

CAN TIME INCONSISTENCIES REDUCE THE EFFECT OF HIV TREATMENT PROGRAMS?

THEORY AND EVIDENCE FROM SOUTH AFRICA

Abstract

This paper interprets the HIV adherence decision from the perspective of an optimising agent who attempts to maximise intertemporal utility. The agent does this by weighing up the present cost and the future benefits of following a drug regime. Time inconsistencies emerge due to the lag between these two effects, causing hyperbolic agents to choose adherence levels below their long run preferences. Using the baseline survey data of a World Bank project, we compare the demand for one such commitment mechanism among different types of agents. Finally, we proceed to investigate how different agents respond to the tying in of *in kind* incentive by exploiting the experimental variation within our data.

JEL codes: D91, C12, I11

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1. Introduction

South Africa has one of the highest human immunodeficiency virus infection/acquired immunodeficiency syndrome (HIV/AIDS) rates in the world. An estimated 5.5 million South Africans are living with HIV, 11% of the total population (ASSA 2008). The prevalence rate is even higher, 18%, if we only look at those individuals between the ages of 15 and 49 (UNAIDS, 2010). Recent increases in HIV medication regimes such as the highly active antiretroviral therapy (HAART) have helped HIV patients live longer and healthier lives than was previously imaginable. Unfortunately, the successful treatment of HIV or AIDS requires near perfect adherence to these regimes. Adherence levels below 95% could lead to the emergence of a more threatening, drug resistance variant of HIV/AIDS, which could seriously undermine the effectiveness of future treatment therapy (Chesney, 2003). As a result, any deterrent to adherence could carry serious long term consequences.

Following a theoretical framework, our paper attempts to explicitly modelling the adherence decision from the vantage point of a rational agent who is trying to maximise intertemporal utility over the long run. Our model shows how non-adherence can be both a desired outcome (for agents for whom the costs outweigh benefits) and an undesired outcome (for agents who have hyperbolic time preferences or systematically forget to take pills). These two groups will adhere similarly, but will react differently when offered a commitment mechanism. We proceed to compare the take up levels of two randomly assigned adherence support programmes among different types of patients.

Finally, we proceed to investigate how different agents respond to the tying in of *in kind* incentive by exploiting the experimental variation within our data. While the tying in of incentive may help policymakers to increase take up, it does so at a cost. Both our model and our empirical results suggest that the tying in of *in kind* incentives will compromise the natural selection within these programs, since those patients who are lured in by tied in incentives are unlikely to gain as much from the commitment device as those patients who would have selected in in the absence of such incentives.

The following section familiarizes the reader with the basic literature on HIV and the factors that could influence adherence. In section 3, a theoretical model is developed to help and explain how

cost, benefits and time preferences may influence pill adherence and the decision to take up the intervention. Based on the model, some key predictions are derived in section 4. After introducing the data in section 5, we will proceed in section 6 to test the validity of our earlier derived propositions. Section 7 concludes.

2. Background and Literature

Until 2004, public provision of antiretroviral treatment was non-existent in South Africa (Johnson and McLeod, 2007). Initial roll out of antiretroviral provision was obstructed by the Mbeki government who were sympathetic to the view of high profile AIDS-denialists, who were suspicious of link between HIV and AIDS, as well as the impact antiretroviral drugs. According to Chigwedere et al. (2008), this delay in ARV treatment programs has cost South Africa an estimated 330,000 lives. Since the introduction of the “*Operational plan for comprehensive HIV and AIDS care, management and treatment for South Africa*” (Department of Health, 2003) there has been a strong increase in adherence provision. The portion of newly eligible adults that initiate treatment has grown from 4.9% in 2004 to 40.2% in 2008¹. In the same year, 2008, an estimated 568 000 adults and children were receiving antiretroviral treatment in South Africa. An estimated 80% of these patients received their medication through public health care (Adam and Johnson, 2009). The current prevalence rate in South Africa among adults aged between 15 to 49 is estimated at around 18% (UNAIDS, 2010; HSRC, 2008).

Over the last few years there have been dramatic improvements in the antiretroviral treatment regimens that HIV patients can follow, which allows patients to live longer and healthier lives. Preliminary evidence (Fang et al., 2007) shows a drastic extension in the life expectancy rates of patients who successfully adhere to the highly active antiretroviral therapy (HAART). The 5 year survival rates for patients who now start off on HAART are more than 3 times higher than they were on the older treatment programs (Aracena-Genao et al., 2008). Adherence is vital in determining the success of treatment, especially since near perfect adherence levels are required

¹If eligibility is defined using the Department of Health official criteria

in order to maintain virologic suppressions. Despite these dangers, patients often adhere below 90% (Bartlett, 2002)².

