

Does AIDS Treatment Stimulate Negative Behavioral Response? A Field Experiment in South Africa.

Plamen Nikolov*
Harvard University†

October 16, 2011

Preliminary - please do not quote or cite without permission.

*I am grateful to Josh Angrist, Frikkie Booysen, Lorenzo Casaburi, Amitabh Chandra, Jessica Cohen, David Cutler, Erica Field, Roland Fryer, Seema Jaychandran, Lawrence Katz, Rodrick Kisenge, Zoe McLaren, Sendhil Mullainathan, Emily Oster, Tristan Zajonc and seminar participants at The Harvard Development Lunch, The EIEF 11th Workshop in Economic Theory and Econometrics, the European Economics Association Annual Meeting (2011), Hewlett Foundation PRB Annual Conference in Marseilles (2011) and the University of Dar es Salaam Economics Department Seminar for extremely helpful comments. Chantell Jacqueline de Reuck, Anja Pienaar, Theresa Neuhoff and Musilo Machere provided scrupulous care collecting the field data and staff at the University of the Free State's (UFS) Centre for Health Systems Research & Development (CHSR&D) and Department of Economics provided outstanding administrative assistance. I thank The Harvard Institute for Quantitative Social Studies, Harvard University Weatherhead Center for International Affairs, The Harvard Global Health Institute for funding. Overall project funding through The World Bank; The Programme to Support Pro-Poor Policy Development (PSPPD) in South Africa, a partnership programme of the South African Presidency and the Delegation of the European Union (EU), New Frontiers in Poverty Alleviation Research Cluster and Faculty of Economic & Management Sciences at the University of the Free State (UFS), the Health Economics and AIDS Research Division (HEARD) at the University of Kwazulu-Natal made the project possible. All views, opinions and errors are my own.

†National Bureau of Economic Research, 1050 Massachusetts Avenue, Third Floor, Cambridge, MA 02138. E-Mail: pnikolov@fas.harvard.edu.

1 Introduction

The AIDS epidemic has exacted a terrible toll worldwide and Sub-Saharan Africa has borne a disproportionate burden of the virus. In particular, South Africa’s HIV/AIDS adult prevalence rate of 18.3%¹ is among the highest in the world. While prevalence rates in Sub-Saharan Africa have been projected to increase (Young, 2005), free AIDS treatment has been rapidly extending to most of the continent. This rapid diffusion of HIV drugs has transformed the disease from a death sentence into a manageable condition. South Africa’s treatment program is the largest in the world² even though it has treated only a fraction of its *HIV+* individuals. Treatment provides enormous mortality benefits to infected individuals³ but because it immunologically insulates people, it could also stir potential perverse behavioral responses subsequent to provision. Using a field experiment conducted in South Africa, I examine whether changes the individual economic incentives (due increased longevity and immunological risk-insulation) induce subsequent unsafe sexual behavior by *HIV+* patients. Specifically, I focus on two key outcomes: (1) *HIV+* patient’s own sexual behavior, (2) *HIV-* family members’ sexual behavior.

The first major contribution of this paper relates to testing the theoretical possibility that treatment could induce perverse behavioral responses. This prediction is the so-called *Peltzman effect*, which posits that that people offset safety measures by increasing unsafe behaviors (Peltzman, 1975). I empirically examine whether AIDS treatment induces patients’ moral hazard⁴. The concept underlying our test is simple: when the price of more unsafe sexual behavior (e.g., the probability of dying) is lowered, people choose to engage in more unsafe sex. Peltzman’s (1975) economic framework in the context of seatbelts – that drivers exhibit a demand for accident risk that is inversely related to risk price – formalizes this testable prediction. Any activity that alters the probability of a fatality, i.e. risk price, will “induce individual responses working in the opposite direction” (Peltzman, p. 698). One objection to the proposed argument is that unsafe sex is not in any sense a “commodity” that people would ever choose to purchase. But despite the risks associated with them, unsafe sex and recklessness are “commodities” in the sense that people seem to engage in them and thus “want” them. Choosing to engage in more unsafe sex or more reckless behavior is tantamount to choosing more accidents, at least in a probabilistic sense.

Treatment could induce negative behavioral responses for both *HIV+* and *HIV-* individuals. From the

¹Figure 1 details epidemic statistics within South Africa.

²The government began providing free ARV drugs in July 2004 – much later than most developing countries – but scaled up rapidly, increasing patient enrollment to about 1.4 million by May 2011 (AVERT, 2011).

³See Hammer et al. (1997), Hogg et al. (1998), Palella et al. (1998), Florida et al. (2002), Laurent et al.(2002), Marins et al. (2003), Koenig et al. (2004) for evidence in high-income countries and Coetzee et al. (2004), Wools- Kaloustian et al. (2006).Coetzee et al. (2004), Bekker et al. (2006), Sterne et al. (2005), Cole et al. (2003), Cole et al. (2005), Fairall et al. (2008) for evidence in low-income countries.

⁴I postulate a framework where (1) objectives of principal and agent conflict, (2) there is non-contractible information, (3) the action chosen can not be inferred by the outcome alone, and (4) different actions have different utility costs to the agent and the principal cannot observe the action the agent takes.

perspective of an *HIV+* individual, availability of free AIDS treatment lowers the cost of future dangerous behavior because treatment provides an immunologic benefit by boosting the CD4 count. From the perspective of an *HIV+* individual, even though infected, one still has an incentive to refrain from future unsafe sex (possible reinfection with more aggressive HIV strains, either because of *co-infection* or *superinfection*⁵ decreases CD4 count even faster and significantly decreases life expectancy⁶). However, ARV provision partially insulates *HIV+* individuals from health and immunology risk and patients may, in theory, start behaving differently than if they were fully exposed to the future health risk. From the perspective of an *HIV-* individual, living with a patient and being able to observe that a patient achieves a dramatic health improvement subsequent to treatment may engender complacency. Such perceptions could stir current risky sexual behavior of family members who are currently uninfected.

This potential positive relationship between treatment and risky behavior has important implications for welfare and behavior. HIV treatment may not have unambiguous benefits for the uninfected. While the cost of being infected falls, the risk of infection rises with the stock of *HIV+* people, who now live longer and more sexually active lives. Treatment provision can reduce expected welfare among the uninfected if the cost of higher infection risk more than offsets the welfare gain from the reduced cost of infection.

A second major contribution of the paper relates to its robust identification strategy. Because we use a field experiment, we can identify true *causal effects*. In general, existing epidemiology studies have found no association between treatment and unsafe sexual activity (Hethcote and Yorke, 1984; Over and Piot, 1993). There is anecdotal evidence of fatalism among some IV drug users and homosexuals in developed countries (Marder, 2005). Crepaz, Hart and Marks (2004) explore the overall effect of treatment on unsafe sex without exploring the contribution of each submechanism of behavior change. They find no support to reject the hypothesis that AIDS treatment influences unsafe sexual activity. However, the robustness of their causal methods is questionable. Their analysis relies on OLS estimates. Magnitude of selection bias and reverse causality with nonexperimental estimators can often be quite large in practice.

A final contribution relates to our ability to identify the magnitude of sub-mechanisms driving the behavior change for the outcome of interest. Several mechanisms underlie the empirical analysis of the field experiment. First, holding testing likelihood constant, ARVs lower the cost of getting HIV: this is the direct and most straightforward effect (let's call this margin (a)). However, as treatment quality improves, HIV prevalence is expected to rise: this stems from the increase in the proportion of healthier *HIV+* individuals present in the market for risky exposures: this is the indirect effect (call this margin (b)). ARVs also reduce

⁵Co-infection is infection with two separate strains either simultaneously or within a brief period of time before infection with the first strain is established; Superinfection is sequential infection with a heterologous strain after an immune response has been established to the initial strain.

⁶See Jost et al. (2002); Koelsch et al. (2003); Gottlieb et al. (2004, 2007); Grobler et al. (2004); Smith et al. (2004); Jurriaans (2008).

the average concentration of virus, thus lowering the risk of transmission per risky act (margin (c)). Finally, AIDS treatment improves physical health and individual capacity to carry out sexual activity regardless of any other mechanism. Margins (a), (c) and (d) suggest more risk-taking while margin (b) suggests less: which among these is theoretically ambiguous *ex ante*.

The structure of the paper is as follows. Section two describes the conceptual framework guiding the comparative statics. Section three describes the experimental design, data sources and provides some summary statistics. Section four describes the empirical strategy and main results. Section five offers some preliminary conclusions and interprets the results in terms of the estimated benefits of mortality reduction compared to the costs of increased unsafe sexual activity.

2 Theoretical Framework

We outline a simple theoretical model to analyze sexual behavior choice in a world with ARV treatment following very closely Kremer (1996) and Lakdawalla, Sood and Goldman (2006)⁷.

An individual lives for a maximum of two periods. γ_1 represents HIV prevalence among the sexually active population. We assume that all individuals engage and value risky sexual activity. *HIV+* individuals engage in risky behavior σ_{pos} . They receive the corresponding level of per-period utility $w(\sigma_{pos})$ ⁸. With probability $(1-p)$, an *HIV+* individual dies or gets too sick to be able to engage in sex. For a sexually active infected individual $w(\sigma_{pos})/(1-p\beta)$ represents his or her lifetime utility; β is the period discount factor.

Uninfected individuals derive utility $u(\sigma_{neg})$ from risky behavior σ_{neg} . The probability of infection ϕ rises in a person's own risky behavior σ_{pos} , the prevalence of HIV γ_1 , and the risky behavior of the *HIV+* individuals $\sigma_{pos} : \phi(\sigma_{neg}; \sigma_{pos}, \gamma_1)$. r_{neg}^* represents the equilibrium level of risky behavior among the uninfected and γ_2 is next period's prevalence. The uninfected person lifetime utility can be written as⁹:

⁷This model differs both from traditional epidemiological models, which take behavior as independent of treatment, and from the few attempts to introduce behavioral considerations into epidemiology, which does not formally model how decisions about the rate of partner change depend on the availability of treatment. Models without behavioral response typically generate a unique stable steady state. Analyses in which behavior is an *ad hoc* declining function of prevalence typically imply that anti-AIDS policies, such as prevention efforts or imperfect vaccines, will be counteracted by behavioral response, at least partially, and possibly more than fully [Philipson and Posner 1993; Castillo-Chavez and Hadeler 1994]. If these analyses implicitly or explicitly assume that behavior is a function of lagged prevalence, they will generate cycles in prevalence [Avery, Heymann, and Zeckhauser 1994; Velasco-Hernandez, Brauer and Castillo-Chavez 1994]. In contrast, this paper's framework formally models the way in which decisions about the rate of partner change depend on the availability of ARV treatment. This modelling suggests that there may be positive feedbacks in behavior, and possibly even multiple steady states. This implies that behavioral response may sometimes decrease the effectiveness of treatment policies.

