Risk Compensation and HIV Therapy: A Field Experiment in South Africa

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ABSTRACT  Risk compensation—the phenomenon positing that people adjust their risky behaviours in response to changes in perceived risks—could have the adverse effect of worsening health outcomes. Consequently, understanding potential behavioural responses is critical for designing effective public policies. This study examines the relationship between improved human immunodeficiency virus (HIV) therapy and subsequent risky sexual behaviour. Using a field experiment in South Africa, I estimate the causal effects of improved HIV therapy adherence on subsequent risky sexual behaviour among HIV-positive patients. I find that access to HIV therapy induces a substantial increase in the demand for unsafe sex.

KEYWORDS: Field experiment; risk compensation; ex-post moral hazard; health; HIV; Sub-Saharan Africa

JEL CLASSIFICATION CODES: D81; D84; H40; I12; I18; J13; K32; O15

1. Introduction

Consumer demand models based on the seminal work by Peltzman (1975) posit that when new health technologies reduce the risk associated with specific individual behaviours, consumers may respond by increasing their demand for these behaviours. For example, in the context of car seatbelts, Peltzman (1975) argues that safety regulations might cause unintended harm because drivers feel safer and potentially take higher risks that could affect others. This behavioural response to an invention applies to all new health technologies (Keeler, 1994). When individuals misperceive the protective benefits of innovation or respond with an offsetting behavioural response, the net effect could be worse health outcomes than before the innovation was applied.

In this paper, I investigate the behavioural response to human immunodeficiency virus (HIV) therapy in terms of its effects on subsequent risky sexual behaviour among HIV-positive patients in South Africa. I examine the impact of improved therapy on two outcomes: self-reported number of sexual partners and the likelihood to use a condom conditional on a sexual encounter. HIV/acquired immunodeficiency syndrome (AIDS) is the leading cause of death in...
Sub-Saharan Africa. In this context, governments spend billions of dollars providing and expanding HIV therapy and implementing comprehensive prevention policies (UNAIDS, 2013). Examining the behavioural response to HIV therapy in South Africa may be especially important given the high prevalence of the disease (Young, 2005) and the widespread availability of HIV therapy there in the last fifteen years. Despite a national programme treating almost 80 per cent of HIV-positive adults (SANAC, 2016) and a preventative mother-to-child transmission programme coverage rate of more than 95 per cent in 2017 (UNAIDS, 2018), South Africa has an HIV/AIDS prevalence rate of 18.8 per cent in adults aged 15–49. UNAIDS estimates that, as of 2017, 7.2 million people in the country live with HIV (UNAIDS, 2019). Therefore, examining compensatory responses in South Africa is especially appropriate since it is a country with a high disease toll and an extensive national HIV therapy programme.

Using data from an experimental intervention in South Africa’s Free State province, this study tracks 648 individuals with access to HIV therapy. Upon enrolment in the study, participants are randomised into one of three treatment groups: HIV therapy only (Treatment Group A), HIV therapy and biweekly visits from an encouragement supporter (Treatment Group B), and HIV therapy, biweekly visits from an encouragement supporter and nutritional supplementation (Treatment Group C). Following Angrist, Imbens, and Rubin (1996), I use assignments to Groups B and C as instrumental variables (IVs) for take-up of HIV therapy and estimate the causal effect of therapy on unsafe sexual activity.

I use CD4 blood count to proxy the adherence to the drug regimen of the study participants. A CD4 count measures the number of CD4 T lymphocytes (CD4 cells) in a blood sample. A glycoprotein, CD4, is located on the surface of immune cells (e.g. T helper cells, monocytes, macrophages, and dendritic cells), and HIV infection leads to a progressive reduction in the number of T cells expressing CD4. While not a direct HIV test, the CD4 count can help assess the immune system and a patient’s compliance with the therapy regimen. It is the most critical laboratory indicator of how well a patient’s immune system works and is the strongest predictor of HIV progression.

All three treatment groups comprise HIV-positive individuals eligible for HIV therapy. Therefore, I exploit the exogenous source of variation from the differences in the actual take-up of HIV drugs between Group B and Group C (patients who receive encouragement to adhere to their drug regimen) and Group A (patients with access to HIV drugs only). The study relies on two primary data sources to measure effects: (1) patient, administrative and clinical data from hospitals and clinics, and (2) a dataset constructed from self-reported individual and household surveys.

I find that access to HIV therapy induces significant effects on risky sexual behaviour. Using both self-reported and clinical panel data collected between 2007 and 2010, I find that therapy causes (1) an increase of 0.95 sexual partners relative to the average at the onset of the study and (2) a 33-percentage point decrease in the likelihood of condom use. However, the magnitude of the inoculation effect dwarfs the magnitude of increased behavioural response due to viral suppression attributed to HIV therapy (Cohen et al., 2011). Therefore, the increased frequency of unsafe sex among HIV-positive individuals on HIV therapy is unlikely to fuel the future course of the epidemic.

This paper contributes to the applied microeconomic literature by providing empirical evidence on behavioural responses in the context of a significant drug policy intervention in Sub-Saharan Africa. It advances this literature in several ways. First, previous compensatory behavioural response studies primarily address car safety regulations in the US and find evidence consistent with riskier behaviour in response to safety improvements. Second, in the context of compensatory behavioural responses to HIV therapy among HIV-positive individuals, the best causal empirical evidence comes from the US and uses a unique study population (Lakdawalla, Sood, & Goldman, 2006). Lakdawalla et al. (2006) examine the effect of HIV therapy breakthroughs on the sexual activity of Medicaid-eligible patients in the US. The study finds that access to HIV
therapy increases a compensatory effect with more sexual activity. Third, in the context of HIV in Africa, recent economic studies focus on HIV-related issues, but none examine the impact of treatment provision on HIV-positive individuals. Previous studies in developing countries generally examine behavioural responses to information or preventive measures rather than treatment (Godlonton, Munthali, & Thornton, 2016). Finally, a significant contribution of this paper is its robust causal estimation, facilitated by the study’s experimental design. To date, no experimental evidence investigates the effects of behavioural responses to HIV drug therapy on sexual behaviour in a developing country; this study fills that gap. Since I use a field experiment, I can identify the causal effects of therapy on the two behavioural outcomes.

The structure of the study is as follows. Section 2 describes the conceptual framework and testable predictions, and Section 3 outlines the South African programme. Section 4 presents the experimental design, data sources, and data summary. Section 5 describes the empirical strategy, while Section 6 presents the results. Section 7 examines various robustness checks, and Section 8 concludes.

2. Provision of HIV therapy and behavioral responses

Several conceptual mechanisms underlie the testable hypothesis of this field experiment. First, infected patients who practice safe sex have a higher life expectancy than those who do not. Unsafe sex can lead to co- or super-infection with a new HIV strain, both of which cause early mortality and lower life expectancy (Streeck et al., 2008). Second, providing HIV drugs reduces the economic incentive for health precautions because HIV drugs insulate infected individuals from co-infection. Third, receiving HIV therapy modifies the perception of mortality risks for infected individuals and, on average, increases risky sexual behaviour. Moreover, as therapy improves, HIV prevalence is expected to rise because a higher proportion of healthier HIV-positive individuals will comprise the market for risky exposures (Kremer, 1996). This mechanism is the indirect, general equilibrium effect—HIV therapy suppresses HIV replication to fewer than 50 copies of the virus in most patients over several weeks (Vergidis, Falagas, & Hamer, 2009). Overall, therapy suppresses plasma viral loads to undetectable levels, thus lowering the risk of transmission during risky acts (Margin [c]). HIV therapy also reduces the prevalence of anaemia, which occurs commonly with HIV infection, thereby improving physical health (Murphy et al., 2001) and patients’ capacity to have sex (Margin [d]). Finally, HIV-positive individuals can exhibit altruism by choosing not to engage in risky (i.e. unprotected) sex with anyone not known to be HIV-positive (Margin [e]).

Appendix B (available in the Supplementary Materials) details a comprehensive theoretical model (under various assumptions and possible scenarios) accounting for these specific mechanisms. Based on this model, I derive theoretical predictions (reported in Appendix B available in the Supplementary Materials) regarding the relationship between improved HIV therapy and individual risk-taking among HIV-positive individuals.

3. Background

3.1. South Africa’s HIV ART programme

The South African government began providing antiretroviral therapy (ART) via its public health clinics in June 2004 (McLaren, 2015). Since the president and the health minister had both perpetuated myths that HIV did not cause AIDS and that ART was ineffective, HIV/AIDS therapy was a highly politicised issue at the time, with an estimated 3.4 million people in need of therapy. Nevertheless, through a court order by the High Court of South Africa, the
programme was introduced and aimed to achieve universal patient access by 2009 (Department of Health, 2007; McLaren, 2015), with an estimated annual cost of between US$1 billion and US$1.09 billion.

Provincial governments oversaw clinic rollout and operated under reasonable autonomy. Local health departments identified facilities eligible for accreditation according to the following criteria: (1) one clinic per district, (2) meeting accreditation requirements within a year, (3) large HIV-infected populations within the district, and (4) the geographic placement of facilities maintaining low transportation costs for patients. The National Department of Health provided feedback and visited onsite to determine whether facilities met the accreditation criteria. In most cases, proposed therapy centres were already providing other HIV/AIDS care, so they could start enrolling patients from this care pool in the new ART programme as soon as they became accredited.

