention studies, and our analysis of cumulative incidence captures much longer follow-up than most RCT's, which observe incidence over a shorter, e.g. 3yr[46], horizon.

Fourth, as discussed above, our analysis relies on the assumption that conditional on age, period, district of birth, and a smooth trend in birth cohort, there were no other cohort-specific effects that would have led to a discontinuous change in HIV risk coinciding with the reform. There are many reasons why HIV risk might change across birth cohorts but the likely candidates – infection rates among sexual partners, access to HIV treatment, changes in prevention programming – are phenomena that are either gradual over time (changes in the epidemic context) and/or affect people of many different ages (e.g. a national prevention campaign or the introduction of ART): in both cases, these phenomena would result in gradual changes in HIV infection across birth cohorts, which we control for. For example, the scale-up of ART may have reduced infectiousness among respondents' sex partners; however, we expect both selection of sex partners and take-up of ART (among those sex partners) to be smooth across birth cohorts. One example of a potential confounder would be an HIV prevention program implemented in a specific year, targeted to a specific grade in

school, and thus only affecting specific birth cohorts. However, Botswana's school-based HIV curriculum was not in place in 1996.[47] To generate confidence that our results are not confounded by other policy changes, we estimated difference-in-differences models and a placebo check, exploiting the fact that the reform was expected to affect primarily people with at least nine years of schooling. Indeed, the reform had no effect on HIV risk for people with less than nine years of schooling; and it had a larger effect for people born in districts where a higher share of the population had exactly nine years of schooling pre-reform. Finally, as with all infectious diseases, we expect spillover effects on incidence beyond the individuals directly affected by the reform. Given that people have sexual relationships across birth cohorts, these spillovers would be expected to be smooth across birth cohorts and would not bias our estimates. However, by excluding these benefits, we may be underestimating the cost-effectiveness of secondary schooling.

Expanding access to secondary school had a large protective effect against HIV infection in Botswana. Our findings confirm what has been long suspected: that secondary schooling is an important structural determinant of HIV infection and this relationship is causal. Further, our estimates indicate that secondary schooling is very cost effective as an HIV prevention intervention, in addition to its other societal benefits. Investing in expanded access to secondary schooling would be an effective HIV preventive measure and should be considered as part of "combination HIV prevention" strategies in other settings with large, generalized HIV epidemics.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Appendix

Study Population

Age in the study population ranged between 18 and 32 years old. Mean age was 22•7 (SD 3•1) for women and 22•6 (SD 3•2) for men in the BAIS II survey, and 24•9 (SD 4•2) for

women and 24•7 (SD 4•3) for men in the BAIS III survey (Table 1). In the full sample (N= 29,606), the median age of respondents who were students at the time of the surveys, and completed exactly ten years of schooling, was 16·8 years old; whereas in those who completed either exactly eleven or twelve years of schooling it was 18·3 years old. In the 1980's, the modal age in year of school entry was 7 years old, and secondary school-going age range 14 – 18 years.

Description of the 1996 Education Reform in Botswana

Botswana's K12 public education system is divided into primary, junior secondary and senior secondary education. In 1994, the National Commission on Education (NCE) brought up several problems associated with the existing '7+2+3' grade structure, in which two years of junior and three years of senior secondary school followed seven years of primary education. In particular, two years of junior secondary education was insufficient to prepare students for work or further training and did not offer sufficient time for students to adjust from primary to secondary school. The NCE recommended a switch to a '7+3+2' structure with primary and junior secondary school forming 'Ten Years of Basic Education' (rather than 'Nine Years of Basic Education'). Botswana's public education system is strongly centralized and the policy was implemented rapidly and universally; it also serves the vast majority of the population with less than 1% of secondary school students attending private schools (Government of Botswana 2013).

The reform lowered barriers to grade ten. Since there are many more junior secondary schools than senior secondary schools, the reform increased the number of grade ten seats and reduced student travel times. It established continuity with grade nine. It also implied that completing grade ten was required to graduate from junior secondary and obtain a Junior Certificate. Finally, if education affects preferences for later schooling (e.g. some students may discover that they like school), then increasing grade ten completion might have increased progression through later secondary and even tertiary education. A previous study has shown that the reform led to an increase in total years of schooling – with a large increase in grade ten completion, an increase in labor force participation, and a reduction in unemployment among women (Borkum 2009).