⁸We assume $w(\cdot)$ to be concave

⁹A single uninfected individual is assumed to have no control over r_{neg}^* .

$$v(\gamma_1) = \max \left\{ u(\sigma_{neg}) + \beta \phi(\sigma_{neg}; \sigma_{pos}, \gamma_1) \frac{w(\sigma_{pos})}{1 - p\beta} + \beta(1 - \phi(\sigma_{neg}; \sigma_{pos}, \gamma_1))v(\gamma_2) \right\} \quad (1)$$

subject to:

$$\gamma_2 = \frac{\gamma_1 p + (1 - \gamma_1) \phi(\sigma_{neg}; \sigma_{pos}, \gamma_1)}{\gamma_1 p + (1 - \gamma_1)} \quad (2)$$

The value function $v(\gamma_1)$ is convex decreasing¹⁰. Therefore, the fixed point of the mapping must also be convex decreasing. We provide assumptions that we need for the uniqueness and existence of a steady state.

We detail the assumptions in **Appendix A**:

Most importantly, the unique pair of *steady-state risk-taking* and disease prevalence levels satisfies:

$$\frac{du}{d\sigma_{neg}} - \beta \frac{\partial \phi}{\partial \sigma_{neg}} \left(v - \frac{w(\sigma_{pos})}{1 - p\beta} \right) = 0 \quad (3)$$

$$\gamma_1(1 - p) - \phi(\sigma_{neg}; \sigma_{pos}, \gamma_1) = 0 \quad (4)$$

2.1 Comparative Statics

Each equation defines a relationship between and σ_{neg} (risky behavior by *HIV-* individuals), γ_1 (*HIV* prevalence among the sexually active population), indexed by the two correlates of *HIV+* individual's health, p (probability of living to engage in sex) and σ_{pos} (risky behavior by *HIV+* individuals).

- Equation (3) characterizes the relationship between overall prevalence and individual risk-taking: uninfected individuals are more cautious when faced with higher disease prevalence. Breakthroughs in treatment shift this curve up by improving the welfare of the *HIV+* (i.e., raising (σ_{pos}, p)), which

¹⁰Because its associated functional mapping satisfies Blackwell's sufficient conditions for a contraction mapping and because that mapping takes a convex decreasing function and produces another convex decreasing function (cite relevant Theorem from Stokey and Lucas, 1989): if v is convex decreasing, and ϕ is concave increasing in γ_1 , $\beta \phi * [(w)/(1 - p\beta) - v(\gamma_2)] + \beta v(\gamma_2)$ is convex decreasing in γ_1 .

makes the uninfected more willing to risk infection at a given level of exposure risk. This translates to **Proposition 1** below.

On the other hand, the “*Steady-State Risk*” relationship, defined by Equation (4), characterizes the steady-state relationship between risk-taking and prevalence: more risk-taking results in higher prevalence. Break-throughs shift the curve, which is defined by this question, down, because a given level of prevalence can be supported by less risk-taking on the part of the uninfected. In other words, increases in survival and sexual activity among the *HIV+* multiply the effects of risky behavior among the uninfected, so that less of it is needed to sustain the disease at a given level.

- $\frac{d\sigma_{pos}}{dp} > 0$. If the probability of dying before reaching the next period is lower, the number of sexual partners increases. From (3), $\gamma_1(1 - p) - \phi(\sigma_{neg}; \sigma_{pos}, \gamma_1) = 0$. From this equation, if p increases $\phi(*; \sigma_{pos}, *)$ decreases. From previous assumptions imposed on $\phi(\cdot)$, it follows that σ_{pos} increases. This translates to **Proposition 2** below.

This very simple framework formalizes several intuitions. Equation (3) can illustrate two forces that combine to increase prevalence: there are more sexually active *HIV+* people who can spread infection; this is reinforced, because the uninfected take more risk at any given level of prevalence. However, the effects on precaution are offsetting and ambiguous: the lower cost of infection encourages risk-taking, while higher prevalence discourages it.

2.2 Testable Hypotheses

- **Proposition 1.** On average, *HIV-* individuals, in a household cohabited by a *HIV+* patient, should increase their risky sexual behavior.

The household survey includes household-wide questions on *HIV-* individuals. I can test the second proposition using both self-reported and indirect measures of sexual activity for *HIV-* household members.

- **Proposition 2.** *On average, HIV+ individuals should increase their unsafe sexual practices as ARV treatment increases.*

Because the baseline and follow-up household surveys as well as the patient survey include sexual behavior questions, I can test this hypothesis using both self-reported and indirect measures of sexual activity.

3 Background and Study Design

The experimental intervention¹¹ targeted 648 ARV households at 12 sites in South Africa’s Free State starting in October 2007¹² and was started on top of the national South Africa ARV Programme described below. The criteria for ARV initiation in adults and adolescents was $CD4 < 200$ cells/mm³ irrespective of stage **or** WHO Stage IV AIDS-defining illness, irrespective of CD4 count, **and** expressed patient willingness and readiness to adhere to ARV treatment.

3.1 South Africa’s Treatment Program

The South African National Department of Health (DOH) launched the national “roll-out” plan for anti-retroviral treatment (ARV) via the public sector in 2003 because of dramatic drop in costs for triple-drug therapy strong causal evidence of the effectiveness of ARV in slowing the progression of AIDS (Concorde Coordinating Committee, 1994; Havlir, 1998, Lange et al, 2004; Zewdie, Lange and Kuritzkes, 2004). Its aim was to achieve universal ARV access (an estimated 1.4 million people in need of ARVs at that stage) within 5 years (Department of Health, 2003). Although the OP’s stated aim was to provide comprehensive HIV care in an integrated fashion, the roll-out was delivered as a vertical programme with dedicated funding, staffing and administration, and closely controlled national accreditation of ARV sites¹³.

From 2005, the provincial scale-up of ART programmes across South Africa’s nine provinces began though at a staggering pace. In the absence of guidelines, norms or standards issued by the National Department of Health, the Free State developed its own systems for scale-up (Schneider et al. 2010: 13). The province struggled to initiate patients onto ARVs quickly enough to meet the high demand for treatment, and its model of ARV provision through a small number of centrally located clinics meant that treatment remained

¹¹The treatment regimes used in the public service are the following: regimens 1a (d4T /3TC / efavirenz), 1b (d4T / 3TC / NVP), and 2 (AZT /ddI / lopinavir / ritonavir). The treatment readiness assessment, monitoring processes and other protocols are described in detail in the relevant guidelines (National Department of Health, 2004: 3).

¹²The Centre for Health Systems Research & Development University of the Free State has collected baseline and follow-up information on the subjects has been collected since October 2007

¹³For more details on program roll-out, see Figure 2 and Tables 2 and 3 of **Appendix C**.

inaccessible for many. This was partly the result of the laborious accreditation process for ARV sites, and partly because of human resource shortages and infrastructural constraints. The concentration of services in urban centres meant that many patients had to travel long distances to access care, and lengthy waiting lists at central facilities indicated the high unmet demand for ARVs (Ingle et al. 2010: 3). By the middle of 2008, 568,000 HIV-infected patients were receiving ART in South Africa, with the public health sector accounting for 79% of this total. Based on the 2008 Department of Health criteria for defining anti-retroviral eligibility (CD4+ count $< 200/\mu L$ or World Health Organization [WHO] stage IV), anti-retroviral coverage in adults was only 40.2% in 2008. Moreover, coverage varied significantly between the provinces, with the Free State, the subject of this study, ranking last (25.8%).

3.2 The Free State Program

In the Free State province, the first patients started receiving ARV treatment in June 2004. ARV treatment was available at some thirty-one primary health clinics or community health centers¹⁴. After testing HIV positive at any Free State clinic, patients were referred to nurse-run assessment sites. Patients with World Health Organization (WHO) stage 4 or a CD4 cell count of 200 cells/ μL or less were referred to a physician at a treatment site for possible initiation of HAART. For patients with active tuberculosis or other serious opportunistic infections, HAART was deferred until they were clinically stable or until the intensive phase of tuberculosis treatment was completed. Of patients receiving HAART, 68.3% started treatment with stavudine, lamivudine, and efavirenz, and 29.7% started treatment with stavudine, lamivudine, and nevirapine. Cotrimoxazole prophylaxis was indicated if the CD4 cell count was 200 cells/ μL or less or if WHO stage 3 or 4 disease was present. By December 31, 2006, the number of patients on ARV treatment in Free State stood at approximately 12,000 according to officials from the Free State Department of Health. Because the Free State is among the poorest regions in South Africa (Ardington et al, 2005) and one with the highest prevalence and incidence rates of HIV infection (Shisana et al, 2005; Dorrington et al, 2006; National Department of Health, 2007b), fiscal capacity to carry out an effective ARV roll-out was most acute.

3.3 Study Design

We conducted the study in twelve phase I ARV assessment sites in the Free State province¹⁵.

¹⁴ For more details on the Free State program, see Figures 3-4 and Tables 5 and 6.

¹⁵ Sites where ARV treatment first became available when the ARV treatment program was launched in the Free State province in 2004.

Table 1 and Figure 2 (**Appendix B**) respectively detail the list of specific primary health clinics (*PHC*) or community health centers (*CHC*) facilities and their location in the study in the Free State province. We track 648 households with a member currently on ARV treatment. Upon enrollment, participants were randomized into one of four groups of households, three of which include patients on ARV treatment (Table 2 in **Appendix B**).

1. ARV provision only (**Group A**),
2. ARV provision with encouragement support (**Group B**),
3. ARV provision with encouragement support and a nutritional supplementation. (**Group C**).

The study also randomly selects households from the general community served by the selected health facility, excluding households where someone is known to receive ARV treatment (n=180) [**Group D**]

3.4 Overview of outcomes

- **Own Sexual Behavior:** Sexual behavior is measured using a variety of self-report measures: reported number of regular and non-regular partners, and reported frequencies of protected and un-protected intercourse with regular and non-regular partners.
- **Family Member Sexual Behavior:** Sexual behavior is measured using a variety of self-report measures: reported number of regular and non-regular partners, and reported frequencies of protected and un-protected intercourse with regular and non-regular partners.
- **Fertility:** We capture the number of children born per female after the study enrollment.

4 Identification Strategy

4.1 Effects on Sexual Behavior Outcomes

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

$$S_i = \varsigma_1 T_i + \varsigma_2 X_i + \epsilon_i \tag{5}$$

where S_i is a measure of sexual behavior of individual i , T_i is the ARV provision status for the same individual and X_i is a set of individual and cluster-level controls. To identify true causal effects of the research questions outlined in 2.2, the relevant counterfactual to *HIV+* on treatment (i.e. Group A) (treatment effect on outcome for this group is $E[S_i|T_i = 1]$) is individuals from Group D (outcome in absence of treatment for this group is $E[S_i|T_i = 0]$). However, Group D while randomly picked, it is not experimentally assigned (i.e. ARV treatment was not denied to them), so simply comparing the observed sexual activity differential by treatment status is a biased measure of the effect of treatment on the treated, because $E[S_{0i}|T_i = 1] - E[S_{0i}|T_i = 0]$ (the selection into treatment bias) is unlikely to be zero. Naïve OLS estimation of (5) in practice will likely suffer from a number of econometric issues (selection, sorting, omitted variable bias and possibly reverse causality).

While our main identification route for estimating causal effects of treatment is the encouragement design we outline below, we also detail two alternative strategies for estimating the behavioral response to free ARV provision in **Appendix E**. We do this mainly for robustness reasons.