McLaren (2015) shows that most people elected to attend the nearest clinic to reduce travel costs; such costs were the primary obstacle to obtaining access. Most patients faced high travel costs because they had to meet stringent eligibility requirements for therapy and be monitored for drug adherence. Patients were asked to return to their clinic approximately four times in the first two months of therapy and then monthly or quarterly if no complications arose.

By mid-2008, 568,000 HIV-infected patients received ART in South Africa, with public sector patients accounting for 79 per cent of this total. Based on the 2008 Department of Health criteria for defining antiretroviral eligibility (i.e. CD4 count < 200/μL or World Health Organization [WHO] Stage 4), antiretroviral coverage among eligible HIV-positive adults in the country was 40.2 per cent that year (Adam & Johnson, 2009). The average antiretroviral coverage masks significant regional heterogeneity in the gap between the number of people eligible for antiretroviral therapy and the number receiving treatment. Coverage varied significantly across the provinces, with the Free State province ranking last among all South African states in 2008 (25.8%).

3.2. The Free State programme

At the start of the national ART programme in 2004, HIV prevalence in the Free State was 29.5 per cent (Department of Health, 2005), the third-highest out of all provinces in South Africa. Based on the national ART eligibility criteria outlined earlier, the coverage in the province increased from 5 per cent (or 2,200 patients) in 2004 to 60 per cent (or 66,000 patients) in 2010 (Johnson, 2012). However, the regional model of providing therapy through a few centrally located clinics created geographic and transportation barriers. HIV therapy was available at 31 primary health clinics or community health centres.

4. Experimental design and data

4.1. Study sample

Launched in October 2007, the experimental intervention enlisted 12 primary clinics and community health centres across five districts in the Free State that were already part of South Africa’s ART programme. Based on the patient pool of the recruited clinics, the study enrolled a random sample of individuals presenting for care who had already been on HIV therapy (Appendix Table A1, available in the Supplementary Materials, and Figure 1 detail the list of clinics and their locations). This sample was recruited between October 2007 and October 2008. Upon enrolment, individuals were randomised into one of three groups (see Table 1):

- HIV/AIDS therapy only (Treatment Group A)
- HIV/AIDS therapy with encouragement support (Treatment Group B)
- HIV/AIDS therapy with encouragement support and a nutritional supplement (Treatment Group C).
4.2. Individual group randomisation

Individuals recruited into Group A received HIV therapy following South Africa’s ART treatment guidelines. The criteria for HIV therapy initiation in adults and adolescents at the study’s onset were a CD4 count of fewer than 200 cells/mm³, irrespective of the stage, or a

| Treatment group | Description | Observations a |
|-----------------|-------------|----------------|
| Treatment Group A | Households including ART patients who receive the ART treatment and associated support provided as part of government’s ART treatment program | 216 |
| Treatment Group B | Households including ART patients who receive the ART treatment and associated support provided as part of government’s ART treatment program PLUS adherence support provided by a trained peer adherence supporter during twice-weekly visits to the patient | 216 |
| Treatment Group C | Households including ART patients who receive the ART treatment and associated support provided as part of government’s ART treatment program PLUS adherence support provided by a trained peer adherence supporter during twice-weekly visits to the patient PLUS nutritional supplementation (i.e. weekly delivery of two 400-gram cans of meatballs and spaghetti in tomato sauce by peer adherence supporter) | 216 |

aNumber of observations reported at the household level.

4.2. Individual group randomisation

Individuals recruited into Group A received HIV therapy following South Africa’s ART treatment guidelines. The criteria for HIV therapy initiation in adults and adolescents at the study’s onset were a CD4 count of fewer than 200 cells/mm³, irrespective of the stage, or a
WHO Stage 4 AIDS-defining illness, regardless of CD4 count, and an expressed willingness and readiness by the patient to adhere to ART treatment. Before therapy, individuals underwent a therapy readiness assessment and were introduced to monitoring processes and study protocols (Department of Health, 2007).

In addition to undergoing HIV therapy, individuals recruited into Group B were assigned a peer adherence supporter who agreed to provide support throughout the study. The adherence supporters had prior experience with ART and were not currently working for the South African national ART programme or non-governmental organisations providing similar services. The study employed 56 peer adherence supporters and 12 peer adherence supporter reserves, one at each study site. The peer adherence supporters were provided with theoretical and practical training on ART treatment and adherence support (four 5-day training sessions were conducted in Bloemfontein, Sasolburg, Welkom, and Phuthaditjhaba).

The peer adherence supporters were paid a monthly stipend of R500 (approximately US$76); each supporter was required to make two visits per week to eight ART patients for the duration of the study. They addressed patient complaints, provided advice on the ART regimen, made facility referrals if required, performed a weekly pill count to assess adherence, and provided counselling regarding medication adherence. The supporters monitored for adverse side effects or medical events during each visit since the last patient visit.

Peer adherence support coordinators, appointed by the University of the Free State Centre, provided supervisory and quality control services to the study in collaboration with staff from the University of the Free State School of Nursing. The supervision entailed monthly visits by a trained peer adherence support coordinator to each study site. During each visit, the supervisors met with peer adherence supporters for a debriefing session and visited several randomly selected patients receiving adherence support for quality assurance purposes. Every month, the supervisors provided feedback to the research team regarding ongoing progress with the peer adherence support intervention.

The existing public sector patients and other stakeholders responded positively to the adherence encouragement intervention. For example, in a pilot pre-study aimed to assess the viability of adherence support among patients, 18 of the 22 patients on HIV therapy (only part of the pilot study) interviewed in HIV clinics before the start of the main study had indicated that they would accept such support. Patient’s main concern was the potential risk of inadvertently disclosing study participants’ HIV status. Such concerns were taken seriously and accounted for when developing the consent guidelines—the training materials trained personnel to prevent such occurrences.

In addition to adherence support, individuals recruited into Group C received a nutritional supplement in the form of two 400 g cans of meatballs and spaghetti in tomato sauce per week for 12 months. Each can contain (per 100 g) 420 kJ of energy, 6.6 g of protein, 9.9 g of carbohydrates, 4.1 g of fat, 29.9 mg of magnesium, 116.6 mg of phosphorous, and 1.6 mg of iron. Adherence supporters distributed the nutritional supplement to ART patients during their weekly visits.

The objective of the nutritional intervention was to bolster patients’ CD4 cell count by boosting their caloric intake and weight (Sachdeva et al., 2011); metabolic changes during HIV infection necessitate an increased amount of calories and protein (Colecraft, 2008). Further, weight loss and low energy intake lead to faster disease progression (ASSAf, 2007, p. 132). The primary mechanisms for the nutritional supplement to boost CD4 cell count were a positive nitrogen balance and improved muscle mass (Fenton & Silverman, 2008, p. 1011).

4.3. Survey data

4.3.1. Baseline data. The study team collected data from a self-reported survey and clinical and provincial records. The individual and household questionnaires collected socio-economic and
demographic data and information on sexual practices. The sexual behaviour module asked for various self-reported measures, including the number of regular and non-regular partners patients had in the six months before the interview, condom use during the last sexual encounter, and frequency of protected or unprotected intercourse, conditional on the interviewee reporting any sexual partners in the past six months. The average patient age was 37.1 years old. Seventy-six per cent of patients were female, 19 per cent had completed secondary school, 98 per cent were black, and 52 per cent were in the labour force. The average monthly income was R933 (approximately US$120). Table 2 summarises the baseline patient characteristics at the study’s commencement.

Table 2 also presents balancing tests on observable socio-economic characteristics for the three treatment groups. I test the equality of means via group assignment in two ways. With Joint Test 1, I test whether the means for the treatment groups B and C were equal. Joint Test 2 tests the hypothesis that the means of the treatment groups B and C are equal to the mean for Group A. None of the variables exhibit statistical significance, suggesting the successful implementation of random assignment.

4.3.2. Follow-up survey. Two follow-up waves were conducted approximately 464 and 776 days after the baseline interview. These follow-up waves, conducted by trained enumerators using a structured questionnaire, comprised a patient interview, a household interview, and an adult questionnaire. Additionally, the study team also conducted a facility survey, during which semi-structured interviews were conducted with health care providers at each study site. Upon completing the study (in the last quarter of 2010 and early 2011), the team collected clinical biomarkers and additional patient information from paper and electronic files.

Besides individual and household socio-economic characteristics, follow-up data comprise four main types: clinical data on CD4 and RNA viral load, number of sexual partners, type of sexual partners, and sexual practices. The household questionnaire module collected data on the number of children born to each female patient after the onset of the study. Table 3 reports key outcomes by treatment group.

4.3.3. Panel study attrition. Subjects in one of the treatment groups may drop out of the study at different rates than subjects in the other treatment groups. Since substantial differential attrition could result in biased results, I explore this possibility in the context of this panel. The recorded attrition rate for this study was slightly higher than 20 per cent in the panel data used in the field experiment (primarily attributed to mortality). Table 4 reports regression results for attrition status in the follow-up wave on baseline treatment assignment and patient covariates. The results in Table 4 do not support the possibility of differential attrition in the study sample.