Description of Cost-Effectiveness Calculations

We used a simple model to estimate the number of DALY's resulting from an HIV infection under two scenarios: the person initiates ART when eligible; the person does not ART. We based all estimates on an infection occurring at age 20. Under the "no ART scenario," we assumed that HIV infection was followed by 7 years of asymptomatic HIV (disability weight 0), one year of symptomatic HIV (disability weight 0·221), two years of AIDS (disability weight 0·547), and then death, occurring ten years after initial infection. Under the "ART scenario," we assumed 7 years of asymptomatic HIV, one year of symptomatic HIV, followed by 27 years of ART (disability weight 0·053). Disability weights were obtained from the 2010 Global Burden of Disease (Salomon et al. 2012). To calculate DALY's, we need an estimate of the number of life years that a person would live if he or she did not contract HIV at age 20; we used WHO life table estimates for Botswana for

2009, indicating a life expectancy of 45 years at age 20 (WHO 2012). Survival on ART was estimated as 75% of "normal" life expectancy at age of ART initiation (36 years at age 30), based on published estimates from neighboring South Africa (Johnson et al. 2013). We used per patient per year cost estimates of $202 for pre-ART and $880 for ART published in Menzies et al. (2011).

Based on these inputs, and discounting both DALY's and costs at 3%, we estimated that an HIV infection at age 20 would lead to: 16·3 DALY's for a person who did not initiate ART; and 3·5 DALY's for a person who initiated ART, with a present-discounted lifetime treatment cost of $12,400. These estimates of lifetime treatment costs are of similar magnitude to an independent estimate of the present-discounted fiscal costs of an HIV infection in Botswana, projected at twice GDP per capita (Lule & Haacker, 2012).

For the "no ART scenario", we divided the $27,753 per infection averted by the 16·3 DALYs per HIV infection in the absence of ART, yielding a cost-effectiveness ratio (CER) of $1,703/DALY. For the "ART scenario", we divided the net cost of secondary school ($27,753 - $12,400 = $15,353) by the 3·5 DALYs per HIV infection with ART initiation when eligible, yielding a CER of $4,387/DALY. Each of these CERs is less than Botswana's $5,178 per capita GDP, implying that secondary school is very cost-effective as an HIV prevention intervention.

Mechanisms

Although a detailed analysis of mechanisms is beyond the scope of this paper, there are several potential explanations for the protective effect of secondary schooling on HIV risk. Completion of later grades may reduce HIV risk through improved cognitive skills to reduce exposure to HIV; changing attitudes and preferences; less risky sexual networks; and/or by giving young adults something to do with their time. One key mechanism could be increased economic independence of women due to higher labor force participation, a consequence of the reform reported elsewhere (Borkum 2009). Although the reform cohorts in our study completed secondary school in the 1990's and early 2000's, before Botswana launched a formal HIV curriculum in the schools, education could lead to greater access to information, e.g. through media sources (Agüero & Bharadwaj 2014).

Robustness Checks

Attrition and Consent Rates

One concern is that results could be biased by differential consent rates by birth cohort or by selection bias from mortality risk associated with being born in or after 1981. Increased education may have improved access to ART, which became available in 2003 in Botswana; however, this would lead to a higher HIV prevalence among those with more education, and hence a bias against the direction of our results. Figure S3 shows the proportion of respondents without an HIV test and the size of the surviving birth cohorts in the study sample. There was no evidence of a post-1980 cohort effect in either of these variables that might bias our estimates. We also assessed whether differential non-consent might have biased our results, by imputing HIV status for respondents with missing HIV biomarker data

among Batswana. Results using imputed HIV estimates were similar to our main model. Further, we show in a regression context that consent rates do not change much with exposure to the reform. To do so, we ran the main intention-to-treat regression model, but using "missing HIV data" as the outcome. Table S3 in the Appendix displays ITT results for consent rates by sex and in the pooled sample. There appeared to be a relatively small effect of the reform, but this did not reach conventional benchmarks for significance, and the small size of the effect is unlikely to explain the large effects of schooling on HIV risk we observe. Lastly, we note that the datasets we used do not contain information on interviewer identity, which would have allowed us to use Heckman-type selection models to correct for selection on unobserved variables (Bärnighausen et al. 2011).