4.2 Encouragement Design Experiment

To overcome estimation problems plaguing the OLS method, we employ an instrumental variables (*IV*) strategy exploiting the encouragement design experiment. The encouragement design is a special case of an experimental design used in situations with little control over subjects' compliance or when randomizing into treatment arms might be unethical. The key idea is that instead of randomizing the application of the intervention itself, what is randomized is encouragement to receive the treatment. Although treatment remains endogenous, for such cases, Imbens and Angrist (1994) and Angrist et al. (1996) showed that econometric instrumental variables (*IV*) methods can be interpreted as estimating a well-defined causal effect under the potential outcomes approach to causal inference advocated by Rubin (1974, 1978, 1990b), often referred to as the Rubin Causal Model (Holland, 1986)¹⁶.

Because of designing a variable (the instrument, which we call Z_i), that is correlated with the causal variable of interest, T_i , but uncorrelated with any other determinants of the dependent variable, we can obtain unbiased estimates of both the encouragement and the intervention itself^{17,18}. We can use the encouragement

¹⁶The IV estimand is predicated on the notion that the first stage is not zero, but this is something we can and do check in the data. We also consider the consequences of econometric 'exclusion' restrictions that disallow, for various subpopulations, direct links between assignment and outcome other than through the effect of assignment on the treatment received.

¹⁷This is shown formally by Theorem 4.4.1 in Angrist and Pischke (2009).

¹⁸ Here, the phrase "uncorrelated with any other determinants of the dependent variables" is like saying $Cov(\text{error term}; Z_i) =$

support provided (present in both Group B and Group C) as an instrument Z_i for treatment take-up. In particular, we instrument for treatment status using indicator variables for whether the person is getting an encouragement support (i.e. whether individual is in Group B) or is getting encouragement support + nutritional intervention (i.e. individual falls in Group C):

$$T_{it} = \alpha_i + \beta_1 Encouragement_{it} + \beta_2 EncouragementAndNutrition_{it} + \varepsilon_{it} \quad (6)$$

where T_{it} is the actual treatment status.

For each person i we observe: a binary variable $Encouragement_{it}$, the ‘assignment’ or ‘encouragement’, equal to one if patient i received encouragement support visitor bi-weekly and zero otherwise; a binary variable $EncouragementAndNutrition_{it}$, equal to one if patient i received encouragement support visitor bi-weekly and nutritional supplement and zero otherwise; a binary variable T_{it} , the ‘treatment’, equal to 1 if person i received the ARV treatment and 0 otherwise; a binary outcome S_i , equal to the number of sexual partners or 1, which we define as having used a condom during the last sexual encounter, and 0 otherwise. The vectors $Encouragement$, $EncouragementAndNutrition$, T , S and are N -dimensional vectors with it th elements equal to $Encouragement_{it}$, $EncouragementAndNutrition_{it}$, T_{it} , S_i , respectively. The $N \times Z$ matrix X_{obs} has it th row equal to $(X_{i1}^{obs}, X_{i2}^{obs})$. For simplicity, we assume that each patient has a distinct encouragement supporter, so that i indexes distinct doctor–patient pairs. Table 6 (**Appendix D**) presents some summary statistics for the sample, classified by assignment, $Encouragement_{it}$, $EncouragementAndNutrition_{it}$, and treatment status, T_{it} . As can be seen in Table 6, the randomization of the assignment leads to the pretreatment variables being closely balanced in the two subsamples defined by assignment¹⁹.

The conventional *ITT* approach to estimation of treatment effects compares outcomes by assignment, that is, by the receipt of the encouragement by the patient, ignoring the actual receipt of treatment, that is, ignoring the receipt of the ARV drugs. In our case the ‘assignment’ is merely an encouragement to take the treatment, so that nonencouraged patients may end up receiving the treatment, but this does not compromise the validity of standard methods for estimating *ITT* effects, which rest on the randomization of the encouragement groups.

0; or, equivalently, Z_i is uncorrelated with ε_i .

¹⁹ The randomization does not, however, imply that the pretreatment variables are balanced in the subsamples defined by the actual treatment status.

Our experiment partitions the population of patients by ‘compliance’ behavior (the same as the encouragement). The combination of responses to the two assignments defines the compliance behavior of unit i , which we denote by C_i :

$$C_i = \left\{ \begin{array}{lll} c & (\text{complier}) & T_i(z) = z \quad z = 0, 1 \\ n & (\text{never-taker}) & T_i(z) = 0 \quad z = 0, 1 \\ a & (\text{always-taker}) & T_i(z) = 1 \quad z = 0, 1 \\ d & (\text{defier}) & T_i(z) = 1 - z \quad z = 0, 1 \end{array} \right\}$$

We observe the compliance behavior only partially, through the response to the actual assignment, $T_i^{obs} = T_i(Z_i^{obs})$. We do not observe the response to the alternative assignment, $T_i^{mis} = T_i(1 - Z_i^{obs})$. Because the type of a unit is a function of both compliance under assignment to the treatment and compliance under assignment to control, which we can never jointly observe, we generally cannot know a unit’s type, merely that the unit belongs to the subset of types consistent with its observed compliance behavior²⁰. Let $\mathbb{C}(t) = \{i | C_i = t\}$ for $t \in \{c, n, a, d\}$; \mathbb{C} is the \mathbb{N} component vector with i th element C_i , and N_t is the number of units of type t .

We define for $z = 0, 1$ the potential outcomes $S_i(z, T_i(z)) : Y_i(z, T_i(z))$ is equal to 1 if given assignment z and given receipt of treatment $T_i(z)$, unit i is hospitalized, and 0 otherwise; S is the $N \times 2$ matrix with i th row equal to $S_i(0, T_i(0)), S_i(1, T_i(1))$. Using this notation, the *ITT* effect of assignment on the outcome can be defined as the weighted average

$$ITT = \sum_{t \in \{c, n, a, d\}} N_t \cdot ITT_t / N,$$

where, for $t \in \{c, n, a, d\}$,

$$ITT_t = \sum_{i \in \mathbb{C}(t)} [S_i(1, T_i(1)) - S_i(0, T_i(0))] / N_t.$$

is the average *ITT* effect of Z_i^{obs} on S for each of the four subpopulations defined by compliance behavior, and N_t/N is the weight assigned to ITT_t . We observe for each unit i the actual assignment Z_i^{obs} , the actual treatment $T_i^{obs} = T_i(Z_i^{obs})$, the actual outcome $S_i^{obs} = S_i(Z_i^{obs}, D_i(Z_i^{obs}))$, and the pretreatment variables X_{i1}^{obs} and X_{i2}^{obs} .

Random assignment of the encouragement support Z implies

²⁰ Z_i^{obs} is the general notation for the N -dimensional vectors with i th elements equal to *Encouragement* _{i} and *EncouragementAndNutrition* _{i} .

$$Pr(Z_i|T_i(0), T_i(1), S_i(0, 0), S_i(0, 1), S_i(1, 0), S_i(1, 1), X_{i1}, X_{i2}) = Pr(Z_i).$$

A few important assumptions that need to hold for estimation to give an unbiased estimate:

Assumption 1 (Ignorability of Treatment Assignment)²¹

$$Pr(Z_i|T_i(0), T_i(1), S_i(0, 0), S_i(0, 1), S_i(1, 0), S_i(1, 1), X_{i1}^{obs}, X_{i2}^{obs}) = Pr(Z_i|X_{i1}^{obs}, X_{i2}^{obs}).$$

Assumption 2 (Monotonicity)²²

For all i ,

$$D_i(1) \geq D_i(0).$$

This assumption rules out the existence of defiers, patients who would receive the ARV treatment if they were not encouraged to do so, but would not receive the ARV treatment if they were encouraged to do so.

Assumption 3 (Exclusion)²³

$$Pr(S_i(1, T_i(1)) = 1|X_{i1}, X_{i2}, C_i = n) = Pr(S_i(0, T_i(0)) = 1|X_{i1}, X_{i2}, C_i = n)$$

and

$$Pr(S_i(1, T_i(1)) = 1|X_{i1}, X_{i2}, C_i = a) = Pr(S_i(0, T_i(0)) = 1|X_{i1}, X_{i2}, C_i = a).$$

We assume that within subpopulations of never-takers with the same values of the co-variates, the distributions of the two potential outcomes are the same; in the second component of the assumption, we assume that within subpopulations of always-takers with the same values of the co-variates, the distributions of the two potential outcomes are the same.

²¹ The IV variable is in fact randomly assigned; those assigned to encouragement receive it, and the rest do not. We provide evidence with balancing tests that this condition indeed holds in **Appendix D**, Table 6.

²² Put simply, the IV variable (encouragement support) cannot have perverse consequences; it cannot make subjects less likely to receive the treatment. This is often a reasonable assumption, but we provide evidence that as the values of the IV variables increase, treatment likelihood also monotonically increases. This can also be verified by checking that treatment intensity is indeed higher in the group that receives the encouragement than in the group that does not.

²³ Put simply, the IV variable has no direct effect on results, except via increasing the probability of receiving treatment. Encouragement support in this case was kept as simple as possible.

With these assumptions, we can estimate the effect of the treatment by adjusting the *ITT* effect by the amount of non-compliance. This yields to the local average treatment effect (*LATE*), computed as:

$$LATE = \frac{ITT}{Compliance\ Rate}$$

where Compliance Rate = Fraction of Subjects that were treated in the treatment group - Fraction of Subjects that were treated in the control group. If the compliance rate is 100%, $LATE = ITT$ ²⁴, we have perfect compliance, and all assigned to the treatment take the treatment and all those assigned to the control do not take the treatment. The compliance rate can be thought of as the fraction of subjects that fall into the sub-population of “compliers”, the group for whom the decision to take treatment was directly affected by the assignment.

4.3 Characterizing the Compliers

Because of the imperfect compliance to treatment, we can only estimate causal effects for a subset of the population of eligible units. Specifically, the program’s average impact is computed from the group of individuals who take the treatment only when encouraged to do so. Therefore, it is important to characterize the complier group in terms of socio-economic characteristics.

The first component of describing the compliers is the size of a complier group: it is easy to measure. This is just the Wald first-stage. We can also tell what proportion of the treated are compliers since, for compliers, treatment status is completely determined by Z_i . Start with the definition of conditional probability:

$$P[T_{1i} > T_{0i} | T_i = 1] = \frac{P[T_i = 1 | T_{1i} > T_{0i}] P[T_{1i} > T_{0i}]}{P[T_i = 1]} = \frac{P[Z_i = 1] (E[T_i | Z_i = 1] - E[T_i | Z_i = 0])}{P[T_i = 1]}$$

In other words, the proportion of the treated who are compliers is given by the first stage, times the probability the instrument is switched on, divided by the proportion treated. We present the proportion of treated with ARVs in Table 9-10.