5. Identification strategy

In general, the objective of this paper is to estimate an equation of the form:

\[ S_{it} = \theta_{it} + \zeta_1 T_{it} + \zeta_2 X_{it} + \epsilon_i \]  

(1)

where \( S_{it} \) measures an individual \( i \)'s unsafe sexual behaviour, \( t \) indicates a period. \( T_{it} \) indicates HIV therapy access, and \( X_{it} \) is a set of individual and cluster-level controls. A naïve OLS estimate of \( \zeta_1 \) based on specification (1) is likely to be biased in the presence of positive selection into treatment. Therefore, I employ an alternative route to estimate Equation (1) using an estimating strategy based on a randomised IV design.
Table 2. Summary characteristics

| Characteristics                  | Full sample | Treatment Group A | Treatment Group B | Treatment Group C | \(p\)-Value joint test 1\(^a\) | \(p\)-Value joint test 2\(^b\) |
|---------------------------------|-------------|-------------------|-------------------|-------------------|-------------------------------|-------------------------------|
| Age (years)                     | 37.10 (8.95)| 37.40 (9.40)      | 36.87 (8.71)      | 37.19 (8.72)      | 0.86                          | 0.63                          |
| Sex (1 if female)               | 0.76 (0.43) | 0.78 (0.42)       | 0.74 (0.44)       | 0.77 (0.42)       | 0.73                          | 0.53                          |
| Ethnicity (1 if African Basotho) | 0.98 (0.12) | 0.98 (0.15)       | 0.99 (0.10)       | 0.99 (0.12)       | 0.86                          | 0.27                          |
| Currently married (1 if yes)    | 0.23 (0.43) | 0.30 (0.46)       | 0.15 (0.36)       | 0.26 (0.44)       | 0.41                          | 0.63                          |
| Spouse lives here (1 if yes)    | 0.70 (0.46) | 0.72 (0.45)       | 0.67 (0.47)       | 0.70 (0.46)       | 0.92                          | 0.62                          |
| High school graduate (1 if yes) | 0.18 (0.39) | 0.17 (0.37)       | 0.17 (0.38)       | 0.22 (0.41)       | 0.11                          | 0.36                          |
| Currently in school (1 if yes)  | 0.01 (0.12) | 0.02 (0.14)       | 0.01 (0.10)       | 0.01 (0.12)       | 0.97                          | 0.49                          |
| Monetary cost to facility (ZAR) | 7.30 (20.34)| 9.37 (35.12)      | 6.14 (7.23)       | 6.49 (7.50)       | 0.49                          | 0.21                          |
| Minutes to health facility      | 49.59 (51.58)| 50.77 (51.89)      | 49.28 (53.57)     | 48.15 (50.00)     | 0.63                          | 0.64                          |
| Moved to area in last five years (1 if yes) | 0.05 (0.22) | 0.07 (0.26) | 0.04 (0.21) | 0.03 (0.18) | 0.15 | 0.14 |
| Monthly earnings (ZAR)          | 1,530.45 (1,643.95)| 1375.14 (1393.82) | 1670.29 (1936.30) | 1576.27 (1636.87) | 0.82 | 0.41 |
| Currently employed (1 if yes)   | 0.26 (0.44) | 0.29 (0.45)       | 0.24 (0.43)       | 0.27 (0.45)       | 0.75                          | 0.38                          |
| Currently on disability grant (1 if yes) | 0.30 (0.46) | 0.27 (0.45) | 0.29 (0.46) | 0.34 (0.47) | 0.13 | 0.27 |
| Number of rooms in dwelling     | 3.39 (1.50) | 3.27 (1.47)       | 3.42 (1.37)       | 3.46 (1.68)       | 0.43                          | 0.20                          |
| Flush type toilet (1 if yes)    | 0.64 (0.48) | 0.60 (0.49)       | 0.67 (0.47)       | 0.63 (0.48)       | 0.87                          | 0.23                          |
| Power grid (1 if yes)           | 0.84 (0.37) | 0.81 (0.39)       | 0.86 (0.35)       | 0.84 (0.36)       | 0.84                          | 0.23                          |
| Weight (kg)                     | 61.25 (13.84)| 60.87 (13.36)      | 61.66 (14.74)     | 61.35 (13.43)     | 0.91                          | 0.57                          |
| Height (cm)                     | 158.65 (11.81)| 158.31 (12.19)    | 159.11 (11.15)    | 158.66 (12.20)    | 0.99                          | 0.76                          |
| CD4 count                       | 149.94 (99.45)| 148.27 (102.77)   | 146.82 (87.33)    | 155.71 (107.76)   | 0.38                          | 0.32                          |
| Viral Load RNA                  | 152,201.49 (433,972.58) | 182,899.12 (570,774.09) | 151,451.48 (395,073.36) | 120,870.22 (288,002.17) | 0.32 | 0.14 |
| Number sexual partners          | 0.30 (0.28) | 0.32 (0.46)       | 0.32 (0.47)       | 0.33 (0.46)       | 0.41                          | 0.49                          |
| Condom use (1 if yes)           | 0.89 (0.31) | 0.91 (0.29)       | 0.89 (0.31)       | 0.90 (0.30)       | 0.79                          | 0.71                          |
| Observations                    | 648         | 216               | 216               | 216               |                               |                               |

Notes: Mean of the specified variables are reported. Standard deviations in parentheses. At the time of the program (2010), 1 USD was approximately equal to 7 South Africa Rand (ZAR).
\(^a\)Joint test 1: Test of equality of means between the treatment groups (i.e. Treatment Groups B and C).
\(^b\)Joint test 2: Joint test that means in treatment groups (Treatment Groups B and C) are equal to the mean in the control group (Treatment Group A).
Following Angrist et al. (1996), I employ an IV two-stage least squares (2SLS) strategy, exploiting the study’s experimental design for individuals assigned to Groups B and C. The study design is an experimental encouragement design used when the researcher has little control over the subjects’ compliance or when randomising into a particular treatment group might be considered unethical. I use a variable (the instrument, which I call $Z_i$), correlated with $T_i$, but uncorrelated with any other determinants of the dependent variable, unsafe sexual behavior. Using this approach, I can obtain unbiased estimates of the effect of access to HIV

| Table 3. Study outcomes by group | Year | Treatment Group A | Treatment Group B | Treatment Group C |
|----------------------------------|------|-------------------|-------------------|-------------------|
| Panel A Sexual partners (mean)   | 2007 | 0.32 (0.46)       | 0.32 (0.47)       | 0.33 (0.46)       |
|                                  | 2010 | 0.24 (0.42)       | 0.61 (0.42)       | 0.50 (0.43)       |
| Panel B All biological children | 2010 | 0.26 (0.52)       | 0.34 (0.52)       | 0.23 (0.42)       |
| reported at follow-up per       |      |                   |                   |                   |
| treated HH (average)            |      |                   |                   |                   |
| New biological children         | 2010 | 0.22 (0.41)       | 0.32 (0.47)       | 0.23 (0.42)       |
| at follow-up per treated HH     |      |                   |                   |                   |
| (average)                       |      |                   |                   |                   |
| # HHs with incidence ≥1         | 2010 | 50                | 46                | 47                |
| Newborns$^a$                    |      |                   |                   |                   |
| CD4 change within               |      |                   |                   |                   |
| study duration$^b$               |      |                   |                   |                   |
| Observations                    | 216  | 216               | 216               |                   |

Note: St. deviations in parenthesis.

$^a$Reported by females in treated households.

$^b$The study duration is the follow-up (2010) and baseline (2007).

| Table 4. Attrition status and patient baseline characteristics |
|---------------------------------------------------------------|

| Attrition status (=1 if yes)                                  |
|---------------------------------------------------------------|
| Treatment group (=1 if in Group B or C)                       | -0.12 (0.12) |
| Age                                                            | -0.00 (0.02) |
| Sex (=1 if female)                                             | -0.19 (0.24) |
| Ethnicity (=1 if African Basotho)                              | 0.05 (0.67)  |
| Currently married (=1 if yes)                                  | -0.14 (0.23) |
| Spouse lives here (=1 if yes)                                  | -0.01 (0.25) |
| High school graduate (=1 if yes)                               | 0.12 (0.27)  |
| Monetary cost to facility (in ZAR)                             | 0.02 (0.02)  |
| Minutes to facility                                            | 0.00 (0.00)  |
| Moved to area in last five years (=1 if yes)                   | 0.12 (0.49)  |
| Monthly earnings (in ZAR)                                     | -0.00 (0.00) |
| Currently employed (=1 if yes)                                 | 0.76 (0.65)  |
| Currently on disability grant (=1 if yes)                     | -0.11 (0.24) |
| Number of rooms in the dwelling                                | 0.01 (0.08)  |
| Weight (in kg)                                                 | -0.00 (0.01) |
| Height (in cm)                                                 | 0.00 (0.01)  |
| $R^2$ statistic                                                | 0.25         |
| Observations                                                   | 648          |

Notes: Clustered standard errors by clinic in parentheses. Linear Probability Model.

*Significant at the 10 per cent level.
**Significant at the 5 per cent level.
***Significant at the 1 per cent level.