Alternative Explanations

In our main results, we controlled flexibly for age using single-year indicators, included indicators for district of birth and survey year, and included a linear trend for year of birth. Our identifying assumption is that there are no other cohort-specific exposures that influence HIV risk for persons born in or after 1981, conditional on long run trends in HIV risk across birth cohorts. This assumption could be violated if long run trends are non-linear, or if some other intervention affected specific birth cohorts (or equivalent, targeted specific age groups in specific years). First, to assess the robustness of our results to different specifications of age, we replaced our single-year age dummies with a quadratic function of age, and with a cubic spline in age. Second, to assess the robustness of our results to the presence of non-linearities in long-run cohort trends in HIV risk, we controlled for a quadratic in year of birth, quadratic trend in year of birth interacted with survey wave, a Probit model for the outcome, and separate linear functions for year of birth on either side of the 1981 cut-off for exposure to the reform, in addition to the linear term included in the main analysis. Third, we also estimated our main model for a narrower window of birth cohorts – 1981 +/− 4, 5 years; and when including all earlier cohorts (1903 < YOB < 1990). The assumption that underlying trends are approximately linear is more plausible the narrower the window of cohorts included. Fourth, to rule out the possibility that other national policy changes might have affected HIV risk for the same cohorts, we used an alternative identification strategy. The education policy reform would be expected to have the greatest impact on years of schooling in those districts where a large fraction of students completed exactly nine years of schooling in the pre-reform period. Figure S4 displays educational attainment by districts with either a high or low proportion of people with exactly nine years of schooling. We created an indicator for whether a subject's district of birth had high vs. low grade-nine completion, and used as our instrument the interaction of this variable with the indicator for reform cohort, while controlling for the main effects of each variable. Fifth, we conducted a placebo test. Dividing the sample into "people with at least 9 years of schooling" and "people with less than 9 years of schooling", we estimated the effect of the policy change among those with at least 9 years – i.e. those people whose schooling increased due to the reform; and those with less than nine years of schooling, who experienced no increase in schooling (Table S4).

Weighting

In analytical inference, the use of sample weights is subject to controversy. We added sample weights to our main model as an additional robustness check. In all descriptive results, we used sample weights.

Exclusion of the 1980 birth cohort

It is best practice in analysis of natural experiments to use the legal structure of the policy change as an instrument for the actual changes in schooling observed, since it is the legal changes that are exogenous to individuals' choices about schooling. (Revealed choices about educational attainment, in contrast, may be correlated with other determinants of HIV risk.) The first cohort that would have been affected *de jure* was the 1981 birth cohort. However, we also observed elevated levels of grade 10 completion in the 1980 birth cohort, which may have included individuals who entered school late or repeated grades (Figure 3, main text). We assessed the robustness of our results excluding respondents born in 1980, as this cohort was partially affected by the reform.