From the demand side, the most common barriers for adherence are costs (both the ticket price of the pills as well as the opportunity cost of getting the pills), side effects and the risk of being found out by people to whom you haven't disclosed your status. In a comparable study in Botswana where respondents were directly questioned about their adherence roughly 50% of the patients stated that there were side effects to taking ARV drugs, but only about 10% believed that these side effects posed as a barrier to their adherence (Weiser, et al, 2003).

One way of inducing more patients to adhere more frequently would be through self commitment devices. These devices can take on many forms and are observed in many different aspects of everyday life where agents may face conflicts between their long term goals and short term temptation, such as our inability to save for retirement, exercise or kick addictions. These commitment devices come with varying degrees of formality, some people may make New Year's resolution, while other may hire personal trainers or make bets (Bryan et al., 2010). One such device that is commonly analyzed is the rotating savings and credit associations (ROSCAs), which is commonly used as a forced method of saving among the poor in developing countries (Banerjee and Mullainathan, 2010). In an interesting study in the Philippines, Ashraf et al. (2006) show that people who exhibit time inconsistencies, and are sophisticated enough to be aware of their time inconsistencies have a strong preference for commitment savings accounts that restrict one from making early withdrawals.

3. A model of ARV adherence under hyperbolic discounting

i. The Model

There are both cost and benefits to be had by adhering regimen. The costs (which could come in the shape of side effects, price of pills, or opportunity costs) are usually immediate, while the

² Ironically, as pills have become better side effects have increasingly become a deterrent to adherence, since patients were more willing to tolerate side effect when they perceived their situations to be life threatening (Bartlett, 2002).

health benefits are only amassed over time. Assuming that both cost and benefits only last for 1 period, the instantaneous utility at period t can be formulated as:

$$u_{it} = (bx_{t-1} - \frac{1}{2}\kappa x_{t-1}^2) - c_i x_t \quad [1]$$

Where $(bx_{t-1} - \frac{1}{2}\kappa x_{t-1}^2)$ represent the total benefit that is derived from adherence in the previous period and $c_i x_t$ denotes the total cost to adherence which is realised immediately. The marginal benefit of each additional pill is assumed to decline as adherence increases ($\kappa > 0$), while marginal cost remains constant at c_i . For the sake of our model we will assume that the benefits, b , are identical across all patients.

An agent's intertemporal additive utility can be derived by summing up a series of instantaneous utility functions. For the sake of simplicity, we will restrict our model to only encapsulate three periods. Following the work of (Frederick et al, 2002), we opt for a more general functional form that allows for sharp levels of discounting in the short run and less steep discounting in the medium to long run. In our 3 period model instantaneous utility is discounted according to two time parameters, the long run discounting parameter, δ , and the short-run (hyperbolic) discounting term, β .

$$U_t(.) = u_t + \beta\delta u_{t+1} + \beta\delta^2 u_{t+2} \quad [2]$$

The discounting between the first and second period, $\beta\delta$, differs from the discount factor between the second and third period (evaluated at the from the first period) which is δ . The difference between current and future discounting ($\beta\delta$ and δ) is brought about by the hyperbolic discounting term, β . This term drives the time inconsistencies and is central to our discussion throughout.

We set our current period as $t = 1$. Substituting the instantaneous utility into equation 2 and assuming, for the sake of simplicity, that adherence will be zero in period $t = 0$ and $t = 3$, we derive the following intertemporal utility function.

$$U_1(x_{1i}, x_{2i}) = -c_i x_{1i} + \beta_i \delta (bx_{1i} - \frac{1}{2}\kappa x_{1i}^2 - c_i x_{2i}) + \beta_i \delta^2 (bx_{2i} - \frac{1}{2}\kappa x_{2i}^2) \quad [3]$$

Measured at period $t = 1$ the optimal level of pill adherence for period $t = 1$ will be $x_{1i}^* = \frac{b}{\kappa} - \frac{c_i}{\beta_i \delta \kappa}$ while the optimal level for period $t = 2$ will be $x_{2i}^* = \frac{b}{\kappa} - \frac{c_i}{\delta \kappa}$ ³. Assuming that cost and benefits remain constant, x_{2i}^* will always be larger or equal to x_{1i}^* . Consequently, most agents will prefer an adherence path line with lower adherence now and higher adherence in the future, where the delayed benefits will be discounted by δ and not $\beta\delta$.