Following Angrist et al. (1996), I employ an IV two-stage least squares (2SLS) strategy, exploiting the study’s experimental design for individuals assigned to Groups B and C. The study design is an experimental encouragement design used when the researcher has little control over the subjects’ compliance or when randomising into a particular treatment group might be considered unethical. I use a variable (the instrument, which I call $Z_i$), correlated with $T_i$, but uncorrelated with any other determinants of the dependent variable, unsafe sexual behavior. Using this approach, I can obtain unbiased estimates of the effect of access to HIV
therapy. I instrument for therapy take-up using dummy indicators for whether a patient received encouragement support (i.e. was in Group B) or received encouragement support plus a nutritional intervention (i.e. was in Group C). The specification for HIV therapy take-up ($T_{it}$) is as follows:

$$T_{it} = z_{it} + \beta_1 Encouragement_{it} + \beta_2 EncouragementAndNutrition_{it} + \beta_3 X_{it} + \epsilon_{it}$$  \hspace{1cm} (2)

For each person $i$, I observe a binary variable $Encouragement_{it}$, set to 1 if patient $i$ received encouragement support from a biweekly visitor, and 0 otherwise. The binary variable $EncouragementAndNutrition_{it}$ is set to 1 if patient $i$ received biweekly visitor support and nutritional supplements, and 0 otherwise. The binary $T_{it}$ is set to 1 if person $i$ received the therapy, and 0 otherwise. For robustness, I employ both binary and continuous specifications of the endogenous variable $T_{it}$. The continuous variable is the actual CD4 count obtained from clinical records. The binary-defined treatment is set to 1 if $\Delta CD4 > 0$ and 0 if $\Delta CD4 \leq 0$. $X_{it}$ is a vector of baseline demographic factors, and the outcome variable is $S_{it}$. Since all three groups could receive HIV therapy, I exploit exogenous sources of identification from differences in actual $T_{it}$ between Group B and Group C vs. Group A.

Under the standard assumptions—direct effect on the endogenous variable, independence, exclusion restriction, and monotonicity—for IV estimation (Angrist et al., 1996), I estimate the causal effects of HIV therapy, $T_{it}$, on $S_{it}$ by estimating $\gamma_1$ of Equation (1). Although I specify $T_{it}$ as both a binary and a continuous variable for robustness purposes, in the estimation regarding ‘whether a condom was used in last sexual encounter’ (specified as a binary variable), I employ $S_{it}$, $T_{it}$, and $Z_{it}$, all of which are binary indicators. If $S_{it}$, $T_{it}$, and $Z_{it}$ are binary specified, then:

$$\beta_{iv} = \frac{P(S = 1|Z = 1) - P(S = 1|Z = 0)}{P(T = 1|Z = 1) - P(T = 1|Z = 0)}$$

$$\hat{\beta}_{iv} = \frac{P(S = 1|Z = 1) - P(S = 1|Z = 0)}{P(T = 1|Z = 1) - P(T = 1|Z = 0)}$$

If all four IV assumptions hold, I estimate the local average treatment effect (LATE) as follows:

$$\text{LATE}_{HIV \ \text{THERAPY}} = \frac{E[S_{it} - S_{it}|T_{i}(1) - T_{i}(0) = 1] - (E[S_{i}|Z_{i} = 1] - E[S_{i}|Z_{i} = 0])}{P(1) - P(0)}$$  \hspace{1cm} (3)

Equation (3) describes the estimand I present in the results section.

6. Results

6.1. Predicting take-up of HIV therapy

I analyse the take-up of HIV therapy for the individuals randomly assigned to Groups B and C. The intent to treat (ITT) estimates the effect on patients of being offered biweekly encouragement based on:

$$S_{it} = \delta_{it} + \phi_1 Encouragement_{it} + \beta_3 X_{it} + \epsilon_{it}$$  \hspace{1cm} (4)

$\phi_1$ provides an ITT effect estimate.
Under the assumption that the encouragement groups had no direct effect on risky sexual activity except through HIV therapy, I estimate the treatment on the treated (TOT) effect by dividing the ITT by the take-up rate. I could also obtain this estimate through an instrumental variable estimation using the random assignment to treatment as an instrument for take-up, as described by Equation (2). The TOT effect is:

\[
\text{TOT} = \frac{\text{Outcome}(\text{Group B and C}) - \text{Outcome}(\text{Group A})}{\text{Prob}[\text{treated} | (\text{Group B or Group C})] - \text{Prob}[\text{treated} | (\text{Group A})]} = \frac{\text{Outcome}(\text{Group B and C}) - \text{Outcome}(\text{Group A})}{0.22}
\]

Appendix Table A3 (available in Supplementary Materials) reports the ITT effects.

6.2. Impact of HIV therapy on sexual behavior

Because the Equation (1) estimate is over-identified, I can somewhat test the exclusion restriction and whether the excluded instruments are appropriately independent of the error process. To do so, I compute a Sargan statistic in a particular case of Hansen’s J under the assumption of conditional homoscedasticity and fail to reject the null hypothesis that the instruments are all orthogonal to the error term. The magnitude of the coefficients associated with Groups B and C are large and positive, implying significant effects of treatment group assignment on the instrumented therapy variable. To verify the robustness of the validity of the first-stage estimation, I conduct several over-identification tests. Table 5 presents first-stage IV estimates for both the discrete and continuous definition of HIV therapy, \(T_i\), and reports the result of the

Table 5. First-stage of 2SLS estimation: impacts on sexual behavior

|                  | HIV therapy continuous definition IV (LPM) | HIV therapy discrete definition IV (LPM) |
|------------------|--------------------------------------------|-----------------------------------------|
| Panel A          |                                            |                                         |
| Group B (=1 if yes) | 44.79***                                   | 0.19***                                  |
|                  | (7.03)                                      | (0.03)                                   |
| Group C (=1 if yes) | 46.07***                                   | 0.18***                                  |
|                  | (6.84)                                      | (0.03)                                   |
| F-statistic      | 33.58                                      | 26.79                                    |
| Observations     | 625                                         | 625                                      |
| Anderson’s       |                                            |                                         |
| Canonical Statistic\(^a\) |                                         |                                         |
| Cragg-Donald     |                                            |                                         |
| Stock & Yogo\(^a\) |                                            |                                         |
| Panel B          | Over-identification Tests                  |                                         |
|                  | 34.38                                       | 25.07                                    |
|                  | 19.93                                       |                                         |
| Over-identification tests |                                        |                                         |
| Hansen J Statistic | 0.639                                      | 0.681                                    |
| (\(p\)-val = 0.42) |                                            | (\(p\)-val = 0.41)                      |

Note: Clustered standard errors by clinic in parentheses.

\(^a\)Significant at the 10 per cent level.

\(^\ast\)Significant at the 5 per cent level.

\(^\ast\ast\)Significant at the 1 per cent level.

\(^\ast\ast\ast\)Significant at the 0.1 per cent level.

\(^a\)H\(_0\): equation is weakly identified.
over-identification tests: all tests reject the null hypothesis of a weakly identified first-stage equation.

Table 6 reports the results of the OLS, 2SLS, and limited information maximum likelihood (LIML) estimations for two outcomes: (1) the number of sexual partners and (2) condom use in the last sexual encounter.

Generally, IV results indicate an increase in sexual partners and a substantial decrease in condom use. Ceteris paribus, HIV therapy increases the number of sexual partners by 0.95 partners and decreases the probability of condom use by 0.33. The 2SLS estimate is higher than the OLS estimates. Several reasons can account for this discrepancy. First, the 2SLS procedure estimates a LATE, likely based on individuals with a high marginal return to HIV therapy. For example, it could be that the instrument affects patients with a high discount rate or from more disadvantaged backgrounds. Second, IV estimates could reduce the downward attenuation bias due to measurement error. Third, the IV estimates may be biased upward from non-classical measurement error given the discrete nature of the HIV therapy data. Further, and interestingly, the OLS results indicate a positive association between HIV therapy and condom use, unlike the 2SLS estimate for the condom use outcome. The OLS results are similar to those of the only observational study in the literature conducted in an African context, which finds that patients decrease their risky sexual behaviour six months after initiating ART by 70 per cent.

The difference between the OLS and 2SLS results indicates that the OLS is likely affected by either strong selection-into-treatment bias or measurement issues. Since the independent variable, $T_i$, is measured using biomarker data from clinical records, it is unlikely to suffer from random measurement error. OLS estimation yields a positive coefficient for $\zeta_1$ in Equation (1); however, the 2SLS estimation yields a negative coefficient estimate for $\zeta_1$. This sign reversal indicates a negative correlation between $T_i$ and $S_i$ (people who seek therapy practice safer sex, indicating that the more health-conscious individuals self-select into treatment).