Results of Robustness Checks

Tables S5-S7 show the robustness of our results to additional controls in the pooled sample and by sex, including a quadratic term in age, cubic spline in age, quadratic term in year of birth, year of birth and survey year interactions, using sample weights, year of birth and reform indicator interactions, narrower birth cohort windows, imputed HIV estimates, a 2SLS difference-in-difference estimator using an indicator for whether a subject's district of birth had high vs. low grade-nine completion, including earlier birth cohorts, placebo test, and using a Probit model. In the pooled sample, using either a quadratic term in age, cubic spline in age, quadratic term in year of birth, or a quadratic in year of birth with survey year interactions, Batswana who stayed in school for an additional year had a 7% point lower risk of HIV infection ($p = 0{\cdot}048$, $p = 0{\cdot}041$, $p = 0{\cdot}014$, and $p = 0{\cdot}010$, respectively), using year of birth and survey year interactions or sample weights they had a 8% point lower risk of HIV infection ($p = 0{\cdot}008$ and $p = 0{\cdot}025$, respectively). Using imputed HIV estimates, with or without the use of additional covariates, such as age of sexual debut, to impute HIV estimates, they had a 9 – 11% point lower risk of HIV infection ($p = 0{\cdot}045$ and $p = 0{\cdot}036$, respectively). Using a narrower birth cohort of 1981 +/− 4, 5 years, schooling appeared similarly protective but did not reach conventional benchmarks for statistical significance. Using a 2SLS difference-in-difference estimator, the point estimates were similar to our main results, although the standard errors were larger, as there was less variation in the exposure. In women, however, using a 2SLS difference-in-difference estimator, they had a 13% point lower risk of HIV infection ($p = 0{\cdot}048$). Using a Probit model, results were 3% points lower than when using a 2SLS model ($p < 0{\cdot}001$). In the placebo test, we find that the effect of the policy change was observed only among those with at least 9 years. We found no effect of the policy change on people with less than nine years of schooling, who experienced no increase in schooling. Excluding the 1980 birth cohort, schooling remained protective against HIV infection risk (−0·052% points, $p = 0.035$, pooled sample).

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Panel: Research in Context

Systematic Review

Two previously published systematic reviews synthetized evidence from over 36 cross-sectional, cohort, and case-control studies.[3,8] The authors of these reviews concluded that over time, HIV risk appears to have shifted towards higher prevalence among persons with less schooling in sub-Saharan Africa. This would reverse previous patterns: studies conducted before 1996 found no association between schooling and HIV risk or a higher risk among the more educated. All of the studies reviewed were observational and none used exogenous variation from a natural experiment to identify the causal effect of schooling. We searched PubMed for reports published in English between 2006 and 2014 using the search terms “education” and “HIV risk”. We identified three studies assessing the causal effect of education on HIV infection: one randomized trial, which was underpowered,[16] and two natural experiments, of which the first one was underpowered and the second one did not control for age and may be biased.[9,48]

Interpretation

This study is among the first to estimate the causal effect of education on HIV infection by using an education policy reform as a ‘natural experiment’. We find that secondary schooling has a large protective effect against risk of HIV infection in Botswana, particularly among women. Increasing progression through secondary school may be a cost-effective HIV prevention measure in HIV-endemic settings, in addition to yielding other societal benefits.