Once period two arrives, a time inconsistency arises, as agents realise that the optimal level of adherence for period 2 is not the same as what they envisioned it to be in period 1. In fact, the adherence choice faced by the myopic (short run optimizing) self at $t = 2$ is identical to the short run decision faced by the myopic-self at $t = 1$. The disparity between these two rates cause the myopic-self to renege on his own pledge to his long term self to adhere to the long run optima at x_{2i}^* . Instead the agent chooses to adhere at $x_{2i}^+ = \frac{b}{\kappa} - \frac{c_i}{\beta_i \delta \kappa}$ which is identical to the adherence level in period 1, x_{1i}^* . Paserman (2008) refers to these confliction as a “tension between long-run goals and short run impulses”, where these trade-offs may cause patients to repeatedly postpone pill adherence despite being aware of the long term benefits. Measured at $t = 1$, the renegeing in pill adherence from x_{2i}^* to x_{2i}^+ will lead to a total net loss of.

$$V_a = U_1(x_{1i}^*, x_{2i}^*) - U_1(x_{1i}^+, x_{2i}^+) = \frac{c_i^2 (\beta_i - 1)^2}{2\kappa \beta_i} \quad [4]$$

The extent of the loss in utility is determined by the hyperbolic short run discounting term, β_i , the relative cost of adherence c_i and the quadratic cost parameter κ . Since κ and c_i were defined to be non-negative and the hyperbolic discount rate, β_i , is defined to lie between 0 and 1, the net loss in the disutility brought about by time inconsistency will always either be positive or equal to zero. The only instance where it will not be positive is if either or both $\beta_i = 1$ and $c_i = 0$. In which case, there will be no time inconsistency since x_{2i}^+ would be equal to x_{2i}^* and consequently also no loss in utility due to renegeing.

³ Throughout we will use ‘*’ to denoted the long run optimal level of adherence and ‘+’ to denoted the short run optimal level. Similarly, ‘*’ can be thought of as the optimal levels at of adherence for a patient who is maximising intertemporal utility, measured at period $t = 1$, while ‘+’ is maximising intertemporal utility, measured at period $t = 2$

ii. *Introducing Sophistication*

Within the hyperbolic discounting literature it is useful to distinguish between two types of agents: the sophisticated and the naïve. Sophisticated agents are aware of the ‘dynamic inconsistency’ between what they plan to do and what they will end up doing, while naïve agents are not. Most articles prefer a dichotomous classification between these two groups, either classifying each agent (or all agents) as naïve or sophisticated. We introduce a more general notion of sophistication to our model by allowing s_i to denote the agent’s awareness of the divergence between their optimal long run (x_i^*) and optimal short run (x_i^+) levels of adherence. By allowing s_i to vary agent can lie anywhere between perfectly naïve ($s_i = 0$) and perfectly sophisticated ($s_i = 1$).

Using this parameter we can model the perceived (rather than the actual) costs of the dynamic inconsistency - the patient’s awareness of how much they will deviate in the next period from their optimal long run adherence level⁴.

$$V_p = U_1(x_{1i}^*, x_{2i}^+ + s(x_{2i}^* - x_{2i}^+)) - U_1(x_{1i}^+, x_{2i}^+) = \frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i} \left(s_i - \frac{s_i^2}{2} \right) \quad [5]$$

The perceived cost increases with s_i ⁵. If agents are perfectly sophisticated ($s_i = 1$) perceived cost will be identical to actual cost, $\frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i}$. Conversely, if agents are perfectly naïve ($s_i = 0$), the perceived cost will be 0. Agents who lie between these extremes will have incomplete awareness of a need for a commitment. They will be aware of the possible gains to be had by getting themselves to commit to future adherence levels, but might not be fully aware of the extent of their demand for such a mechanism.

iii. *Introducing a Commitment Mechanisms*

The model is modified to allow for a commitment mechanism, allowing agents to select into a commitment mechanism at period 1, which will be enforced in period 2. In our survey this commitment mechanism will take the form of a bi-weekly visit from an adherence officer, who is

⁴ *Sophistication can also be introduced in a more direct manner by multiplying s_i directly with actual utility, $s_i * (U_1(x_{1i}^*, x_{2i}^*) - U_1(x_{1i}^+, x_{2i}^+))$. The perceived costs would be $s_i \frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i}$. None of the propositions will be affected.*

⁵ *In the absence of any commitment mechanisms s_i will have no effect on the actual cost of renegeing or total utility.*

able to enforce guilt, denoted as g_i , on patients who are not sufficiently adhering. To keep the model as simple as possible, we assume that patients are able to tailor their commitment mechanism to their own desired level of future adherence. Within this flexible framework, agents can fix their future adherence by asking the adherence officers to impose a sufficiently large guilt on them unless they adhere as their optimal future value x_{2i}^* .