### 6.3. Heterogeneous treatment analysis

Next, I repeat the specifications presented in the primary analysis, and I examine for heterogeneous treatment effects based on demographic characteristics, including gender, marital status, secondary school attainment, and earnings of more than ZAR2,000 per month (approximately US$330 per month at the time of the intervention). In Table 7, I repeat the TOT regressions but interacted with the treatment indicator variable, one demographic variable at a time.
|                          | (1)          | (2)          | (3)          | (4)          | (5)          | (6)          | (7)          | (8)          |
|--------------------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|
| HIV therapy (=1 if yes, binary) | 1.12***      | 1.17***      | 0.89***      | 1.39***      | -0.33***     | -0.34***     | -0.35***     | -0.28***     |
|                          | (0.35)       | (0.20)       | (0.20)       | (0.37)       | (0.09)       | (0.10)       | (0.05)       | (0.14)       |
| Female (=1 if yes)       | -0.17        |              | -0.02        |              |              |              |              |              |
|                          | (0.13)       |              | (0.06)       |              |              |              |              |              |
| Female × HIV therapy     | -0.18        |              | 0.10         |              |              |              |              |              |
|                          | (0.40)       |              | (0.14)       |              |              |              |              |              |
| Married (1 = if yes)     |              | 0.33***      |              |              |              |              | 0.16         |              |
|                          |              | (0.12)       |              |              |              |              | (0.19)       |              |
| Married × HIV therapy    | -0.09        |              | -0.55        |              |              |              |              |              |
|                          | (0.37)       |              | (0.56)       |              |              |              |              |              |
| High school graduate (=1 if yes) | -0.13        |              |              |              | -0.08        |              |              |              |
|                          | (0.14)       |              |              |              | (0.08)       |              |              |              |
| High school × HIV therapy| 0.43         |              |              |              | 0.16         |              |              |              |
|                          | (0.42)       |              |              |              | (0.21)       |              |              |              |
| Earnings > 2000 ZAR/mo   |              |              |              |              | 0.60***      |              |              |              |
| month (=1 if yes)        |              |              |              |              | (0.22)       |              |              |              |
| Earnings > 2000 ZAR       |              |              |              |              | -1.38***     |              |              |              |
| ZAR × HIV therapy        |              |              |              |              | (0.65)       |              |              |              |
| Demographic controls     | Yes          | Yes          | Yes          | Yes          | Yes          | Yes          | Yes          | Yes          |
| $R^2$ statistic           | 0.07         | 0.12         | 0.07         | 0.17         | 0.02         | 0.03         | 0.03         | 0.14         |
| Observations              | 625          | 625          | 625          | 140          | 236          | 236          | 236          | 72           |

**Note:** Clustered standard errors by clinic in parentheses.

*Significant at the 10 per cent level.

**Significant at the 5 per cent level.

***Significant at the 1 per cent level.

aFor condom use, the coefficients are the average marginal effects.
Except for the earnings variable, the coefficient on the interaction term is insignificant for all demographic variables. This finding suggests that the average effect of the treatment assignment works uniformly across these characteristics within the treatment groups.

6.3.1. Complier characteristics. Due to possible imperfect compliance with the treatment group assignment, I can only estimate causal effects with Equation (3) for a subset of the population of eligible individuals. Further, due to imperfect compliance, the instrumental variable estimation results should be interpreted as LATEs for patients who take up HIV therapy only because of the encouragement (Imbens & Angrist, 1994). I characterise the complier group by socio-economic characteristics; Appendix Table A7 (available in Supplementary Materials) describes the complier group in terms of their likelihood of exhibiting a particular characteristic. Most notably, compliers are 2.14 times more likely to be currently attending school, 2.61 times more likely to live within 30 min of a clinic, and 1.92 times more likely to have a body mass index (BMI) <25.

7. Robustness checks

7.1. Proxy measures of sexual behavior

7.1.1. Childbearing as a proxy measure. I follow Dupas (2011) and use childbearing as a proxy for risky sexual activity (note that childbearing is not a perfect proxy for the incidence of risky sex\(^{10}\)). Data on childbearing comes from the demographic data on new biological children collected in 2010. The results in Table 8 indicate a slight positive increase in childbearing incidence and the number of newborns. However, due to the small sample size, none of the estimates is statistically significant.

7.1.2. Continuous definition of therapy. I proxy HIV therapy status using CD4 and BMI clinical data. As noted previously, I specify the therapy status \(T_{it}\) as both a binary and a continuous variable. The binary therapy definition is \(T = 1\) if \(\Delta CD4 > 0\) and \(T = 0\) if \(\Delta CD4 \leq 0\). For robustness, I also specify therapy as a continuous variable captured by the actual CD4 count. The direction of the result and statistical significance is robust to how I measure HIV therapy.

| # Newborns\(^a\) | # Newborns\(^a\) | Newborn incidence \((=1\text{ if yes})^b\) | Newborn incidence \((=1\text{ if yes})^b\) |
|-----------------|-----------------|-----------------|-----------------|
| 2SLS (1) 2SLS (2) 2SLS (3) 2SLS (4) | 0.01 0.01 0.01 0.01 | (0.07) (0.06) (0.06) (0.06) | No Yes No Yes |
| Controls |
| if yes, binary |
| \(R^2\) statistic 0.01 0.01 0.01 0.01 |
| Observations 494 494 494 494 |

Notes: Clustered standard errors by clinic in parentheses. Demographic controls include education, gender, income, marital status, population group, household assets, HH members, BMI. The analysis is conducted on the female subsample only.

*Significant at the 10 per cent level.
**Significant at the 5 per cent level.
***Significant at the 1 per cent level.
\(^a\) Number of newborns since study baseline.
\(^b\) Newborn incidence is set to 1 if the household reported at least one newborn between the baseline and the follow-up.
7.2. A count data model for sexual partners

In addition to modelling the outcomes as a continuous variable, I estimate an alternative ‘count data’ estimation approach: I model the number of partners as a negative binomial distribution (therapy take-up continues to be modelled only as a linear probability model [Greene, 2008]). The count data model is particularly suitable for data with excess zeros in the outcome variable. The negative binomial regression model allows for over-dispersion (i.e. when the conditional variance exceeds the conditional mean). Using a maximum likelihood estimation, I estimate the effects of therapy on the number of sexual partners, assuming that the outcome variable is negative binomial distributed. Appendix Table A4 (available in Supplementary Materials) reports the results. The estimated coefficient associated with the HIV therapy variable is positive and suggests that access to therapy increases sexual partners by 0.93.

7.3. LIML estimator and non-linearity as a source of identification

The LIML estimator is approximately median-unbiased for over-identified constant-effects models (Angrist & Pischke, 2008), providing an attractive alternative to just-identified estimation using one instrument at a time (Mariano, 2003). Advantageously, LIML has the same large-sample distribution as 2SLS (under constant effects) while providing finite-sample bias reduction. Table 6 reports the LIML estimates of specification (1); they are virtually identical in magnitude and statistical significance to the 2SLS results.

As an alternative identification strategy for (1), I also use the non-linear fitted values as instruments. I can use $T_i$ from a non-linear (logistic) first-stage estimation as an instrument for $T_i$ in a conventional 2SLS procedure. Non-linear-fits-as-instruments have the advantage that if the non-linear model yields a better approximation of the first-stage conditional expectation function than the linear model, the resulting 2SLS estimates will be more efficient than those using a linear model first-stage estimation (Newey, 1990). The non-linear-fits-as-instruments procedure implicitly uses non-linearities in the first stage as a source of identifying information. The pattern of the results, reported in Appendix Table A5 (available in Supplementary Materials), remains the same as that of the main results reported in Table 6.

8. Conclusion

This paper examines the impact of HIV therapy on subsequent sexual behaviour, specifically risky behaviour. Therapy adherence contributes positively to the welfare of infected individuals by improving their longevity and physical health but may also create mechanisms that fuel the further spread of HIV. Since therapy provides viral insulation, HIV therapy could, according to the Peltzman effect theory, increase sexual activity among infected individuals.

Using experimental data from the Free State province in South Africa, I present evidence consistent with this argument. I find that access to HIV therapy leads to a substantial increase in the demand for unsafe sex. Ceteris paribus, HIV therapy increases the number of sexual partners by 0.95 partners and decreases the probability of condom use by 0.33. This study’s estimated effects are larger than those found in Lakdawalla et al. (2006). They find that treating HIV-positive individuals in the US more than doubles the number of sexual partners. Notably, one difference between my study and Lakdawalla et al. (2006) work is that individuals in the South African sample are, on average, at a much more advanced stage of the disease. The results based on the self-reported proxies of risky sexual behaviour in this study are further corroborated by the results based on biological outcomes, as I show a slight positive increase in childbearing by HIV-positive female patients. One shortcoming of the data used in this paper is that, given the anonymity of the survey data, I cannot observe the HIV status of partners with whom patients in this study increased their sexual activity. This data limitation prevents
additional analyses on how the increase in risky sexual behaviour among HIV-positive adults relates to serodiscordant partnerships and the trajectory of the HIV epidemic.

These results generally highlight the importance of considering the full array of potential consequences on the subsequent sexual behaviour. Health interventions often focus on the benefits margin and fail to fully account for the potential behavioural response. In this study, I present evidence of compensatory behaviour—the HIV therapy programme induced an increase in unsafe sexual behaviour among patients.

Although the results of this study are economically meaningful, I note an important caveat in interpreting the welfare consequences of this study’s behavioural results. Recent evidence highlights the inoculation effect of ART and its reduction of the viral load and the risk of sexual transmission by 96 per cent (Cohen et al., 2011). However, even if the behavioural response is not powerful enough to counter the inoculation mechanism caused by increased adherence to HIV therapy, policymakers must weigh the benefits of future drug access against the potential for unintended behavioural consequences. Further research can focus on the individual heterogeneity of behavioural responses and the various channels through which these behavioural responses operate.