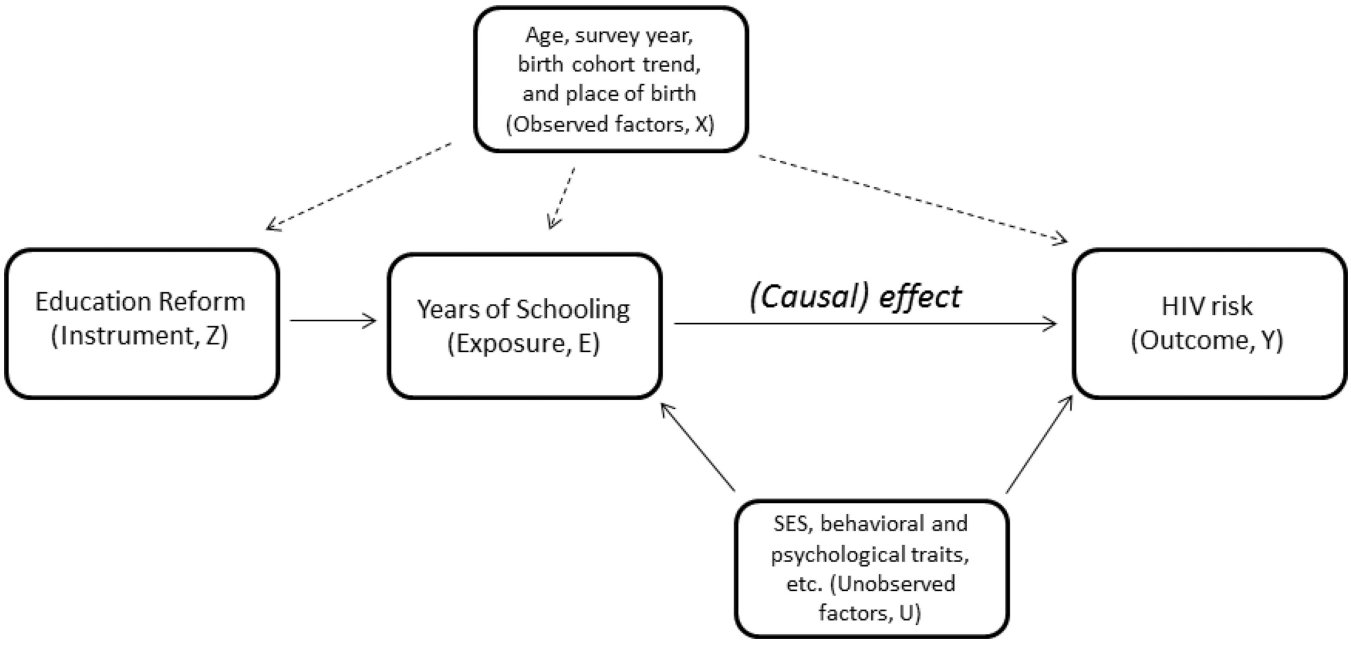

Figure 1. Causal Diagram

Directed acyclic graph showing the instrumental variable assumptions underpinning our study. Conditional on X, Z is a valid instrument if Z causally affects E, Z is uncorrelated with U, and Z affects Y only through E. Under the assumption that Z only affects E in one direction, 2SLS identifies a local average treatment effect (LATE).