Apart from the guilt that adherence officers directly impose there is also an inadvertent cost that they inflict on patients. This could be through the inconvenience of having a stranger visit you or through the fear that the adherence officer's presence could add suspicion regarding ones HIV status among neighbours or friends. We will take this cost to be a once-off that does not depend on one's adherence level, but rather only on whether one agrees to the commitment mechanism or not. The cost, denoted as d_i , is assumed to be nonnegative. Extending our utility function to incorporate the commitment mechanism we get

$$\tilde{U}_1(x_{1i}^*, x_{2i}^*, m_i^*) = u_1 + \beta_i \delta u_2 + \beta_i \delta^2 u_3 - m_i d_i + m_i g_i I(x_{2i}^* < \frac{b_i}{\kappa_1} - \frac{c_i}{\delta \kappa_1}) \quad [6]$$

Which simplifies to

$$\tilde{U}_1(x_{1i}^*, x_{2i}^*, m_i^*) = U_1(x_{1i}^+, x_{2i}^+) + m_i \left(\frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i} \right) - m_i d_i \quad [7]$$

At first glance it may seem counterintuitive to agree to the commitment mechanism, since there are no direct payoffs to having an adherence officer come to your house, only costs. The increase in intertemporal utility is brought about through the officer's indirect effect in helping patients fix their level of future adherence. In some cases the payoff, measured as $\frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i}$, will bring about a large enough increase in utility to compensate for the inadvertent cost of the commitment mechanism d_i . In other cases it will not. Ultimately, the decision into commitment will depend on how the relative cost and benefit to take up compare to one another. Since the decision into take up is a discrete choice (taking a value 0 or 1) it cannot be solved through differentiation, instead we compare total utility under take up to total utility without take up. Our derivations (see appendix) show that total utility under the commitment will be larger than total utility without the commitment if and only if

$$\left(\frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i} \right) - d_i > 0 \quad [8]$$

Patients differ in their awareness of their time inconsistency and the relative benefits that can be obtained through a commitment mechanism. Allowing for the variation in sophistication, the selection into take up becomes

$$\frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i} \left(s_i - \frac{s_i^2}{2} \right) - d_i > 0 \quad [9]$$

In which case the perceived gains in utility from higher future adherence will outweigh the direct costs of having the adherence officer come and visit you. Agents will gain most from selecting into the commitment mechanism, m_i , if the gap between how many pills they want to themselves to take in the future and how many they are likely to take in the future is large and when the relative inconvenience of having someone come and visit you at home is small.

Next, we introduce some propositions to formalise the relationship between take up and our variables of interest. We define $V = \frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i} \left(s_i - \frac{s_i^2}{2} \right) - d_i$ as measuring the net effect of the commitment mechanism. The effect that either of our variables of interest should have on take up can then be measured by differentiating V respect to c_i , d_i , β_i and s_i .

PROPOSITION 1.a. Patients with high marginal cost to adherence, c_i , would be more likely to select into the commitment mechanism.

$$\frac{\partial V}{\partial c_i} = \frac{2c_i (1 - \beta_i)^2}{\kappa \beta_i} \left(s_i - \frac{s_i^2}{2} \right) \geq 0$$

PROPOSITION 1.b. A reduction in the once-off inadvertent cost, d_i , will increase the likelihood of take up.

$$\frac{\partial V}{\partial d_i} \leq 0$$

PROPOSITION 1.c. A reduction in the short run hyperbolic discount rate, β_i , would increase the likelihood of take up.

$$\frac{\partial V}{\partial \beta_i} = \frac{c_i^2}{\kappa} \left(1 - \frac{1}{\beta_i^2} \right) \left(s_i - \frac{s_i^2}{2} \right) \leq 0$$

PROPOSITION 1.d. Sophisticated agents are more likely to opt into treatment

$$\frac{\partial V}{\partial s_i} = \frac{c_i^2 (1 - \beta_i)^2}{\kappa \beta_i} (1 - s_i) \geq 0$$

From these four propositions we infer that take up would be positively associated with the marginal cost to adherence, c_i , and negatively associated to the inadvertent cost of the commitment mechanism, d_i . Hyperbolic discounting has a positive effect on take up. Intuitively this is because the higher the level of hyperbolic discounting is (the lower β_i is) the further the actual level of future adherence is from the optimal one. Not all patients are however aware of their time inconsistencies. Our model predicts that those who are (the more sophisticated) will be more inclined to accept the commitment mechanism. There will be no reason to take up the commitment mechanism if either β_i is equal to 1 or s_i is equal to 0.

Holding marginal cost, c_i , constant, the relative effects of s_i , β_i and d_i can be illustrated schematically⁶.