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Notes

1. Generally, previous empirical studies on risk compensation (i.e. the so-called Peltzman effect) in high-income countries document moderate Peltzman effects (Peltzman, 1975; see also Evans & Graham, 1991; Keeler, 1994).
2. I use clinical data on CD4 count as a proxy of HIV therapy. Changes in individual CD4 count are a function of multiple patient factors, including improved adherence to the drug regimen.
3. South Africa’s HIV prevalence rate is among the highest in the world (UNAIDS, 2015, 2017, 2019).
4. Several countries, especially in Sub-Saharan Africa, subsidise HIV therapy take-up and adherence (Rosen, Fox, & Gill, 2007). South Africa’s public sector programme subsidises take-up and transport to clinics (Cluver et al., 2016; Govender, Fried, Birch, Chimbindi, & Cleary, 2015).
5. The study design is a natural field experiment based on the taxonomy in Harrison and List’s (2004) work.
6. Throughout the paper, I use ‘therapy’ to refer to the key independent variable, HIV therapy (Ti), in this analysis. Since I do not directly observe Ti and because Ti is endogenous, I instrument it with a binary indicator of whether a study unit belongs to Group B or Group C. I use CD4 blood count as a proxy for Ti. I use the term ‘treatment’ to refer to the experimental treatment groups. The experimental treatment groups in this study are patients receiving biweekly encouragement to adhere to an HIV regimen (Group B) and patients receiving biweekly encouragement to adhere to an HIV regimen plus a nutritional supplement (Group C); Group A, the reference group, received HIV therapy (antiretroviral treatment [ART]) only.
7. More generally, several studies examine general behavioural responses to various factors, such as information (Dupas, 2011; Gong, 2015; Paula, Shapiro, & Todd, 2014), belief changes (Godlonton, Munthali, & Thornton, 2016), longevity (Fortson, 2011; Oster, 2012; Thirumurthy, Pop-Eleches, Habyarimana, Goldstein, & Zivin, 2012), risk (Robinson & Yeh, 2011), and testing (Delavande & Kohler, 2012) in the context of HIV in Sub-Saharan Africa. Friedman (2018) examines the effect of HIV therapy among HIV-negative individuals in Kenya. In the context of the US, previous studies also examine the effect of the availability of HIV therapy on risky behaviour (Chan, Hamilton, & Papageorge, 2016; Mechoulan, 2007).
8. See Peltzman (1975), Evans and Graham (1991), and Keeler (1994).
9. Friedman (2018), Godlonton and Thornton (2013), and Godlonton et al. (2016) examine compensatory behaviour in response to HIV-related factors in the context of a developing country and report contradictory
findings. The studies that specifically examine risk compensation in response to information about the benefits of circumcision or in response to circumcision (Godlonton et al., 2016) find little evidence of risk compensation. In contrast, the studies that examine risk compensation in the context of HIV therapy or HIV testing rates (Friedman, 2018; Godlonton & Thornton, 2013) find some or substantial evidence of risk compensation, especially in response to HIV therapy provision. A potential reason for this discrepancy is that the protective benefit of HIV drugs (viral inoculation) far surpasses any benefits of the interventions studied by Godlonton et al. (2016) (i.e. information, circumcision, information about the HIV status of potential other partners within the community). Therefore, it is likely that compensatory behavioural responses are more substantial once affected individuals internalise the much more significant protective benefits of particular interventions (e.g. HIV drugs). A novel feature of my study is that I focus only on HIV-positive individuals, whereas Friedman (2018)'s sample includes individuals who are HIV-negative.

10. Using data from the US, Delavande, Goldman, and Sood (2010) and Sood, Wagner, and Wu (2015) examine how policy changes can influence HIV-related behavioural responses. Delavande et al. (2010) examine how the stringency of law enforcement for HIV-positive individuals who willfully expose others to the infection influences subsequent risky sexual behaviour and find that prosecutions deter sexual activity, increase safe sex and increase sexual encounters with more promiscuous partners. Sood et al. (2015) find that an increase in insurance coverage increases HIV testing rates and disproportionately increases testing rates among high-risk groups.

11. Godlonton et al. (2016) examine behavioural responses to providing information on the efficacy of male circumcision or the effects of male circumcision.

12. Various observational studies, such as those by Thirumurthy et al. (2012) and Kennedy, O'Reilly, Medley, and Sweat (2007), explore the overall effect of therapy on unsafe sex but do not investigate the contribution of each sub-mechanism of behaviour change. All these studies rely on ordinary least squares (OLS) or propensity score matching, which do not fully account for selection into treatment based on unobservable individual characteristics.

13. Models III and IV of Appendix B (available in the Supplementary Materials) specifically focus on the relationship between improved HIV therapy and risky sexual behaviour among HIV-positive individuals.

14. The treatment regimens used in the public service were 1a ( stavudine, lamivudine and efavirenz), 1b ( stavudine, lamivudine and nevirapine) and 2 ( azidothymidine, didanosine, lopinavir and ritonavir).

15. Inclusion criteria included being of a minimum age of 18 years, having commenced ART within the past five weeks and residing in the town or village in which the relevant health facility was located.

16. The trial adopted a Zelen-type double randomisation consent design. This design is appropriate when blinding is not practicable or possible, the use of classical randomisation and informed consent procedures significantly threatens internal validity, the interventions are highly attractive, the control group receives standard care or the study focuses on a clinically relevant objective(s) and offers important new insights (Kaptchuk, 2001; MacLehose et al., 2000; Rains & Penzien, 2005). Within such a design, study participants are only offered the treatment to which they are randomised and can accept or reject treatment.

17. The actual therapy regimens in the intervention were 1a (d4T/3TC/efavirenz), 1b (d4T/3TC/NVP), and 2 (AZT/ddI/lopinavir/ritonavir).

18. ART readiness was assessed with various qualitative readiness indicators related to self-reported readiness to start antiretrovirals, motivation to start antiretrovirals, mindset about a positive outcome of the therapy and intention to start ART within 30 days.

19. A brief survey instrument was used to collect key information from the peer adherence supporters working in the project. The summary statistics for this group reveal that they were predominantly female (98%), their mean age was 35.8, the majority (56%) walked when they visited a study patient, the average time to a patient was approximately 39 minutes by foot and the majority (98%) met their clients at home.

20. To be considered eligible to become adherence supporters, individuals had to have been on ART for ≥ 6 months, have at least a grade-10 certificate and live within walking distance of the relevant clinic. Peer adherence reporters received basic training in ART and adherence support from staff at the School of Nursing at the University of the Free State. The training focused on seven main themes: facts about HIV/AIDS, ART, adherence support required by an ART client, nutrition, infection control at home and use of a health care team approach. On the fifth day of training, peer adherence supporters had their knowledge and practical skills assessed by the trainers via an oral test and a practical exercise.

21. The study team underscored the importance that the nutritional supplements were for study participants' consumption. This was done at the time of obtaining written informed consent as well as at each visit by the peer adherence supporter. However, due to monitoring limitations, it is likely some participants shared the supplements with other household members.

22. Before the study, the research team had conducted a qualitative survey regarding the acceptability of the supplement to patients. All patients had indicated that, if provided with such services, they would eat meatballs and spaghetti in tomato sauce and would eat it on a weekly basis when it was provided as part of the research study.

23. While the accuracy is usually higher since the defined recall period is closer to the time of inquiry, this also creates a caveat for my results. This is based on numerous examples of recall difficulties in sex research,
notwithstanding many other methodological factors affecting self-reported behaviour. In my setup, if \( E(e_i,S_i) = 0 \) in (1), then OLS estimates unbiased by standard errors will be higher. Conversely, if \( E(e_i,S_i) > 0 \), then OLS will be biased and inconsistent. I return to this point in the Results section when I compare the OLS with the IV estimates.

24. Self-reported outcomes are prone to potential misreporting (Mensch, Hewett, & Erulkar, 2003). Such misreporting occurs due to social norms and social networks (Mensch et al., 2003; Tavory & Swidler, 2009), urbanicity and gender (Fenton, Johnson, McManus, & Erens, 2001). To this end, and consistent with other studies, I report results based on self-reported outcomes as well as biological data, such as childbearing (e.g. Fishbein & Pequegnat, 2000). Importantly, even if misreporting or under-reporting of risky sexual behaviour occurred, the random assignment likely considerably mitigated any potential bias.

25. Written, informed consent was obtained from study participants by the nursing personnel at the respective clinics (for antiretroviral patients), as well as by the enumerator (for ART patients and ART patient/comparison households).

26. Because risky sexual behaviour is a sensitive subject, an obvious concern relates to misreporting and measurement error (Helleringer, Kohler, Kaililani-Phiri, Mkandawire, & Armbruster, 2011). Previous studies document that under-reporting numbers of sexual partners is a particular issue with regard to self-reported data. This phenomenon occurs due to social norms or potential stigma in most African cultures (Palen et al., 2008). To gauge the reliability of the self-reported outcomes, and consistent with previous studies (Fishbein & Pequegnat, 2000; Minnis et al., 2009), I report results based on the self-reported outcomes and outcomes based on biological data (childbearing). It is important to underscore the random assignment feature of this study in relation to possible reporting biases; even if under-reporting of risky sexual behaviour occurred, the only way the estimated effect size is biased is through a relationship between the reporting of sexual activity and the treatment group assignment. Since assignment into Group B and Group C was randomised, the measurement error or any potential reporting bias is likely to be unrelated to the instruments I employ in this study.