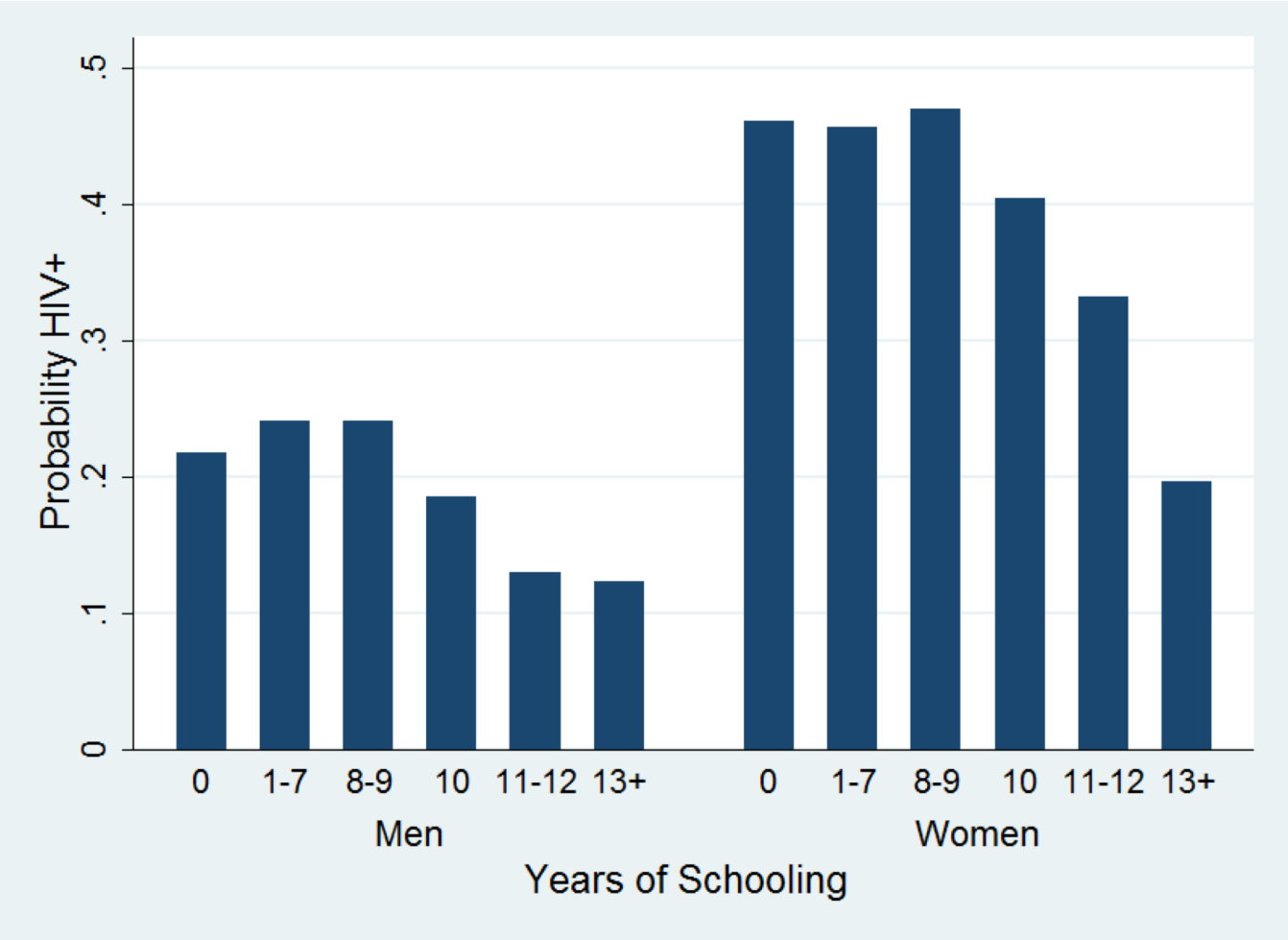

Figure 2. HIV Prevalence by Years of Schooling in Botswana

HIV prevalence by years of schooling completed and gender. Sample includes survey respondents who were citizens of Botswana, at least 24 years old at the time of the surveys, born in or after 1975, and had a valid HIV test result. Source: Botswana AIDS Impact Survey II (2004) and III (2008).