In terms of describing the actual characteristics, we can describe the distribution of the complier characteristics. What we need to know can be learned from variation in the first stage across co-variate groups. For example, the relative likelihood a complier is a college graduate is given by the ratio of the first stage for college graduates to the overall first stage. More formally, for each characteristic X_i , the we can formally describe the population for each characteristic X_i by:

²⁴This is the effect of the encouragement itself

$$\frac{P[x_{1i} = 1 | T_{1i} > T_{0i}]}{P[x_{1i} = 1]} = \frac{P[T_{1i} > T_{0i} | x_{1i} = 1]}{P[T_{1i} > T_{0i}]} = \frac{E[T_i | Z_i = 1, x_{1i} = 1] - E[T_i | Z_i = 0, x_{1i} = 1]}{E[T_i | Z_i = 1] - E[T_i | Z_i = 0]}$$

4.4 Limitations

An important limitation of the identification strategy used is the local estimate of the *causal effect*. It is local in two ways: both because it comprises only the complier group and local because it represents the causal effect at the average for the treatment provided. We present characteristics of the complier group to the entire population in **Appendix D**.

5 Results, Discussion and Conclusions

We present very preliminary results in **Appendix D** of the paper. We plan to discuss them at the presentation. Based on our specifications treating the endogenous variable as both a dummy (defined by any positive movement in the CD4 count) and continuous variable (using the CD4 bloodcell count), we test our hypotheses on two key outcomes, condom use and number of sexual partners. We find preliminary evidence that access to AIDS treatment does increase unsafe sexual behavior and decreases condom use among *HIV+* individuals but moderately so.

We provide preliminary analyses on both behavioral responses among *HIV+* and *HIV-* family members after AIDS treatment in **Appendix D** below.

References

- [1] Angrist, J., and Jorn-Steffen Pischke (2009). *Mostly Harmless Econometrics: An Empiricists Companion*.
- [2] ANGRIST, J. D., IMBENS, G. W. AND RUBIN, D. B. (1996). Identification of causal effects using instrumental variables (with discussion). *Journal of the American Statistical Association* 91, 444–455.
- [3] AVERT. (2011). HIV and AIDS in South Africa. In *Averting HIV and AIDS*. Retrieved October 6th, 2011, from <http://www.avert.org/aidssouthafrica.htm#contentTable3>.
- [4] Avery, Chris, Jody Heymann, and Richard Zeckhauser, "Individual Choice and the Spread of Infectious Diseases," Working Paper, Kennedy School of Government, 1994.
- [5] Bekker LG, Myer L, Orrell C, Lawn S, Wood R. Rapid scale-up of a community-based HIV treatment service: programme performance over 3 consecutive years in Guguletu, South Africa. *S Afr Med J* 2006;96(4):315-20.
- [6] Beegle, Kathleen. 2005. "Labor Effects of Adult Mortality in Tanzanian Households." *Economic Development and Cultural Change* 53.
- [7] Bhargava A., Bouis H. & Scrimshaw N. (2001). Dietary intakes and socioeconomic factors are associated with haemoglobin concentration of Bangladeshi women. *Journal of Nutrition*, 131, 758-64.
- [8] Bhargava A. & Sargan J.D. (1983). Estimating dynamic random effects models from panel data covering short time periods. *Econometrica*, 51, 1635-1660.
- [9] Blower S. & Farmer, P. (2003). Predicting the public health impact of antiretrovirals: preventing HIV in developing countries. *AIDScience*, 3(11), available <http://www.aidsscience.org/Articles/aidsscience033.htm> [accessed 16 January 2005].
- [10] Castillo-Chavez, Carlos, and K. P. Hadeler, "A Core Group Model for Disease Transmission," Working Paper, Cornell University, 1994.
- [11] Coetzee D, Hildebrand K, Boule A, Maartens G, Louis F, Labatala V, et al. Outcomes after two years of providing antiretroviral treatment in Khayelitsha, South Africa. *AIDS* 2004;18(6):887-95.
- [12] Cole SR, Hernan MA, Robins JM, Anastos K, Chmiel J, Detels R, et al. Effect of highly active antiretroviral therapy on time to acquired immunodeficiency syndrome or death using marginal structural models. *Am J Epidemiol* 2003;158(7):687-94.
- [13] Cole SR, Hernan MA, Margolick JB, Cohen MH, Robins JM. Marginal structural models for estimating the effect of highly active antiretroviral therapy initiation on CD4 cell count. *Am J Epidemiol* 2005;162(5):471-78.
- [14] Concorde Coordinating Committee. (1994). MRC/ANRS randomised double-blind controlled trial of immediate and deferred zidovudine in symptom-free HIV infection. *Lancet*, 343, 871–81.
- [15] Crepaz N., Hart T.A. & Marks, G. (2004). Highly active antiretroviral therapy and sexual risk behavior: A meta-analytic review. *JAMA*, 292(2), 224-236.
- [16] Deb, P., and P. Trivedi (2004). "Provider Networks and Primary Care Signups: Do They Restrict the Use of Medical Services?" Technical report. Hunter College, CUNY.
- [17] Department of Health. Operational Plan for Comprehensive HIV and AIDS Care, Management and Treatment for South Africa 2003. Pretoria: South African Department of Health, 2003.

- [18] Dorrington R.E., Bradshaw D., Johnson L. and Daniel T. (2006). The Demographic Impact of HIV/AIDS in South Africa: National and Provincial Indicators for 2006. Cape Town: Centre for Actuarial Research, South African Medical Research Council and Actuarial Society of South Africa.
- [19] Fairall LR, Bachmann MO, Louwagie GM, van Vuuren C, Chikobvu P, Steyn D, et al. Effectiveness of antiretroviral treatment in a South African program: a cohort study. *Arch Intern Med* 2008 Jan 14;168(1):86-93.
- [20] Floridia, M., V Fragola, CM Galluzzo, G Giannini, MF Pirillo, M Andreotti, C Tomino and S Vella. 2002. "HIV-Related Morbidity and Mortality in Patients Starting Protease Inhibitors in Very Advanced HIV Disease," *HIV Medicine*, 3(2): 75-84.
- [21] Gottlieb GS, Nickle DC, Jensen MA, et al. Dual HIV-1 infection associated with rapid disease progression. *Lancet*. 2004;363(9409):619-622.
- [22] Gottlieb GS, Nickle DC, Jensen MA, et al. HIV type 1 superinfection with a dual-tropic virus and rapid progression to AIDS: a case report. *Clin Infect Dis*. 2007;45(4):501-509.
- [23] Grémy I. & Beltzer, N. (2004). HIV risk and condom use in the adult heterosexual population in France between 1992 and 2001: return to the starting point? *AIDS*, 18, 805-809.
- [24] Grobler J, Gray CM, Rademeyer C, et al. Incidence of HIV-1 dual infection and its association with increased viral load set point in a cohort of HIV-1 subtype C-infected female sex workers. *J Inf Dis*. 2004;190(7):1355-1359.
- [25] Habyarimana, James, Bekezela Mbakile and Cristian Pop-Eleches. 2008. "HIV/AIDS ARV Treatment and Worker Absenteeism: Evidence from a Large African Firm." Georgetown University: Mimeo.
- [26] Hammer, S.M., K. Squires, M. Hughes, J. Grimes, L. Demeter, J. Currier, J. Eron, J. Feinberg, H. Balfour, L. Deyton, J. Chodakewitz and M. Fischl. 1997. "A controlled trial of two nucleoside analogues plus indinavir in persons with human immunodeficiency virus infection and CD4 cell counts of 200 per cubic millimeter or less." *New England Journal of Medicine*, 337:725-733.
- [27] Havlir D.V. & Lange J.M. (1998). New antiretrovirals and new combinations. *AIDS*, 12(Suppl A), S165-S174.
- [28] Hethcote, Herbert W., and J. A. Yorke, "Gonorrhea: Transmission Dynamics and Control," *Lecture Notes in Biomathematics*, LVI (1984), 1-105.
- [29] Hirano, K., G. Imbens, and G. Ridder, (2003), "Efficient Estimation of Average Treatment Effects Using the Estimated Propensity Score," *Econometrica*, 71(4): 1161-1189. July
- [30] Hogg, Robert S., K.V. Heath, B. Yip, K.J. P. Craib, M.V. OShaughnessy, M.T. Schechter, J. S. G. Montaner. 1998. "Improved survival among HIV-infected individuals following initiation of antiretroviral therapy." *Journal of the American Medical Association*, 279: 450-454.
- [31] HOLLAND, P. (1986). Statistics and causal inference. *Journal of the American Statistical Association* 81, 945-970.
- [32] IMBENS, G.W. AND ANGRIST, J. D. (1994). Identification and estimation of local average treatment effects. *Econometrica* 62, 467-476.
- [33] IMBENS, G. W. AND RUBIN, D. B. (1997a). Bayesian inference for causal effects in randomized experiments with noncompliance. *Annals of Statistics* 25, 305-327.
- [34] Jost S, Bernard MC, Kaiser L, et al. A patient with HIV-1 superinfection. *N Engl J Med*. 2002;347(10):731-736.

- [35] Jurriaans S, Kozaczynska K, Zorgdrager F, et al. A sudden rise in viral load is infrequently associated with HIV-1 superinfection. *J Acquir Immune Defic Syndr*. 2008;47(1):69-73.
- [36] Kaptchuk, T., 2001. The double-blind, randomized, placebo-controlled trial: Gold standard or gold calf? *Journal of Clinical Epidemiology* 54(6): 541-549.
- [37] Koelsch KK, Smith DM, Little SJ, et al. Clade B HIV-1 superinfection with wild-type virus after primary infection with drug-resistant clade B virus. *AIDS*. 2003;17(7):F11-16.
- [38] Koenig, Serena P., Fernet Leandre, and Paul E. Farmer. 2004. "Scaling-up HIV treatment programmes in resource-limited settings: the rural Haiti experience." *AIDS*, 18: S21-S25.
- [39] Kremer, M. (1996) "Integrating Behavioral Choice Into Epidemiological Models of AIDS," *Quarterly Journal of Economics* 111 (2): 549-73.
- [40] Lange J., Perriens J., Kuritzkes D. & Zewdie D. (2004). What policymakers should know about drug resistance and adherence in the context of scaling-up treatment of HIV infection. *AIDS*, 18(Suppl 3), S69-S74.
- [41] Larson, B., M. Fox, S. Rosen, M. Biib, C. Sigei, D. Shaffer, F. Sawe, M. Wasunna and J. Simon. 2008. "Early effects of antiretroviral therapy on work performance: preliminary results from a cohort study of Kenyan agricultural workers." *AIDS*, 22(3).
- [42] Laurent, C., N. Diakhate, N. F.N. Gueye, M.A. Toure, P.S. Sow, M.A. Faye, M. Gueye, I. Laniece, C.T. Kaned, F. Liegois, L. Vergne, S. Mboup, S. Badiane, I. Ndoye and E. Delaporte. 2002. "The Senegalese government's highly active antiretroviral therapy initiative: an 18-month follow-up study." *AIDS*, 16: 1363- 1370.
- [43] MacLehose, R.R., Reeves, B.C., Harvey, I.M., Sheldon, T.A., Russell, I.T. & Black, A.M.S., 2001. A systematic review of comparisons of effect sizes derived from randomised and non-randomised studies. *Health Technology Assessment* 4(34): 1-153.
- [44] Maddala, G. (1986). *Limited Dependent and Qualitative Variables in Econometrics*. Cambridge: Cambridge University Press.
- [45] Marder, J. (2005). *The Meth Menace*. In *Press-Telegram*. Retrieved October 7, 2011, from http://lang.presstelegram.com/Meth_Menace/day1_Meth.html.
- [46] Marins, J.R.P., L.F. Jamal, S. Y. Chen, M.B. Barros, E.S. Hudes, A.A. Barbosa, P. Chequer, P.R. Teixeira and N. Hearst. 2003. "Dramatic improvement in survival among adult Brazilian AIDS patients." *AIDS*, 17:1675-1682.
- [47] Miguel, Edward and Michael Kremer. 2004. "Worms: Identifying impacts on education and health in the presence of treatment externalities." *Econometrica*, 72(1), January, 159-217.
- [48] Mohanan, Manoj. 2008. "Causal Effects of Health Shocks on Consumption and Debt: Quasi-Experimental Evidence from Bus Accident Injuries." Harvard University. Mimeo.
- [49] National Department of Health. (2004). *National Treatment Guidelines*. Pretoria: National Department of Health.
- [50] National Department of Health. (2007a). *HIV and AIDS and STI Strategic Plan for South Africa, 2007-2011*. Pretoria: National Department of Health.
- [51] National Department of Health. (2007b). *National HIV and Syphilis Prevalence Survey South Africa 2006*. Pretoria: National Department of Health.
- [52] Over, Mead, and Peter Piot, "HIV Infection and Sexually Transmitted Diseases," *Disease Control Priorities in Developing Countries*, Dean T. Jamison and W. Henry Mosley, eds. (New York: Oxford University Press, 1993).