27. Relative to participants in Group B or C, participants in Group A attrited at a higher rate but the difference was not statistically significant.

28. Table A2 (in Appendix A available in the Supplementary Materials) reports the \( t \)-tests of the comparison of the attrition rates for each pair of treatment groups.

29. Angrist and Pischke (2008) formally prove this claim with Theorem 4.4.1. Given the features of Groups B, C and A, there is no simple way to disentangle the treatment effect due to the specific features of the adherence support provided.

30. I control for individual characteristics, such as education, gender, income, marital status, population group, household assets, household members and BMI.

31. The CD4 count is a proxy measure of the individual response to treatment.

32. I assume that the changes in the risky sexual behaviour of ‘untreated’ individuals (i.e. those who adhere less to the HIV therapy) are unrelated to the individuals who are treated more (i.e. those who adhere to HIV therapy more). Further, I assume that the nutritional supplement and the peer adherence support influence sexual activity only via changes in the individual CD4 count. Finally, I assume that the more one receives nutritional supplementation or adherence counselling support, the more one takes up his or her HIV therapy.

33. Though this test is supportive of the procedure used in the identification strategy, the method is not foolproof: such over-identification tests might not lead to a rejection even when not all instrumental variables are valid.

34. For both specifications, the F-statistics are well above 10.

35. As a robustness check (not reported), I also estimate specification (2) only using Treatment Groups A and C; the pattern of the results based on this robustness exercise is very similar to the results reported in Table 6.

36. For some of the patients for whom I lack CD4 data, I proxy treatment status when I observe a 10 per cent change in BMI between time points.

37. See Bunnell et al. (2006); Lakdawalla et al. (2006) analyse the impact of highly active antiretroviral therapy (HAART) on risky sexual behaviour among HIV-positive individuals in the US using arguably exogenous variations of the state portion of the Medicaid insurance generosity toward drug reimbursements as an instrument for treatment status. As in this study’s results, the authors show that a simple OLS method generates a spurious negative correlation between HAART and unsafe sexual activity (although I find this negative relationship only with the condom use variable). With the IV method, they find a positive relationship between access to treatment and risky behaviours.

38. Even if there is a random measurement error in \( T_e \), the results will be biased downwards, not upwards.

39. First, a female who is in a long-term relationship with one partner is more likely to become pregnant than one who is having several short-term relationships. Second, pregnancy caused by younger males is unlikely to result in marriage or child support. Therefore, younger females who become pregnant might be more likely to abort if the father of the child is also young. Other biological proxy measures, such as sexually transmitted infections (STIs), are significantly more intrusive to collect than self-reported methods. Comprehensive data on STIs were not collected for this sample.
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References

Academy of Science of South Africa. (2007). *HIV/AIDS, TB and nutrition: Scientific inquiry into the nutritional influences on human immunity with special reference to HIV infection and active TB in South Africa* (pp. 1–319). Pretoria, South Africa: Academy of Science of South Africa. doi:10.17159/assaf/0037

Adam, M. A., & Johnson, L. F. (2009). Estimation of adult antiretroviral treatment coverage in South Africa. *South African Medical Journal*, 99(9), 661–667. doi:10.7196/SAMJ.327

Angrist, J. D., & Pischke, J.-S. (2008). * Mostly harmless econometrics: An empiricist’s companion* (1st ed.). Princeton, NJ: Princeton University Press.

Angrist, J. D., Imbens, G. W., & Rubin, D. B. (1996). Identification of causal effects using instrumental variables. *Journal of the American Statistical Association*, 91(434), 444–455. doi:10.1080/01621459.1996.10476902

Bunnell, R., Ekwaru, J. P., Solberg, P., Wamai, N., Bikaako-Kajura, W., Were, W., … Mermin, J. (2006). Changes in sexual behavior and risk of HIV transmission after antiretroviral therapy and prevention interventions in rural Uganda. *AIDS*, 20(1), 85–92. doi:10.1097/01.aids.0000196566.40702.28

Chan, T. Y., Hamilton, B. H., & Papageorge, N. W. (2016). Health, risky behaviour and the value of medical innovation for infectious disease. *The Review of Economic Studies*, 83(4), 1465–1510. doi:10.1093/restud/rdv053

Cluver, L. D., Toska, E., Orkin, F. M., Meinck, F., Hodes, R., Yakubovich, A. R., & Sherr, L. (2016). Achieving equity in HIV-treatment outcomes: Can social protection improve adolescent ART-adherence in South Africa? *AIDS Care*, 28(sup2), 73–82. doi:10.1080/09540121.2016.1179008

Cohen, M. S., Chen, Y. Q., Gamble, T., Hosseinipour, M. C., Kumarasamy, N., … Piotto, J. H. S. (2011). Prevention of HIV-1 infection with early antiretroviral therapy. *New England Journal of Medicine*, 365(6), 493–505. doi:10.1056/NEJMoa1105243

Colecraft, E. (2008). HIV/AIDS: Nutritional implications and impact on human development. *The Proceedings of the Nutrition Society*, 67(1), 109–113. doi:10.1071/S0029665108006095

Delavande, A., & Kohler, H.-P. (2012). The impact of HIV testing on subjective expectations and risky behavior in Malawi. *Demography*, 49(3), 1011–1036. doi:10.1007/s13524-012-0119-7

Delavande, A., Goldman, D., & Sood, N. (2010). Criminal prosecution and human immunodeficiency virus–related risky behavior. *The Journal of Law and Economics*, 53(4), 741–782. doi:10.1086/658506

Department of Health. (2005). *National HIV and syphilis antenatal sero-prevalence survey in South Africa 2004*. Pretoria, South Africa: Department of Health. Online Report, National Department of Health, Directorate: Health Systems Research, Research Coordination and Epidemiology. http://www.health.gov.za/index.php/2014-03-17-09-09-38/reports/category/137-report2004?download=374:national-hiv-and-syphilis-antenatal-sero-prevalence-survey-in-south-africa-2004

Department of Health. (2007). *HIV and AIDS and STI strategic plan for South Africa, 2007*. Pretoria, South Africa: Department of Health. Online Report, National Department of Health, Directorate: Health Systems Research, Research Coordination and Epidemiology. http://www.health.gov.za/index.php/2014-03-17-09-09-38/reports/category/137-report2004?download=374:national-hiv-and-syphilis-antenatal-sero-prevalence-survey-in-south-africa-2004

Dupas, P. (2011). Do teenagers respond to HIV risk information? Evidence from a field experiment in Kenya. *American Economic Review Applied Economics*, 3(1), 1–34. doi:10.1257/app.3.1.1

Evans, W. N., & Graham, J. D. (1991). Risk reduction or risk compensation? The case of mandatory safety-belt use laws. *Journal of Risk and Uncertainty*, 4(1), 61–73. doi:10.1007/BF00057886

Fenton, K. A., Johnson, A. M., McManus, S., & Erens, B. (2001). Measuring sexual behaviour: Methodological challenges in survey research. *Sexually Transmitted Infections*, 77(2), 84–92.

Fenton, M., & Silverman, E. C. (2008). Medical nutrition therapy for human immunodeficiency virus (HIV) disease. In L. K. Mahan & S. Escott-Stump (Eds.), *Krause’s food and nutrition therapy* (pp. 991–1020). Philadelphia, Pennsylvania: W.B. Saunders Company.

Fishbein, M., & Pequegnat, W. (2000). Evaluating AIDS prevention interventions using behavioral and biological outcome measures. *Sexually Transmitted Diseases*, 27(2), 101–110.

Fortson, J. G. (2011). Mortality risk and human capital investment: The impact of HIV/AIDS in Sub-Saharan Africa. *The Review of Economics and Statistics*, 93(1), 1–15. doi:10.1162/REST_a_00067

Friedman, W. H. (2018). Antiretroviral drug access and behavior change. *Journal of Development Economics*, 135, 392–411. doi:10.1016/j.jdeveco.2018.07.011

Godlonton, S., & Thornton, R. L. (2013). Learning from others’ HIV testing: Updating beliefs and responding to risk. *The American Economic Review*, 103(3), 439–444. doi:10.1257/aer.103.3.439

Godlonton, S., Munthali, A., & Thornton, R. (2016). Responding to risk: Circumcision, information, and HIV prevention. *Review of Economics and Statistics*, 98(2), 333–349. doi:10.1162/REST_a_00516

Gong, E. (2015). HIV testing and risky sexual behaviour. *The Economic Journal*, 125(582), 32–60. doi:10.1111/econ.12125
Govender, V., Fried, J., Birch, S., Chimbindi, N., & Cleary, S. (2015). Disability grant: A precarious lifeline for HIV/AIDS patients in South Africa. BMC Health Services Research, 15(1), 227–227. doi:10.1186/s12913-015-0870-8

Greene, W. (2008). Functional forms for the negative binomial model for count data. Economics Letters, 99(3), 585–590. doi:10.1016/j.econlet.2007.10.015

Harrison, G. W., & List, J. A. (2004). Field experiments. Journal of Economic Literature, 42(4), 1009–1055. doi:10.1257/0022051043004577

Hellerlanger, S., Kohler, H. P., Kalilani-Phiri, L., Mkandawire, J., & Armbruster, B. (2011). The reliability of sexual partnership histories: Implications for the measurement of partnership concurrency during surveys. AIDS, 25(4), 503–511. doi:10.1097/QAD.0b013e3283434485

Imbens, G. W., & Angrist, J. D. (1994). Identification and estimation of local average treatment effects. Econometrica, 62(2), 467–475. doi:10.2307/2951620

Johnson, L. F. (2012). Access to antiretroviral treatment in South Africa, 2004–2011. Southern African Journal of HIV Medicine, 13(1), 22–27. doi:10.4102/sajhivmed.v13i1.156

Kaptchuk, T. J. (2001). The double-blind, randomised, placebo-controlled trial: Gold standard or golden calf? Journal of Clinical Epidemiology, 54(6), 541–549. doi:10.1016/S0895-4356(00)00347-4

Keeler, T. E. (1994). Highway safety, economic behavior, and driving environment. The American Economic Review, 84(3), 684–693.