Figure 1.a.: Perceived loss in utility due to time inconsistency

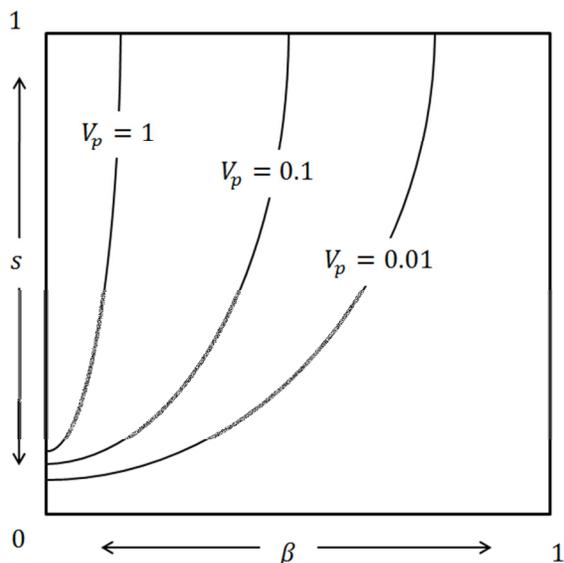
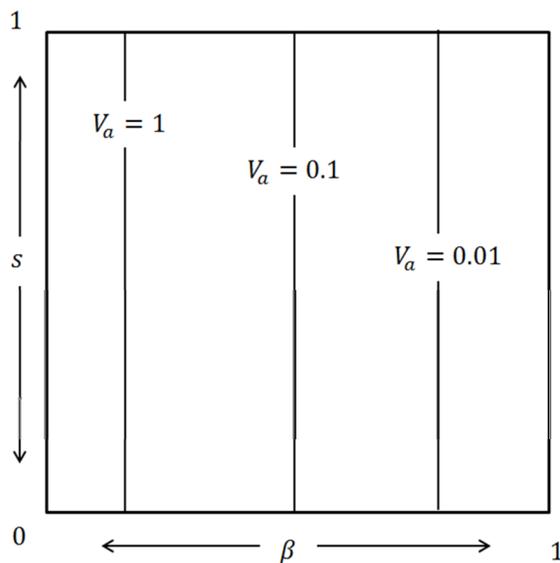


Figure 1.b.: Actual loss in utility due to time inconsistency



The first figure shows the perceived loss, V_p , in utility due to an agent's time inconsistency (equation 5), while the second figure measured the actual loss in utility, V_a , due to time inconsistency (equation 4). In both cases, the isoquant measure those point within the $\{\beta, s\}$ -space for which the relative loss in utility would be the same. Patients who agree to the commitment devices are able to recover this loss. Therefore, V_p , can also be taken as a measure

⁶ For all values of β_i and s_i . Setting $\kappa = 1$ and $c_i = 1$.

of the actual gross gain to be had by agreeing to the commitment mechanism. From Figure 1.a., we observe that those agents with high discounting (β_i close to 0) and high sophistication (s_i close to 1) have most to gain.

The graph also allows us to see which agents would be most willing to take up the control mechanism and at what cost. For instance, if we were to assume that all patients faced an identical once-off inadvertent cost, $d_i = 0.1$, then all agents lying to the north west of the indifference curve $V_p = 0.1$ will find it worthwhile to select into treatment. Starting from this point, if the once-off cost was suddenly set higher, to $d_i = 1$, then all the patients lying between the indifference curve $V_p = 0.1$ and $V_p = 1$ would be drawn out of the commitment mechanism. Conversely, if the once-off cost was lowered to $d_i = 0.01$ then the group who would find it worthwhile to select into take up would be extended to also include those patients who lie between $V_p = 0.1$ and $V_p = 0.01$.

Figure 1.b shows that the actual loss and perceived loss are not the same. This distinction might seem exorbitant, but it has strong welfare implications; those who have the most to gain by joining commitment mechanism and those who perceive to have the most to gain (and who would subsequently be the most willing to select into treatment) need not be the same people. In this sense, having patients with varying degrees of sophistication is likely to distort the inherent self-selection into take up.

iv. Adding monetary incentives to the Commitment Mechanism

There are many reasons why it may be in the interest of government to attract people into adherence programmes. Apart from the obvious health gains, there are also efficiency gains from having a healthier work force. Since government is already subsidising the entire price of the pills, the only manner in which they could induce further take up would be through the tying in *in kind* incentives to adherence or adherence support programmes⁷.

⁷ One would be inclined to assume that any such incentives would have a positive effect on take up, recent literature has however shown that the monetizing of inherently justified motivations often undermine an individual's intrinsic convictions (see Titmuss, 1971; Gneezy and Rustichini, 2000).

In our data some patients were offered food as a tied in incentive to their adherence support. Those who selected into a treatment that offered food would receive a nutritional supplement every time that the adherence officer visited them. The model is augmented to allow for the distinction between these two treatment groups by adding food as an additional parameter, which we discount by $\beta\delta$ since it is received in period 2.

$$\tilde{U}_1(x_1^*, x_2^*, m^*) = U_1(x_1^*, x_1^*) + m_i \left(\frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i} \right) + m_i d_i + m_i \beta \delta f_i \quad [10]$$

As before, we show that utility under the commitment will be larger than total utility without the commitment if and only if

$$\frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i} - d_i + \beta \delta f_i > 0 \quad [11]$$

Allowing for sophistication, the intertemporal choice regarding take up becomes

$$\frac{c_i^2 (\beta_i - 1)^2}{\kappa \beta_i} \left(s - \frac{s^2}{2} \right) - d_i + \beta \delta f_i > 0 \quad [12]$$

PROPOSITION 2. *In kind incentives, f_i , will increase take and will increase take up most for those with the lowest level of hyperbolic discounting (higher β_i).*

$$\frac{\partial V}{\partial f_i} = \beta_i \delta \geq 0 \quad \text{and} \quad \frac{\partial^2 V}{\partial f_i \partial \beta_i} = \delta \geq 0$$

Our model predicts that as long as people don't fully discount the future, food will have a positive influence on take up. The model also suggests that the magnitude of the effect will be larger for those who discount future utility less.