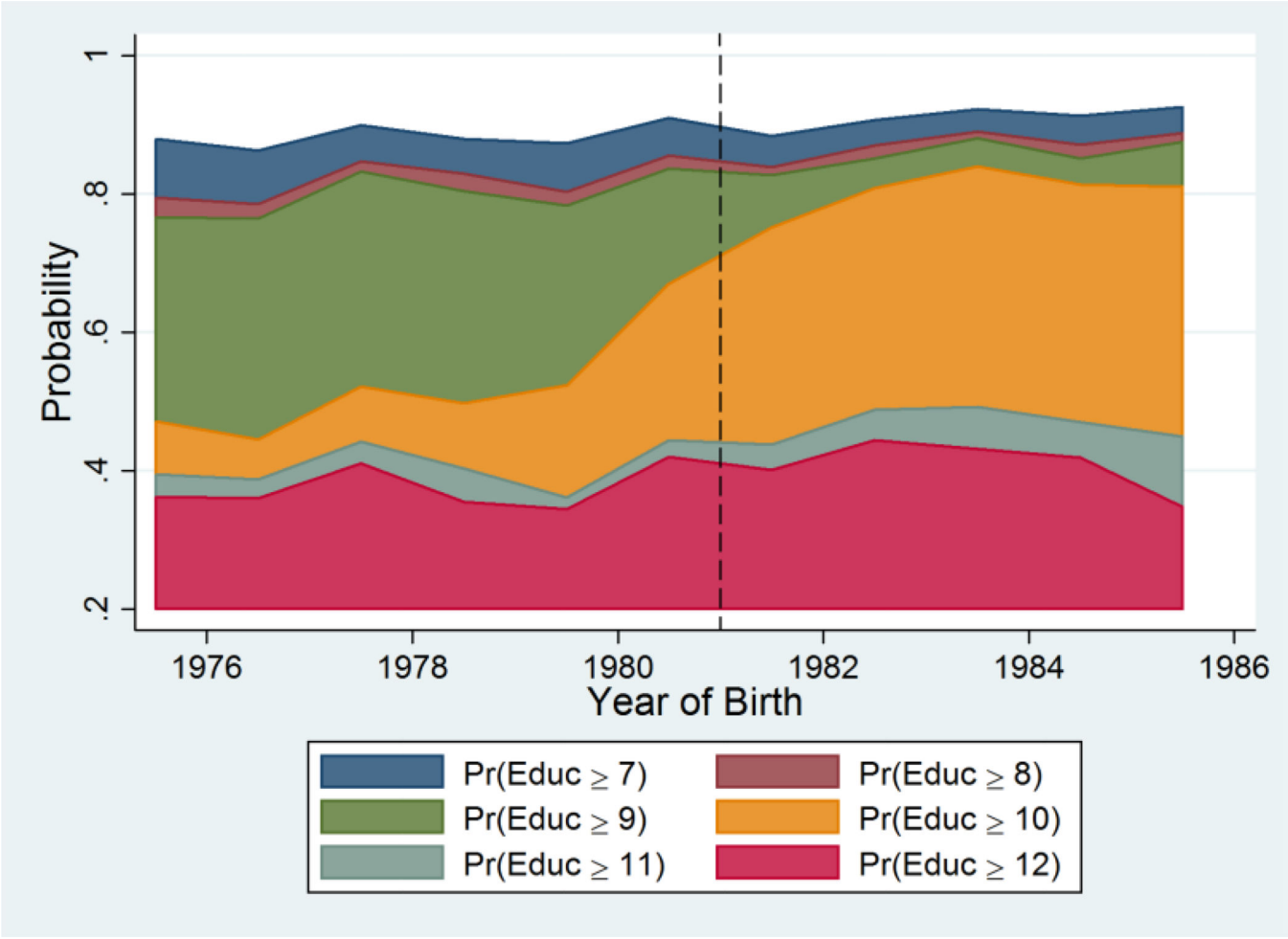

Figure 3. Educational Attainment by Birth Cohort in Botswana

Pr(Educ ≥ X) is the probability that the respondent has attained at least X years of schooling. Sample includes survey respondents who were citizens of Botswana, at least 18 years old at the time of the surveys, born between 1975 and 1985, and had a valid HIV test result. Survey weights used as provided. Source: Botswana AIDS Impact Survey II (2004) and III (2008).

Table 1

Summary Statistics.

Variables	Percent / Mean (SD)			
Survey Year	**BAIS II (2004)**		**BAIS III (2008)**	
Subsample	**Female**	**Male**	**Female**	**Male**
HIV Positive (%)	28·3	11·1	27·3	12·4
Age	22·7 (3·1)	22·6 (3·2)	24·9 (4·2)	24·7 (4·3)
Years of Schooling	10·0 (3·0)	9·7 (4·0)	10·5 (3·2)	10·3 (3·8)
Has At Least Ten Years of Schooling (%)	62·4	65·2	72·6	73·0
Ever Had Sex (%)	88·2	77·9	92·7	83·1
Age at First Intercourse	18·0 (2·0)	17·8 (2·5)	18·2 (2·5)	18·5 (3·0)
Ever Married (%)	4·93	1·00	7·10	2·60
Literacy (%)	83·0	80·0	91·1	86·0
Total N with HIV Result	1,760	1,354	2,205	1,699

Sample includes survey respondents who were citizens of Botswana, at least 18 years old at the time of the surveys, born in or after 1975, and had a valid HIV test result. Total N with Age at First Intercourse was 1,520 for women and 1,012 for men in BAIS II (2004), and 1,987 for women and 1,348 for men in BAIS III (2008). Sample weights used as provided. Source: Botswana AIDS Impact Survey II (2004) (N: 15,479) and III (2008) (N: 14,127).

Table 2

Natural Experiment: First Stage, Intention-To-Treat and 2SLS Results.