- [53] Palella, F.J., K. Delaney, A. Moorman, M. Loveless, J. Fuhrer, G. Satten, D. Aschman and S. Holmberg. 1998. "Declining morbidity and mortality among patients with advanced human immunodeficiency virus infection." *New England Journal of Medicine*, 338: 853-860.
- [54] Philipson, Tomas, and Richard Posner, *Private Choices and Public Health: The AIDS Epidemic in an Economic Perspective* (Cambridge, MA: Harvard University Press, 1993).
- [55] Piot, Peter, Michael Kazatchkine, Mark Dybul et al., "AIDS: lessons learnt and myths dispelled," *The Lancet*, 374 (2009), 260-263.
- [56] Porco T.C., Martin J.N., Page-Shafer K.A., Cheng A., Charlebois E., Grant R.M. & Osmond, D.H. (2004). Decline in HIV infectivity following the introduction of highly active antiretroviral therapy. *AIDS*, 18, 81-88.
- [57] Rains, C. & Penzien, D.B., 2005. Behavioural research and the double-blind placebocontrolled methodology: Challenges in applying the biomedical standard to behavioural headache research. *Headache* 45(5): 479-486.
- [58] Rosenbaum, P. R., and Rubin, D. B., (1983), "The Central Role of the Propensity Score in Observational Studies for Causal Effects," *Biometrika* 70, 41–55.
- [59] RUBIN, D. B. (1974). Estimating causal effects of treatments in randomized and nonrandomized studies. *Journal of Educational Psychology* 66, 688–701.
- [60] RUBIN, D. B. (1977). Assignment to a treatment group on the basis of a covariate. *Journal of Education Statistics* 2, 1–26.
- [61] RUBIN, D. B. (1978). Bayesian inference for causal effects: the role of randomization. *Annals of Statistics* 6, 34–58.
- [62] RUBIN, D. B. (1980). Discussion of 'randomization analysis of experimental data in the Fisher randomization test', by Basu. *Journal of the American Statistical Association* 75, 591–593.
- [63] RUBIN, D. B. (1990a). Comment: Neyman (1923) and causal inference in experiments and observational studies. *Statistical Science* 5, 472–480.
- [64] RUBIN, D. B. (1990b). Formal modes of statistical inference for causal effects. *Journal of Statistical Planning and Inference* 25, 279–292.
- [65] RUBIN, D. B. AND THAYER (1978). Relating tests given to different samples. *Psychometrika* 43, 3–10.
- [66] Schneider, H., Coetzee, C., Van Rensburg D. & L. Gilson. 2010. Differences in antiretroviral scale-up in three South African provinces: the role of implementation management. *BMC Health Services Research* 10 (Suppl 1), S4-S13.
- [67] Shisana O, Rehle T, Simbayi LC, Parker W, Zuma K, Bhana A, Connolly C, Jooste S, Pillay V et al. (2005). South African National HIV Prevalence, HIV Incidence, Behaviour and Communication Survey, 2005. Cape Town: HSRC Press.
- [68] Smith DM, Wong JK, Hightower GK, et al. The clinical consequences of HIV superinfection. 15th International AIDS Conference, Bangkok, Thailand. July 11-16, 2004. Abstract TuOrB1140.
- [69] Smith DM, Wong JK, Hightower GK, et al. HIV drug resistance acquired through superinfection. *AIDS*. 2005;19(12):1251-1256.
- [70] Sterne J.A.C., Hernán M., Ledergerber B., Tilling K., Weber R., Sendi P., Rickenbach M., Robins J., Egger M. & Swiss HIV Cohort Study. (2005). Long-term effectiveness of potent antiretroviral therapy in preventing AIDS and death: a prospective cohort study. *Lancet*, 366, 378–84.

- [71] Thomas, Duncan; Frankenberg, Elizabeth; Friedman, Jed; Habicht, Jean-Pierre; Hakimi, Mohammed; Ingwersen, Nicholas; Jaswadi; Jones, Nathan; McKelvey, Christopher; Peltó, Gretel, et al. 2006. "Causal Effect of Health on Labor Market Outcomes: Experimental Evidence," California Center for Population Research. On-Line Working Paper Series. Los Angeles, CA.
- [72] Thirumurthy, Harsha, Joshua Graff Zivin and Markus Goldstein. 2008. "The Economic Impact of AIDS Treatment: Labor Supply in Western Kenya." *Journal of Human Resources*, 43:511-552.
- [73] Velasco-Hernandez, X. Jorge, Fred Brauer, and Carlos Castillo-Chavez, "Recruitment Effects in Heterosexually Transmitted Disease Models," Cornell University, August 1993.
- [74] Wilson T.E., Gore M.E., Greenblatt R., Cohen M., Minkoff H., Silver S., Robinson E., Levine A. & Gange S.J. (2004). Changes in sexual behavior among HIV-infected women after initiation of HAARV. *American Journal of Public Health*, 94(7), 1141-46.
- [75] Wools-Kaloustian, Kara, Silvester Kimaiyo, Lameck Diero, Abraham Siika, John Sidle, Constantin T. Yiannoutsos, Beverly Musick, Robert Einterz, Kenneth H. Fife and William M. Tierney. 2006. "Viability and effectiveness of large-scale HIV treatment initiatives in sub-Saharan Africa: experience from western Kenya." *AIDS*, 20:41-48.
- [76] Yamano, Takashi and T.S. Jayne. 2004. "Measuring the impacts of working-age adult mortality on small-scale farm households in Kenya." *World Development* 32:91-119.
- [77] Young, A. (2005). The gift of the dying: The tragedy of AIDS and the welfare of future African generations. *Quarterly Journal of Economics* 120(2): 423-466.
- [78] Zewdie D., Lange J. & Kuritzkes D. (2004). Editorial, *AIDS*, 18 (Suppl 3), S1-S3.

Appendix A: Theoretical Framework Details and Proofs

Assumption 1.

$$\left(\frac{\partial}{\partial \gamma_1}\right) \left[\left(\frac{du}{d\sigma_{neg}}\right) - \beta \left(\frac{\partial \phi}{\partial \sigma_{neg}}\right) \left(v^{pp} - \frac{w}{1-p\beta}\right) \right] < 0;$$

Assumption 2.

$$1 - p - \phi_{\gamma_1} > 0.$$

Assumption 1 means that the private marginal utility of risk-taking falls when prevalence rises. Assumption 2 implies that higher steady-state prevalence levels must be supported by higher levels of risk-taking by the uninfected.

Assumption 1 is nontrivial, because increased prevalence has offsetting effects on the marginal utility of risk-taking: it raises the impact of risk-taking on exposure ($\phi_{\sigma_{neg}\gamma_1} > 0$), but it lowers the lifetime utility of a healthy person relative to a sick one. High prevalence could encourage risk-taking, because staying healthy becomes more costly. This point is emphasized by Kremer [1996]. For observed U. S. prevalence levels (lower than developing country levels), it appears that prevalence discourages risk-taking [Ahituv, Hotz, and Philipson 1996; Philipson 2000].

Assumption 2 is very likely to hold in the pre-ARV equilibrium, and thus is useful for assessing the effect of ARV. Note that, at any steady state, it must be true that $1 - \phi_{\gamma_1} > 0$. Therefore, when survival rates s are low, the assumption condition is also likely to hold. Comparative dynamics that begin with the post-ARV equilibrium might be different, if rates of healthy survival increase dramatically so as to violate this assumption. This would lead to unstable equilibria.

Given Assumption 1, the optimal risk-taking level falls with prevalence (which lowers the marginal utility of risky behavior), and rises with breakthroughs in treatment p (which lower the cost of infection, holding prevalence fixed). Given Assumption 2, the steady-state risk level rises with steady-state prevalence, but falls with p . When p is higher, infected people live longer, and it requires less risk-taking to support a given level of prevalence.

Finally, we make an assumption analogous to Assumption 1.

Assumption 3.

$$\frac{\partial}{\partial \sigma_{pos}} \left[\frac{du}{d\sigma_{neg}} - \beta \frac{\partial \phi}{\partial \sigma_{neg}} \left(v^{pp} - \frac{w}{1-p\beta}\right) \right] < 0$$

This assumption implies that the increase in risky behavior by the *HIV+* lowers risktaking among the *HIV-* individuals. Generally, there are two offsetting effects: increases in σ_{pos} lower the relative benefit of remaining healthy, but raise the risk of infection. Just as with prevalence, we assume that the latter effect dominates the former in developed countries.

Appendix B: Facility Names and Randomization Arms

Table 1: Study Clinics in the Free State.

Facility Name	Town	District
Matjhabeng	Welkom	Lejweleputswa
Phomolong	Hennenman	Lejweleputswa
Welkom	Welkom	Lejweleputswa
Batho	Bloemfontein	Motheo
Heidedal	Bloemfontein	Motheo
MUCPP	Bloemfontein	Motheo
Namahali	Phuthadjithaba	Thabo Mofutsanyana
Tseki	Phuthadjithaba	Thabo Mofutsanyana
Tshiame	Harrismith	Thabo Mofutsanyana
Refenggotso	Deneysville	Fezile Dabi
Zamdela	Sasolburg	Fezile Dabi
Itumeleng	Jagersfontein	Xhariep

Table 2: Randomization Arms.