Prior to the introduction of in kind incentives, f_i , our model was set up so that those who have most to gain from a commitment mechanism would be the first to select into the mechanism. The introduction of tied-in incentives may distort this self-selection, in that patients with high hyperbolic discounting, who would gain most from the commitment mechanism, will discount the tied in-incentives more than those with low hyperbolic discounting. In that sense, the addition of food disproportionately favours those patients who lie to the right in figure 1a – the more patient.

The question regarding self-targeting is an especially important question for policy. If government is bearing the cost of the commitment mechanism, as well as the cost of the tied in incentives, then they would prefer the intervention to be as efficient as possible and not have people who do not stand to gain directly from the treatment join in. Policy makers should try to prevent patients who would have adhered perfectly in the absence of the mechanisms to join in, since these patients are only joining for the sake of the tied in incentives.

Throughout, selection into take up has been assumed to be a deterministic. As we move to the empirical portion of the paper, this assumption will now be relaxed. We do this through the addition of a stochastic term, e , which is said to capture the unabsorbed heterogeneity in our decision rule

$$V + e > 0$$

We assume that e is normally distributed and uncorrelated with observed characteristics. A Probit-regression can now be used to derive the marginal and partial effects on V .

4. Data and Experimental Design

Experimental designs' main contribution is the exogenous source of variation it brings about. In its cleanest form randomisation allows one to bypass the endogeneity that would usually bias ones results (Imbens, 2009). However, without imposing any structure on the data, randomized controls lack any meaning and external validity, since they don't have any 'deep parameters' or economic intuition behind them (Deaton, 2010; Keane, 2010). Contrariwise, structuralists have grown aware of the problems in trying to estimate the parameters of a fully structural model without experimental data. In order to get the most out of the data, Heckman (2010) and Heckman and Urzua (2010) propose a midway – merging the structural and evaluation approach. Randomization will allow one to get further with weaker exogeneity assumptions, while the added structure one imposes aids the ultimate understanding by giving economic meaning to the 'effects' that one would have derived using the randomization alone.

In this paper we adopt a similar approach, marrying the randomized experiment and structural methods to explain why patients select into or out of adherence support programmes.

i. *Experimental Design and Setting*

Our survey was conducted through a combined venture by the World Bank and Free State University entitled “*Effective Aids Treatment and Support for Free State province*”. The study sets out to investigate the benefits of ARV treatment on patients and their families as well as the impact of peer adherence support and nutritional interventions on adherence and future outcomes⁸. Although the project will ultimately consist of 3 waves of data, our analysis is restricted to the first wave which contains the baseline administrative data as well as the subsequent decisions into take up. Patients were randomly allocated to three treatment groups:

<i>Control:</i>	<i>Patients receive ARV treatment as well as any support that they were currently being provided through any conventional treatment program.</i>
<i>Treatment 1:</i>	<i>Patients receive the same as the control, plus a bi-weekly visit by a fellow ARV patient who has been trained as a peer supporter</i>
<i>Treatment 2:</i>	<i>Patients receive the same as the control, plus a bi-weekly visit by a fellow ARV patients who has been trained as a peer supporter as well as a weekly nutritional supplement.</i>

In all, 633 patients were recruited into the study. Only patients who were over the age of 18 and who had commenced ARV treatment within the month prior to the survey were eligible. Patients were selected from either one of the 5 clinics that agreed to participate in the study. Initially the idea was to distribute these patients equally among the three assigned groups. However, due to the low rate of take-up, additional patients were moved from the control to the two treatment groups⁹. The 633 patients were randomly allocated between the three treatment groups.

Table 1: Distribution of patients among groups

	Patients	Share
Control	106	16.8%
Treatment 1	268	42.3%
Treatment 2	259	40.9%
Total	633	100.0%

⁸ Preliminary analysis, done by the author of this paper shows that the commitment mechanism had a positive effect on future (post treatment) adherence.

⁹ At the end, only 16% of the patients were assigned to the control group. Although this may compromise the power of cross treatment comparisons it is of less a concern for our analyses since we avoid making any cross treatment comparisons that include the control group.