Kennedy, C., O’Reilly, K., Medley, A., & Sweat, M. (2007). The impact of HIV treatment on risk behaviour in developing countries: A systematic review. AIDS Care, 19(6), 707–720. doi:10.1080/09540120701203261

Kremer, M. (1996). Integrating behavioral choice into epidemiological models of AIDS. The Quarterly Journal of Economics, 111(2), 549–573. doi:10.2307/2946687

Lakdawalla, D., Sood, N., & Goldman, D. (2006). HIV breakthroughs and risky sexual behavior. Quarterly Journal of Economics, 121(3), 1063–1102. doi:10.1162/qjec.121.3.1063

MacLehose, R. R., Reeves, B. C., Harvey, I. M., Sheldon, T. A., Russell, I. T., & Black, A. M. (2000). A systematic review of comparisons of effect sizes derived from randomised and non-randomised studies. Health Technology Assessment, 4(34), 1–154.

Mariano, R. S. (2003). Chapter 6: Simultaneous equation model estimators: Statistical properties and practical implications (B. Baltagi, Ed.; 1st ed., pp. 122–143). Hoboken, New Jersey: Blackwell Publishing. doi:10.1002/9780470996249.ch7

McLaren, Z. M. (2015). Equity in the national rollout of public AIDS treatment in South Africa 2004–08. Health Policy and Planning, 30(9), 1162–1172. doi:10.1093/heapol/czu124

Mehchouan, S. (2007). Risky sexual behavior, testing, and HIV treatments. Forum for Health Economics & Policy, 10(2), 1–49. doi:10.2202/1558-9544.1064

Mensch, B. S., Hewett, P. C., & Erulkar, A. S. (2003). The reporting of sensitive behavior by adolescents: A methodological experiment in Kenya. Demography, 40(2), 247–268. doi:10.1353/dem.2003.0017

Minnis, A. M., Steinr, M. J., Gallo, M. F., Warner, L., Hobbs, M. M., der Straten, A. V., … Padian, N. S. (2009). Biomarker validation of reports of recent sexual activity: Results of a randomized controlled study in Zimbabwe. American Journal of Epidemiology, 170(7), 918–924. doi:10.1093/aje/kwp219

Murphy, E. L., Collier, A. C., Kalish, L. A., Assmann, S. F., Para, M. F., Flanigan, T. P., … Nemo, G. J. (2001). Highly active antiretroviral therapy decreases mortality and morbidity in patients with advanced HIV disease. Annals of Internal Medicine, 135(1), 17–26. doi:10.7326/0003-4819-135-1-200107030-00005

Newey, W. K. (1990). Semiparametric efficiency bounds. Journal of Applied Econometrics, 5(2), 99–135. doi:10.1002/jae.3950050202

Oster, E. (2012). HIV and sexual behavior change: Why not Africa? Journal of Health Economics, 31(1), 35–49. doi:10.1016/j.jhealeco.2011.12.006

Palen, L.-A., Smith, E. A., Caldwell, L. L., Flisher, A. J., & Vergnani, T. (2008). Inconsistent reports of recent sexual activity: Results of a randomized controlled study. AIDS Care, 15(2), 161–172. doi:10.1080/09540120701203261

Padian, N. S. (2009). … Challenges in applying the biomedical standard to behavioral headache research. American Journal of Epidemiology, 135(10), 1601–1612. doi:10.1093/aje/kwp219

Palla, A. D., Shapira, G., & Todd, P. E. (2014). How beliefs about HIV status affect risky behaviors: Evidence from Malawi. Journal of Applied Econometrics, 29(6), 944–964. doi:10.1002/jae.2342

Peltzman, S. (1975). The effects of automobile safety regulation. Journal of Political Economy, 83(4), 677–725. doi:10.1086/260352

Rains, J. C., & Penzien, D. B. (2005). Behavioral research and the double-blind placebo-controlled methodology: Challenges in applying the biomedical standard to behavioral headache research. Headache, 45(5), 479–486. doi:10.1111/j.1526-4610.2005.05099.x

Robinson, J., & Yeh, E. (2011). Transactional sex as a response to risk in Western Kenya. American Economic Journal: Applied Economics, 3(1), 35–64. doi:10.1257/app.3.1.35

Rosen, S., Fox, M. P., & Gill, C. J. (2007). Patient retention in antiretroviral therapy programs in Sub-Saharan Africa: A systematic review. PLOS Medicine, 4(10), e298. doi:10.1371/journal.pmed.0040298

Sachdeva, R. K., Sharma, A., Wanchu, A., Dogra, V., Singh, S., & Varma, S. (2011). Dietary adequacy of HIV infected individuals in north India—A cross-sectional analysis. The Indian Journal of Medical Research, 134(6), 967–971.
Sood, N., Wagner, Z., & Wu, Y. (2015). The impact of insurance on HIV testing. *American Journal of Health Economics, 1*(4), 515–536. doi:10.1162/AJHE_a_00028

South African National AIDS Council Trust. (2016). South Africa Global Aids Response Progress Report (GARPR) 2015. In *The South African Aids Council Trust Report* (South Africa Global AIDS Response Progress Report; pp. 1–69). South African National AIDS Council Trust. http://sanac.org.za/download/563/resources/2906/garpr_report-high-res-for-print-june-15-2016.pdf.

Streeck, H., Li, B., Poon, A. F., Schneidewind, A., Gladden, A. D., Power, K. A., … Allen, T. M. (2008). Immune-driven recombination and loss of control after HIV superinfection. *Journal of Experimental Medicine, 205*(8), 1789–1796. doi:10.1084/jem.20080281

Tavory, I., & Swidler, A. (2009). Condom semiotics: Meaning and condom use in rural Malawi. *American Sociological Review, 74*(2), 171–189. doi:10.1177/000312240907400201

Thirumurthy, H., Pop-Eleches, C., Habyarimana, J. P., Goldstein, M., & Zivin, J. G. (2012). Behavioral responses of patients in AIDS treatment programs: sexual behavior in Kenya. *Forum for Health Economics & Policy, 15*(1), 1–32. doi:10.1515/1558-9544.1230

UNAIDS. (2013). *Access to antiretroviral therapy in Africa*. [http://www.unaids.org/sites/default/files/media_asset/20131219_AccessARTAfricaStatusReportProgressTowards2015Targets_en_0.pdf](http://www.unaids.org/sites/default/files/media_asset/20131219_AccessARTAfricaStatusReportProgressTowards2015Targets_en_0.pdf)

UNAIDS. (2015). *Global AIDS response progress report 2012 Republic of South Africa*. [http://www.unaids.org/sites/default/files/country/documents/ce_ZA_Narrative_Report.pdf](http://www.unaids.org/sites/default/files/country/documents/ce_ZA_Narrative_Report.pdf)

UNAIDS. (2017). *Ending AIDS: Progress towards the 90-90-90 Target*. [https://www.unaids.org/sites/default/files/media_asset/Global_AIDS_update_2017_en.pdf](https://www.unaids.org/sites/default/files/media_asset/Global_AIDS_update_2017_en.pdf)

UNAIDS. (2018). *UNAIDS data 2017* (pp. 1–248). [https://www.unaids.org/sites/default/files/media_asset/20170720_Data_book_2017_en.pdf](https://www.unaids.org/sites/default/files/media_asset/20170720_Data_book_2017_en.pdf)

UNAIDS. (2019). *UNAIDS data 2019* (pp. 1–471). United Nations Programme on HIV/AIDS (UNAIDS). [https://www.unaids.org/sites/default/files/media_asset/2019-UNAIDS-data_en.pdf](https://www.unaids.org/sites/default/files/media_asset/2019-UNAIDS-data_en.pdf)

Vergidis, P. I., Falagas, M. E., & Hamer, D. H. (2009). Meta-analytical studies on the epidemiology, prevention, and treatment of human immunodeficiency virus infection. *Infectious Disease Clinics of North America, 23*(2), 295–308. doi:10.1016/j.idc.2009.01.013

Young, A. (2005). The gift of the dying: The tragedy of AIDS and the welfare of future African generations. *The Quarterly Journal of Economics, 120*(2), 423–466. doi:10.1093/qje/120.2.424