<p>Group A: Households including ARV patients who receive the ARV treatment and associated support provided as part of government's ARV treatment programme</p> <p>[Sample size: n=216; 18 patient households/site]</p>	<p>Group B: Households including ARV patients who receive the ARV treatment and associated support provided as part of government's ARV treatment programme PLUS Adherence support provided by a trained peer adherence supporter during twice weekly visits to the patient</p> <p>[Sample size: n=216; 18 patient households/site]</p>
<p>Group C: Households including ARV patients who receive the ARV treatment and associated support provided as part of government's ARV treatment programme PLUS Adherence support provided by a trained peer adherence supporter during twice weekly visits to the patient PLUS Nutritional supplementation: weekly delivery of two 400g cans of meatballs and spaghetti in tomato sauce by peer adherence supporter</p> <p>[Sample size: n=216; 18 patient households/site]</p>	<p>Group D (not randomly assigned but randomly selected!):</p> <p>Randomly selected households from the general community served by the selected health facility, excluding households where someone is known to receive ARV treatment [Sample size: n=180; 15 households/site]</p>

Appendix C: General Data Overview

Figure 1: HIV surveillance sites in South Africa.

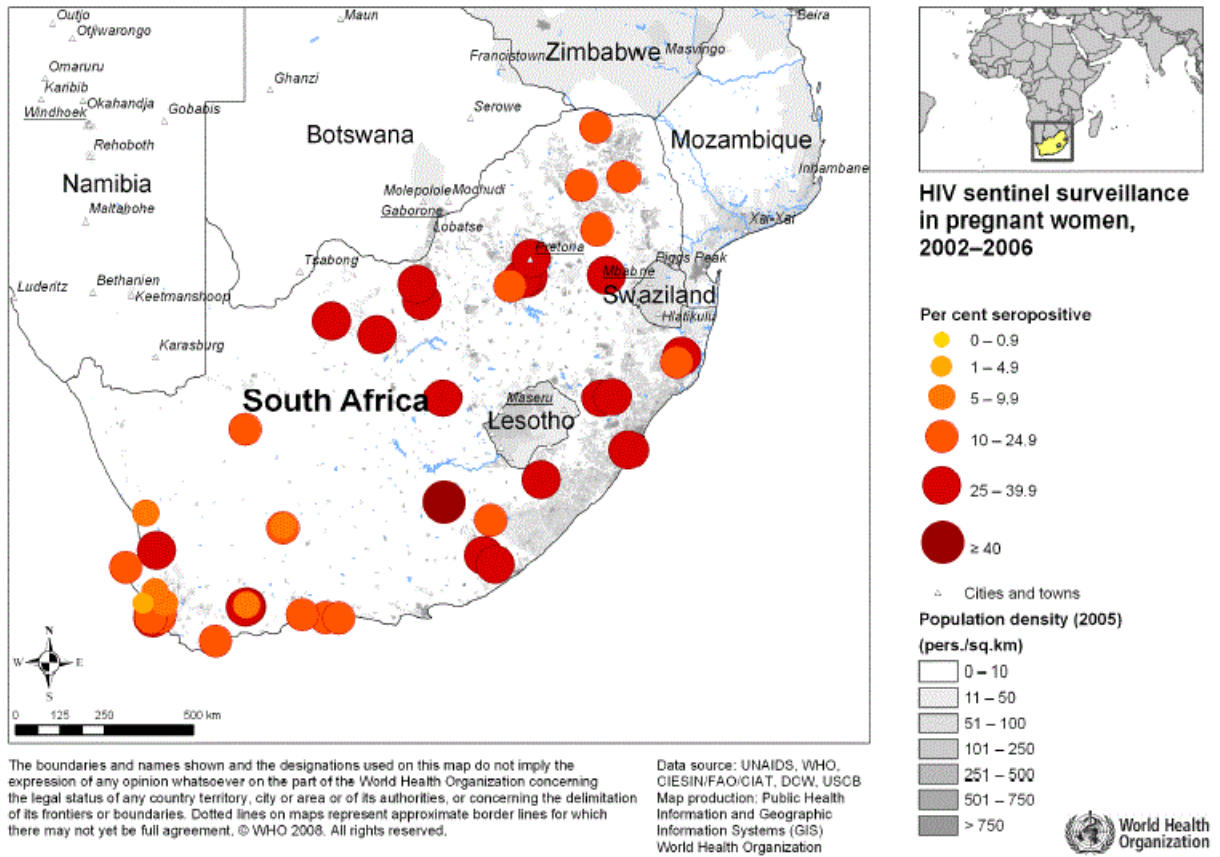


Figure 2: Geographic Distribution of ARV clinics in South Africa.

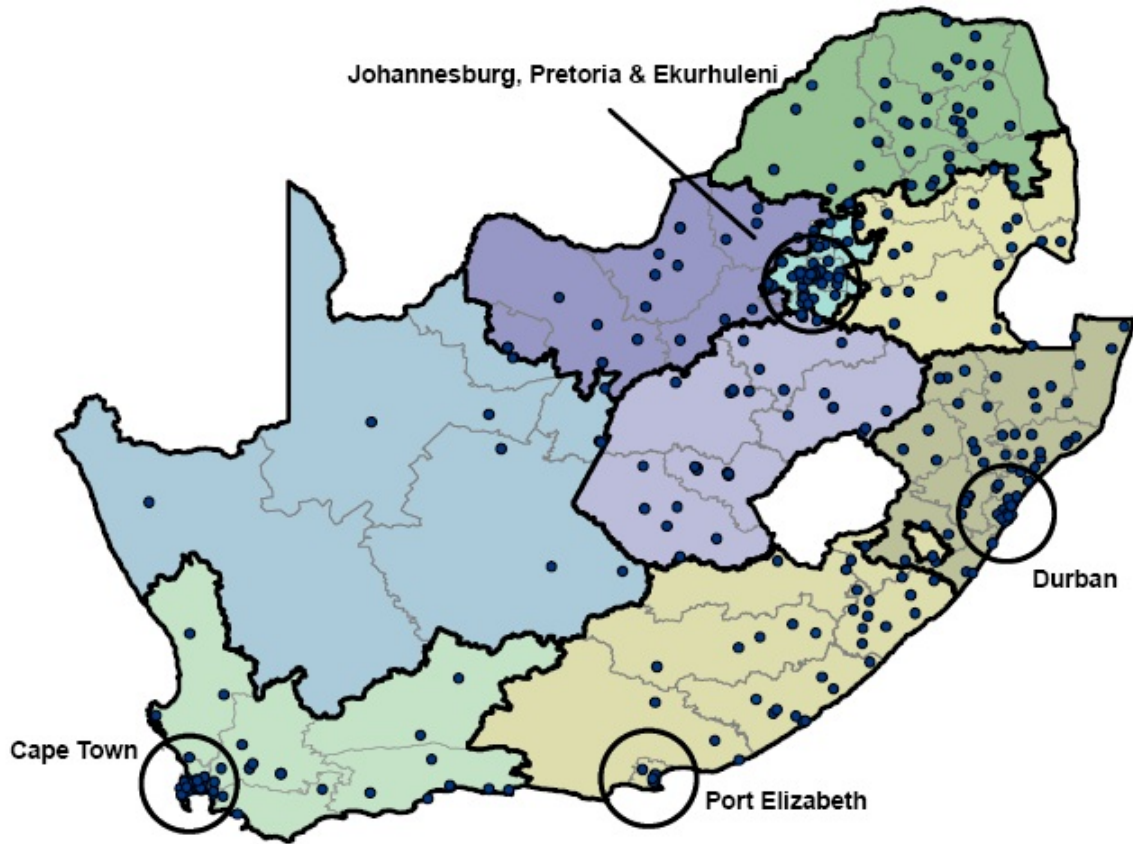


Figure 3: Geographic Distribution of ARV clinics in Free State

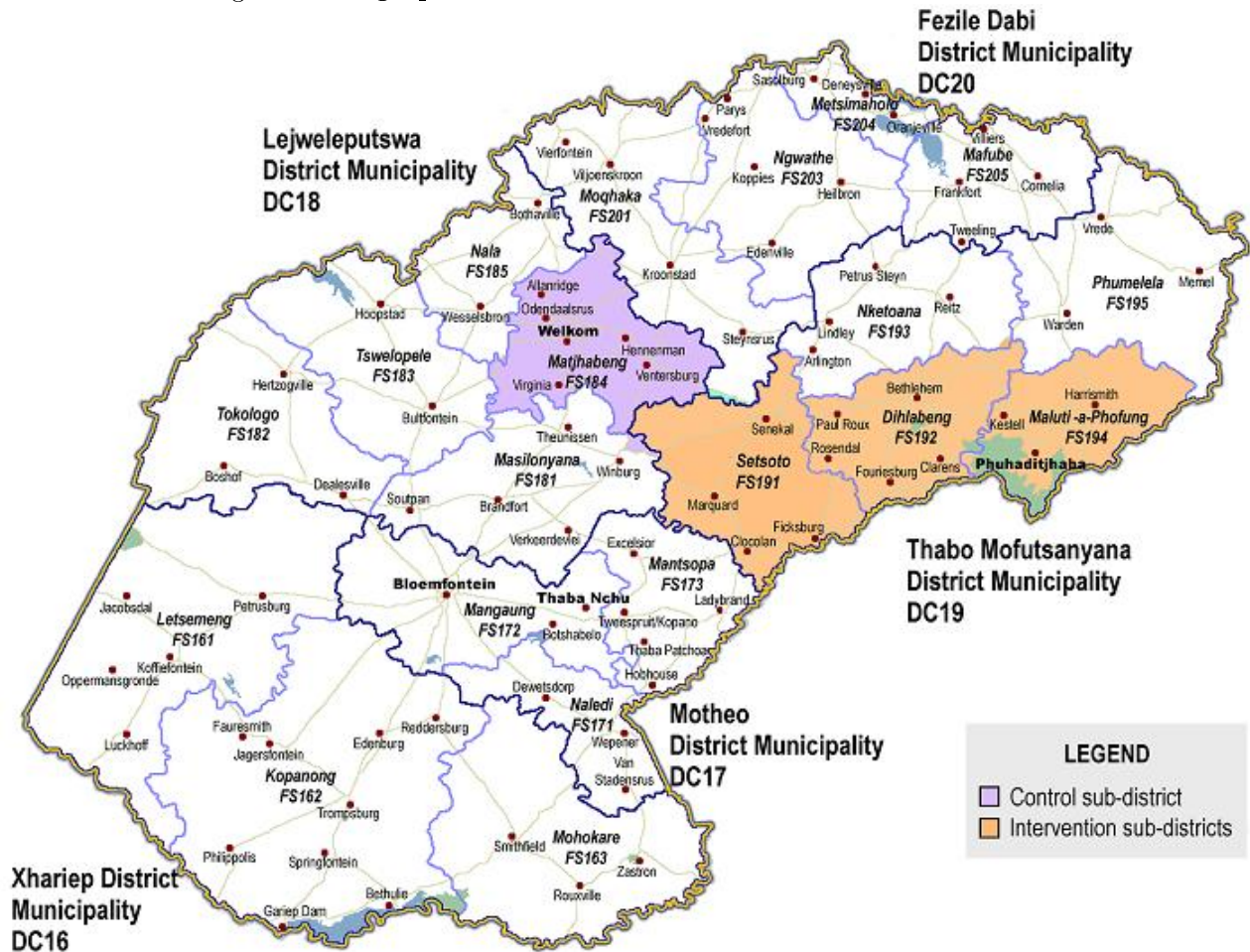


Figure 4: Population Density of the Free State (South Africa) as of 2001.