The sample population was predominantly African (98% African, 2% Coloured), and Female (76%). Most individuals had only some secondary schooling and only 27% of them considered themselves to be either formally or informally employed. Interestingly, the average male in our sample was a full 4 years older than the average female¹⁰.

ii. *Choice of observable determinants*

In all 12 parameters were introduced in the theoretical section of the paper. Three of these (b , κ and δ) are assumed to be constant among individual, while g_i does not factor into our final model or any of our subsequent results, as long as we are willing to assume that optimizing patients would set it high enough to achieve their long run optimal level of guilt. Of the three endogenous variables (x_{1i} , x_{2i} and m_i), only x_{1i} and m_i are explicitly modelled. The choice of both will be covered in the next section. The remaining 5 parameters that were allowed to vary among patients are now introduced in turn:

Marginal Cost of Adherence (c_i) - Conceptually the marginal cost could include the price (both the ticket price and the opportunity cost) of a pill as well as side-effects. Weiser, et al, (2003) found that both of these effects pose a significant barriers to adherence in Botswana. However, since these pills are freely available in South Africa the ticket price is unlikely to play a major role in the adherence decision. We will focus on the role that side-effects play in effecting the marginal cost of each pill. In our sample 31% stated that they “do sometimes feel worse as a result of having to take ARV medication”, 15% said that the side effect are more than moderately disruptive.

*Short-Run Hyperbolic Discounting (β_i)*¹¹ – Patients are assumed to face identical long term discounting rates, but different hyperbolic discounting terms. We used a question on whether “you make yourself feel better by drinking or taking recreational drugs”. We use this measure to proxy hyperbolic time discounting, arguing that the benefits from these activities are immediate, while the cost usually follows a day or two later.

¹⁰ This is in line with the broader literature which suggests that sexually transmitted diseases peak earlier for females than for males (see Evans and Delva, 2009).

¹¹ Ideally, these two parameters are derived through the inclusion of questions that explicitly test time preferences by asking respondents to choose between receiving a fixed amount of money immediately or receiving a slightly larger amount with some delay (Tversky and Kahnema 1989; Shelley 1993). Unfortunately, our survey does not include any such questions.

Clearly, not all agents partake in these activities because of high hyperbolic discounting –some might just enjoy recreational drugs or alcohol outright, so much so that the benefits will always outweigh the costs regardless of time discrepancies. However, on average, one would expect these individuals, who we will loosely refer to as ‘drinkers’, to discount their short run more heavily than their counterparts who do not drink or do drugs.

Sophistication (s_i) - In the pursuit of a measure of sophistication, our paper draws on the complimentary research done on time inconsistencies and savings where rotating savings and credit associations are commonly used as informal commitment mechanisms, especially in developing countries (see Banerjee and Mullainathan, 2010). One such savings account, called ‘*stokvel*’, is especially popular in South Africa. In our study, we use affiliation to these ‘*stokvel*’s (as well as any other savings club or burial society) as a proxy for sophistication. Households who reported owing money or making regular contributions to any of these three groups were categorized as being sophisticated.

Inadvertent cost of the commitment mechanism (d_i) - There are two types of cost that enter our final model: the marginal cost associated with each additional pill and the inadvertent cost that the adherence officer inflict on patients which we denoted as d_i . We will restrict this parameter to only capture the discounted present cost of having ones HIV status unwillingly revealed to others. Allowing for the restriction, it seems plausible to assume that those patients who have not disclosed their status would have more to lose – at least in the short run – by having an adherence support buddy visit them on a bi-weekly basis, since it could raise suspicion among their friends and family. Consequently, we proceed to use a measure of disclosure to capture the parameter d_i . In the survey, patients were asked to list the three people who are closest to them and were then asked whether they have disclosed their HIV status to any of these individuals¹². We use this measure.

¹² Not everyone listed 3 individuals.

In Kind Incentives (f_i) - It is commonly believed that antiretroviral drugs have a greater impact (and less severe side-effects) if the patients are well nourished. In order to formally test this hypothesis, our survey included two treatment groups – one offering adherence support as a standalone treatment, and the other offering adherence support with an additional nutritional supplement. Since we are not directly interested in long term outcomes, but rather in pre-treatment take up, we will regard the addition of a nutritional supplement, which we will loosely refer to as ‘food’, as a tied in in kind incentive.

5. Results

i. Determinants of Adherence

Our primary interest is in estimating take up. However, we are also concerned with whether our chosen determinants pass the first hurdle: whether they correctly predict baseline adherence. Failure here would seriously undermine the credibility of either the chosen determinants or the initial model (equation 3), which forms the basis for the rest of our paper.

A dichotomous variable is constructed to capture adherence. Respondents who claimed to have never missed a dosage in the last month were taken as fully adhering, while those who have missed a dosage were taken as imperfect adherers. This measure deviates somewhat from the continuous baseline adherence level, x_{1i} , that we use throughout the rest of our paper. Our decision to use a binary measure of adherence is driven purely by the framing of the adherence questions within the questionnaire, where the questions were measuring deviations from full adherence and not adherence, *per se*¹³.