	(1)	(2)	(3)
Model	**First Stage**	**Intention-to-treat**	**2SLS (IV)**
Dependent Variable	**Years of Schooling**	**HIV-Positive**	**HIV-Positive**
Reported Coefficient	**Reform Indicator**	**Reform Indicator**	**Years of Schooling**
Female	0·635***	−0·074**	−0·116**
	(0·223)	(0·031)	(0·058)
Observations	3,965	3,965	3,965
R-squared	0·034	0·095	-
Probability HIV-positive, Pre-Reform	-	0·323	0·286
Male	1·005***	−0·050*	−0·050*
	(0·322)	(0·026)	(0·029)
Observations	3,053	3,053	3,053
R-squared	0·033	0·070	-
Probability HIV-positive, Pre-Reform	-	0·168	0·164
Both Sexes	0·792***	−0·064***	−0·081***
	(0·188)	(0·021)	(0·031)
Observations	7,018	7,018	7,018
R-squared	0·036	0·123	-
Probability HIV-positive, Pre-Reform	-	0·255	0·238

Regressions 1 to 2 are OLS models. Regression 3 is a 2SLS model, in which exposure to the reform was used as an instrument for years of schooling. All models included the following controls: single-year age indicators, a linear term for year of birth, an indicator for survey wave and indicators for district of birth. Regressions for the subsample with both sexes additionally control for age*sex, district of birth*sex, year of birth*sex and survey wave*sex interactions. The instrument was defined as = 1 if YOB > 1980. Standard errors in parentheses.

*** p<0·01,

** p<0·05,

* p<0·1.

Sample includes survey respondents who were citizens of Botswana, at least 18 years old at the time of the surveys, born in or after 1975, and had a valid HIV test result. No weights were used. Source: Botswana AIDS Impact Survey II (2004) and III (2008).

Table 3

Sensitivity Analyses.

Dependent Variable: HIV Status	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**
Robustness Check	**2SLS, sampling weights**	**2SLS, HIV status imputed**	**IVProbit, marginal effects**	**2SLS, quadratic in YOB**	**2SLS, slope change in YOB**	**2SLS, diff-in-diff**	**ITT, educ<9yrs (placebo)**	**ITT, educ≥9yrs (anti-placebo)**
Reported Coefficient	**Schooling**	**Schooling**	**Schooling**	**Schooling**	**Schooling**	**Schooling**	**Reform**	**Reform**
Both Sexes	−0·078** (0·035)	−0·091** (0·044)	−0·052*** (0·014)	−0·073** (0·030)	−0·079*** (0·030)	−0·147 (0·231)	−0·019 (0·059)	−0·072*** (0·023)
Observations	7,018	8,281	7,018	7,018	7,018	7,018	1,175	5,843
F-Statistic	13·4	n/a	n/a	17·9	18·5	0·6	n/a	n/a

Models 1, 2, 4, 5, and 6 are 2SLS models. Model 3 is a Probit model using Stata's *ivprobit* command. Models 7 and 8 are OLS models (ITT). In all 2SLS models, exposure to the reform was used as an instrument for years of schooling. Model 2 uses two additional covariates, Age at First Intercourse and Ever Married, to impute HIV status. The instrument was defined as = 1 if YOB > 1980. Standard errors in parentheses.

*** p<0·01,

** p<0·05,

* p<0·1.

Sample includes survey respondents who were citizens of Botswana, at least 18 years old at the time of the surveys, born in or after 1975, and had a valid HIV test result. Source: Botswana AIDS Impact Survey II (2004) and III (2008).

Table 4

Cost-Effectiveness Ratio of Secondary School and Known HIV Prevention Interventions.

Intervention	**Medical Male Circumcision**	**Treatment as Prevention (CD4 ≥350/μL)**	**Pre-Exposure Prophylaxis**	**Antiretroviral Treatment (CD4 <350/μL)**	**Secondary School**
Cost-effectivness Ratio ($ / infection averted)	551; 1,096	8,375	12,500 – 20,000; 6000 – 66,000	6,790	27,753
Study (year)	Kahn et al. (2006), Barnighausen et al. (2012)	Barnighausen et al. (2012)	Pretorius et al. (2010), Hallett et al. (2011)	Barnighausen et al. (2012)	Authors (2014)

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