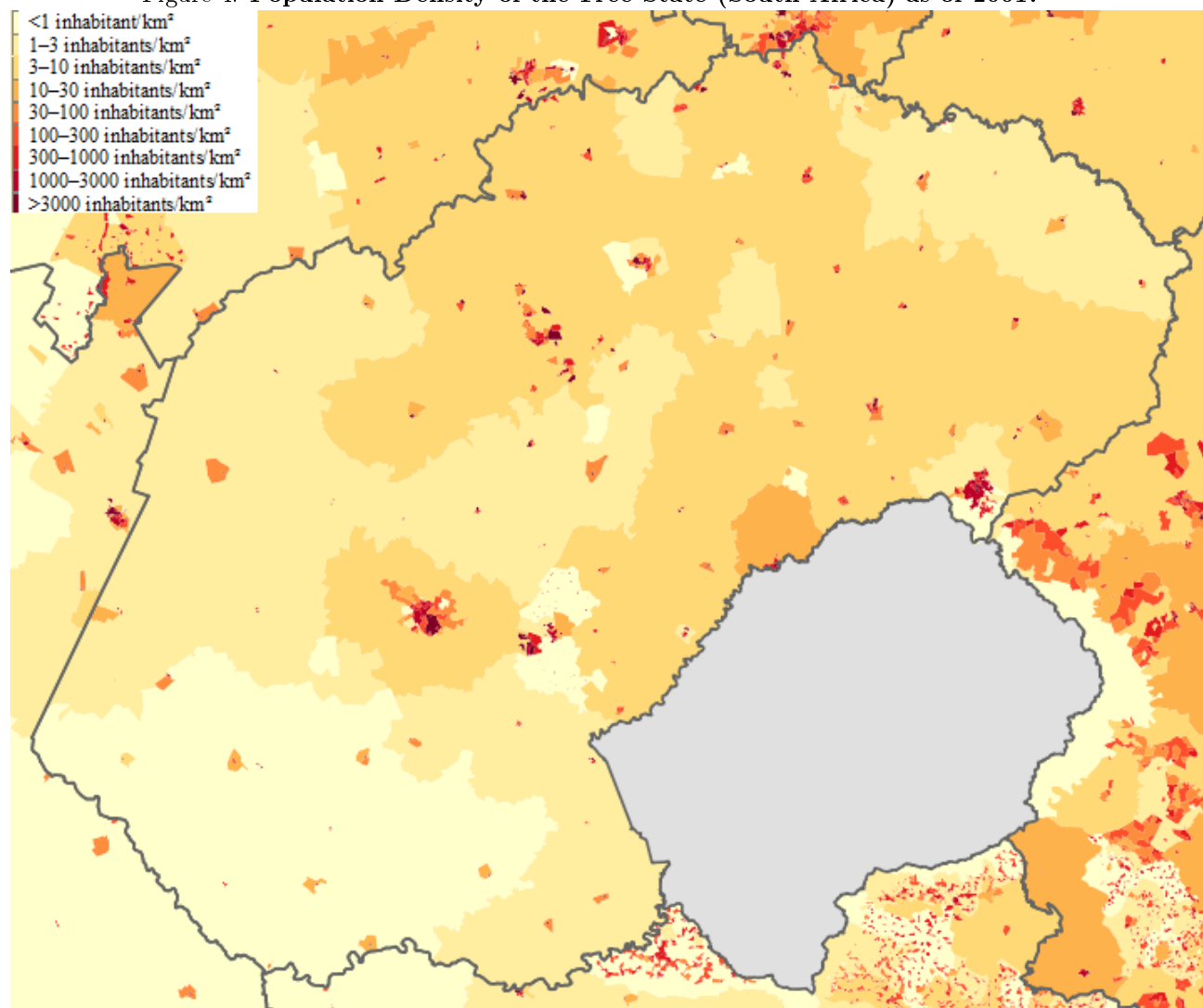


Figure 5: Distance to nearest clinic in South Africa

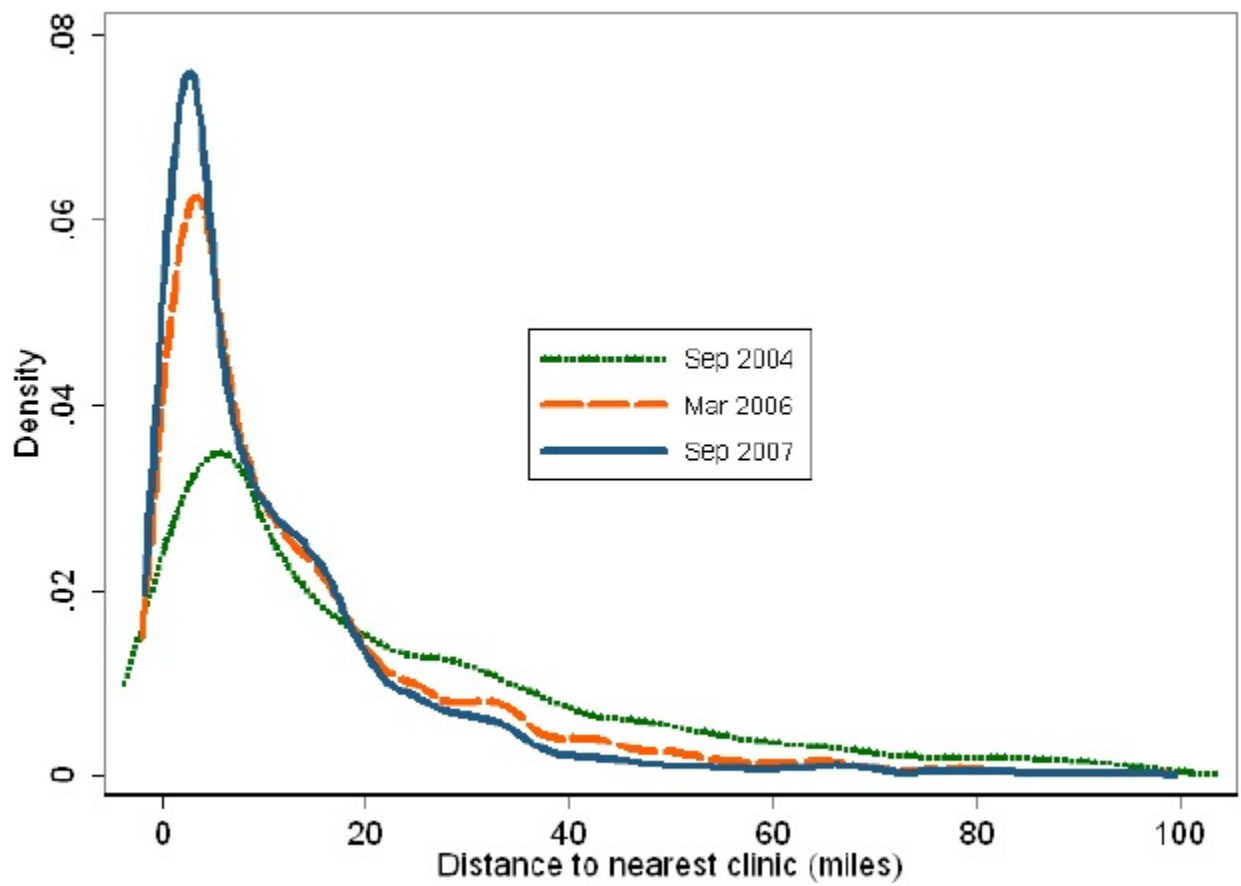


Table 2: ARV clinics in South Africa

Province	% of Population	Health Center	Hospital	Total
Eastern Cape	14	7	33	40
Free State	6	25	9	34
Gauteng	20	22	27	50
Kwa-Zulu Natal	21	13	57	70
Limpopo	12	1	35	36
Mpumalanga	7	0	19	19
Northern Cape	2	6	5	11
North West	8	2	21	23
Western Cape	10	27	30	58
Total Percent	100	103	236	339
		30.4	69.6	100

Column 2 shows percent of South African population residing in each province.

Table 3: **Comparing Characteristics at treatment baseline for all clinics in South Africa for Black men (2004-2007)**

Variable	Nearer Baseline	Farther Baseline	t-Stat on diff
Age	33.06	32.86	1.18
Yrs of primary education	5.75	6.38	-7.88
Yrs of secondary education	2.19	2.93	-8.22
Completed Matric (H.S.)	0.25	0.38	-6.00
Some post-Matric educ.	0.02	0.03	-2.00
Never held a job	0.26	0.22	2.00
Ever married	0.45	0.46	-0.50
Spouse resides in hhold	0.33	0.37	-2.00
Number of adults in hhold	3.17	2.81	4.00
Number of kids in hhld	1.51	1.09	7.17
Senior in hhold	0.23	0.11	12.00
Lived here 6 months ago	0.97	0.96	1.00

Notes: Nearer defined as closer than median (7.5 miles) from nearest clinic in first wave of sample. Change defined as change in value of variable between first wave (Sept 2004) and last wave (Sept 2007) of sample. Distance calculated from centroid of neighborhood to clinic location. Sample includes individuals aged 25-44 who live in households containing a 25-44 year old (including self). Standard errors clustered at the main place level.

Table 4: **Comparing Characteristics at treatment baseline for all clinics in South Africa for Black women (2004-2007)**

Variable	Nearer Baseline	Farther Baseline	t-Stat on diff
Age	33.42	32.97	3.00
Yrs of primary education	5.77	6.37	-10.00
Yrs of secondary education	2.18	2.88	-10.00
Completed Matric (H.S.)	0.24	0.33	-10.00
Some post-Matric educ.	0.01	0.03	-1.00
Never held a job	0.43	0.39	1.50
Ever married	0.56	0.54	1.00
Spouse resides in hhold	0.33	0.42	-4.50
Number of adults in hhold	3.33	3.08	3.57
Number of kids in hhld	2.44	1.88	8.14
Senior in hhold	0.22	0.13	9.00
Lived here 6 months ago	0.98	0.98	0.00

Notes: Nearer defined as closer than median (7.5 miles) from nearest clinic in first wave of sample. Change defined as change in value of variable between first wave (Sept 2004) and last wave (Sept 2007) of sample. Distance calculated from centroid of neighborhood to clinic location. Sample includes individuals aged 25-44 who live in households containing a 25-44 year old (including self). Standard errors clustered at the main place level.

Table 5: **Characteristics of Patients at the Time of Enrollment in the Free State Program**

Variable	All Patients Enrolled in the Program (n=14,267)	Ever Received HAART After Enrollment (n=3125)
Body weight, kg	55 (48-64)	55 (48-63)
Age, years	35 (29-41)	36 (30-42)
Female sex	9320 (65.3)	2022 (64.7)
Previous tuberculosis	1791 (12.6)	993 (31.8)
Active tuberculosis	416 (2.9)	204 (6.5)
Previous HAART	173 (1.2)	105 (3.4)
CD4 cell count		
<25	1140 (8.0)	420 (13.4)
25-49	965 (6.8)	381 (12.2)
50-99	1742 (12.2)	782 (25.0)
100-199	3042 (21.3)	1542 (49.3)
200-349	2352 (16.5)	
>350	2348 (16.5)	
Unknown	2678 (18.8)	
First viral load		
<400	471 (3.3)	145 (4.6)
400-999	33 (0.2)	25 (0.8)
1000-9999	189 (1.3)	133 (4.3)
>10000	1535 (10.8)	1298 (41.5)
Unknown	12039 (84.4)	1524 (48.8)
World Health Organization		
1	450 (3.2)	249 (8.0)
2	1327 (9.3)	750 (24.0)
3	2572 (18.0)	1434 (45.9)
4	518 (3.6)	223 (7.1)
Unstaged	9400 (65.9)	469 (15.0)

Notes: Abbreviation: HAART, highly active triple antiretroviral treatment. Data are given as median (interquartile range) or as number (percentage).

Table 6: Characteristics of Patients at the Time of Enrollment in the Free State Program between 2004-2006.

Variable	Category	2004	2005	2006	Total	χ^2 test
Age		37.9 (9.0)	37.7 (8.3)	36.1 (7.7)	37.4 (8.4)	3.192 (2,844)
Personal Monthly Income		1774.2 (1439.0)	1737.6 (1486.8)	1007.9 (760.3)	1657.8 (1411.9)	1.727 (2,108)
Dwelling Size (Number of rooms)		3.9 (1.9)	3.7 (1.6)	4.0 (1.7)	3.8 (1.7)	0.044 (2,844)
Transport Cost		6.8 (9.1)	8.5 (12.4)	10.1 (14.7)	8.4 (12.2)	3.259 (2,589)
CD4 cell count at ART initiation		109.3 (73.7)	138.9 (73.6)		120.8 (74.8)	4.229 (1,109)
Viral load at ART initiation		327113.4 (710803.5)	255867.3 (511759.9)		302490.1 (646032.4)	0.638 (1, 226)
Days since first HIV+ diagnosis		772.7 (971.7)	625.4 (747.8)	545.9 (748.3)	642.9 (806.9)	3.756 (2, 820)
Sex	Male	35.8	27.6	24.6	28.9	<0.05
	Female	64.2	72.4	75.4	71.1	
Population Group	Black	94.7	94.7	91.1	94.0	<0.05
	Coloured	4.1	5.3	8.9	5.8	
	White	1.2	0.0	0.0	0.2	
Marital Status	Married, living together	21.9	19.0	14.1	18.6	n.s.
	Unmarried, living together	7.3	10.3	10.5	9.7	
	No cohabitation with partner	70.8	70.7	75.4	71.7	
Education	No formal education	3.1	5.2	3.5	4.4	<0.05
	Primary education	23.3	33.8	33.3	31.3	
	Some secondary education	52.3	45.4	46.8	47.2	
	Grade 12 Tertiary education	15.5 (5.7)	14.0 (1.6)	15.2 (1.2)	14.6 (2.5)	
Work for pay	No	80.8	85.4	80.7	83.4	n.s.
	Yes	19.2	14.6	19.3	16.6	
Labor force	No	63.5	57.9	46.1	56.9	<0.005
	Yes	36.5	42.1	53.9	43.1	
Social welfare grant	No	47.7	43.2	46.2	44.8	n.s.
	Yes	52.3	56.8	53.8	55.2	
Dwelling Type	Formal	77.7	74.2	82.5	76.7	n.s.
	Informal	22.3	25.8	17.5	23.3	
Toilet Type	Flush toilet	60.1	68.7	87.1	70.4	
	Pit latrine	30.1	20.8	11.2	21.0	
	Bucket latrine	8.8	9.1	1.8	7.5	
	Chemical toilet	0.5	1.0	0.0	0.7	
Toilet Site	None	0.5	0.4	0.0	0.4	<0.001
	Inside dwelling	28	31.5	49.4	34.3	
	On-site/in yard	69.4	63.5	49.4	62.0	
Toilet Site	Off-site/outside yard	2.6	4.9	1.2	3.7	
Moved for ART	No	98.4	99.0	99.4	98.9	n.s.
	Yes	1.6	1.0	0.6	1.1	
Previous ART	No	88.6	94.0	97.0	93.4	<0.005
	Yes	11.4	6.0	3.0	6.6	

Notes: n.s. (p>0.05)

Appendix D:

Appendix E: Alternative Identification Strategies

Option 2: Treatment Effects Model

The first step is to build a model that allows for the joint determination of treatment status and sexual activity. A simple way to do this is to use a standard “treatment effects” model with instrumental variables. Specifically, we use a two-equation model where the first-stage models the binary receipt of ARV treatment as a probit, and the second-stage is a linear model for the number of sexual partners.

Let T_i^* represent the latent index function that measures the treatment propensity for *HIV+* patient i .

$$T_i^* = \beta_1 X_i + \beta_2 Z_i - \epsilon_{T,i} \quad (7)$$

The vector X_i represents observed exogenous co-variates that determine treatment propensity: age, gender, education, along with state-level social and economic factors.

Z_i is the vector of variables or a variable that influences the probability of the ARV take-up independent of numbers of sexual partners. We can use a combination of any the following variables as plausible instruments:

- time to clinic;
- variables that generate variation (nutritional supplementation or nurse support) for Groups B and C from Section 3.3;
- $CD4 < 200$ as an indicator variable²⁵

Our instruments could fail if they are correlated with unobserved determinants of sexual activity. To address this issue, we will examine evidence that our instruments are related to differences in sexual activity only through their effects on ARV treatment receipt, and not otherwise.

Treatment is also assumed to depend on a random error component $\epsilon_{T,i}$ that is uncorrelated with X_i and Z_i . Define T_i as the indicator variable for whether individual i actually received ARV; it equals unity if and only if the latent index T_i^* exceeds zero.

Let S_i represent the number of sex partners for *HIV+* patient i :

$$S_i = \varsigma_1 T_i + \varsigma_2 X_i - \epsilon_{S,i} \quad (8)$$

For simplicity, we assume S_i depends linearly on X_i . To complete the model and allow for correlation between treatment and sexual activity, we assume the errors $\epsilon_{S,i}$ and $\epsilon_{T,i}$ are jointly distributed as bivariate normal with correlation coefficient ρ . It is useful to think of this correlation ρ as unobserved health. That is, patients with poor unobserved health are more likely to get treatment and they are also less likely to be sexually active. We estimate this joint model via maximum likelihood²⁶.

Computationally, we can use the standard “treatreg” command (with weights and robust standard errors clustered at the state level) in Stata version 10. The command uses maximum likelihood methods to estimate the probit treatment equation and linear outcome equation simultaneously.

A Count Data Model of Sex Partners

While the simple treatment effects approach is standard, its limitation is the modeling of sex partners as a continuously distributed variable, instead of an integer-valued count. Therefore, we present an alternative “count data” approach where the number of partners follows a negative binomial distribution, but ARV receipt continues to be modeled simply as a probit²⁷.

Suppose there is some common unobserved component in the treatment and sex partner equation. Concretely, one can think of this as health status. This component, η , is assumed to be distributed as a standard normal random variable, so that the latent treatment equation reads as:

$$T_i^* = \delta_0 + \delta_1 X_i + \delta_2 Z_i - \nu_{T,i} - \eta_i \quad (9)$$

²⁵We can code the CD4 threshold as a dummy variable.

²⁶Maddala [1986] derives the maximum likelihood estimator.

²⁷See Deb and Trivedi, 2004, for a detailed exposition of the model

We assume that $\nu_{T,i}$ and η_i are distributed standard normal, and that T_i continues to follow the latent index T_i^* . Therefore, conditional on η , T_i follows a probit as before. Effectively, unobserved heterogeneity in the treatment equation is decomposed into one component that is not correlated with sexual behavior ($\nu_{T,i}$) and another one that is (η_i).

The number of sex partners is distributed as a Poisson process with mean/variance parameter $exp(\theta_0 + \theta_1 X_i + \theta_2 T_i - \lambda \eta_i - \nu_{S,i})$. Just as in the treatment equation, heterogeneity in the number of sex partners is decomposed into a component that is uncorrelated with treatment, $\nu_{S,i}$, and one that is, η_i . Notice that λ , is the covariance between the correlated errors in the two equations. If $\nu_{S,i}$ is assumed to follow a $\Gamma(\alpha)$ distribution, S_i is distributed as a negative binomial with mean $\mu \equiv exp(\theta_0 + \theta_1 X_i + \theta_2 T_i - \lambda \eta_i - \nu_{S,i})$. We can estimate the parameters of this model via Maximum Likelihood.

Option 3: Matching Methods and Control Function Estimation

The last option for identification is a semi-parametric generalization of the Heckman selection correction model though either (1) selection on observables accounted for with a matching technique or (2) selection on observables accounted for with a control function estimation. Its advantages are a more general first stage equation and a better diagnostic for assessing the comparability of the treatment and comparison groups (how balanced are the co-variates of treatment and comparison group members with similar propensity scores). Because this approach relies selection on "observables" or "unobservables", this propensity score method is most useful when the econometrician observes all of the variables used in selection but does not know the exact form of the "rule" that leads to selection into treatment.

The exact matching estimator approach will be to match treatment (coming from our Groups A, B, C) and control observations (from Group D) by X (all observable characteristics used in estimation), get the difference in mean outcomes (the treatment effect at $X = x$) at each value of X , and then get the *TOT* (Treatment on the Treated) by averaging these estimated treatment effects over the distribution of X for the treatment group.

Thus, in this case of selection on observables, adjusting for the propensity score removes the biases associated with differences in co-variates. Why is it sufficient to condition just on the propensity score? The reason is that under the Rosenbaum-Rubin assumptions for selection on observables (co-variates X), the co-variates are independent of assignment to treatment conditional on the propensity score. In other words, the distribution of co-variates should be the same across treatment and comparison groups for observations with the same propensity score. This implication of the assumptions for the propensity score approach to be appropriate provides a diagnostic: one can group observations in strata based on the estimated propensity score and check whether the co-variates are balanced across the treatment and comparison groups in each strata.

There are a number of different semi-parametric ways to use the propensity score to estimate the *TOT* given that you have achieved "balance" in the co-variates. The multiplicity of methods arises when the true functional form of the second stage equation is unknown:

1. *Control Function*: Use your first stage equation to form the Heckman selection correction term and add it to your second stage regression
2. *Stratify*: Divide the data into blocks based on the propensity score. Run the second stage equation within each block (this might just be the mean difference in outcomes for treatment and comparison observations in each block). Calculate the weighted mean of the within-block estimates to get the *TOT* (weight by number of treatment observations in each block).
3. *Match*: Match each treatment observation with a comparison observation, based on similar propensity scores (find closest match). Treat the data like panel data (like twins data) and run within-match (match fixed effects) models of the treatment effect.
4. *Weight*: Weight each observation by its propensity score and estimate the second stage equation (Hirano, Imbens and Ridder 2003).

In the context of labor market interventions in the U.S., Dehejia and Wahba (1999) illustrate these methods and show that once one achieves "balance" of co-variates within blocks that these propensity score methods

come quite close to the experimental estimates for the NSW demonstration in the U.S. in contrast to the lack of reliability of other the traditional econometric non-experimental estimators examined by LaLonde (1986).