Table 2 measures the likelihood of full adherence. All four our models were run over our entire sample of respondents. Model 1 and 2 were fitted using Linear Probability Models, with robust standard errors. Model 3 and 4 were fitted using a Probit Model.

¹³ Adherence questions were phrased in a similar way to which Mannheimer (2006) proposes.

Table 2: Determining the level of Adherence

Adherence	(1)	(2)	(3)	(4)
Drink	-0.147*** (0.047)		-0.487*** (0.142)	-0.529*** (0.146)
Side Effect		-0.120** (0.050)	-0.402*** (0.153)	-0.387** (0.154)
Male				0.299** (0.148)
HIV Length				-0.005** (0.002)
Food Security				-0.045 (0.060)
Constant	0.838*** (0.016)	0.830*** (0.016)	1.053*** (0.071)	1.093*** (0.088)
Number of observations	634	634	634	634
Adjusted R ²	0.019	0.010	0.030	0.046

note: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Initially, adherence was regressed on side effect (which we use as a proxy for the marginal cost) and drinking (our hyperbolic discounting term). Both variables have the expected signs and were found to be significant, drinking at a 1% level and side effects at a 5% level. Drinkers and patients with side effects are respectively, 14% and 12% more likely not to be fully adhering (model 1 and 2). Both parameters remained significant (although only at a 5% level) when they were regressed simultaneously (model 3). Measured at the mean, being a drinker (a proxy for hyperbolic discounting) diminished the likelihood of fully adhering by roughly 15%, while the presence of side effects diminished full adherence by about 12%. Neither of the variables was markedly affected by the inclusion of other control variables¹⁴.

ii. Determinants of Take Up among Treatment 1

Some key determinants of take up were introduced in the theoretical portion of our paper. These propositions are now reviewed against our empirical data.

¹⁴ Within our sample sex appears to have a significant effect on adherence, while income (measured through the food security measure) does not. This is somewhat at odds with Weiser et al (2003)'s findings for Botswana. They found that socio-economics status and sex did not significantly impacted adherence to ARV's.

Table 3 shows a restricted analysis of only the first treatment group – the group that was offered adherence officers but not food. The first four models compare the take up rates among: drinkers and non-drinker (model 1), the more and less sophisticated (model 2), those with side effects and those without (model 3), and finally those who have disclosed their status and those who have not (model 4). Finally, we introduce our complete model (model 5) as well as an additional model (model 6) where we include some additional controls to test the robustness of our variables of interest. The first four models were run using a Linear Probability Model with robust standard errors, while model 5 and 6 were ran with a Probit Model.

Table 3: Determinants of Take Up

Take Up	(1)	(2)	(3)	(4)	(5)	(6)
Drink	0.256*** (0.056)				0.836*** (0.262)	0.892*** (0.269)
Stokvel		-0.096 (0.109)			-0.154 (0.286)	-0.094 (0.294)
Side Effects			-0.015 (0.082)		0.014 (0.233)	-0.042 (0.237)
Disclosure				0.161** (0.076)	0.369* (0.202)	0.402* (0.205)
Male						-0.309 (0.195)
HIV Length						-0.005 (0.003)
Food Security						-0.149* (0.085)
Constant	0.635*** (0.032)	0.687*** (0.030)	0.681*** (0.031)	0.549*** (0.070)	0.069 (0.187)	0.208 (0.201)
Number of observations	268	268	268	268	268	268
Adjusted R2	0.039	-0.001	-0.004	0.015	0.051	0.073

*note: *** p<0.01, ** p<0.05, * p<0.1*

We now review *propositions 1.a., 1.b., 1.c., and 1.d.*

Proposition 1.b holds. Drinking (our proxy for hyperbolic discounting) is positively associated with take up and is significant at a 1% level of significance. Those who drink are between 27% and 28% more likely to select into treatment, depending on which model one uses¹⁵. Although this might seem odd – that individuals who drink are more willing to let people into their houses

¹⁵ *In the case of the Probit, the effect was measured at the mean*

– it is in line with our theoretical prediction that agents with hyperbolic discounting have more to gain from a commitment mechanisms.

Proposition 1.c also holds. Disclosure is positively associated with take up (significant at either a 5% or 10% level, depending on which model one prefers). Those patients in our sample who had disclosed their HIV-status to the people closest to them were 16% more likely to select into the treatment program. Measured at the mean, moving from non-disclosure to full disclosure would increase take up by an estimated 13% (model 5).

The evidence for *Proposition 1.a* (which stated that people with larger marginal cost would have more to gain from the commitment mechanism) seems to be unconvincing, although it is worth noting that this effect is significant if we measure it over both treatment groups.

Proposition 1.d does not hold either. Sophistication (measured through ‘*stokvel*’ membership) did not have any effect on take up. The insignificance can be interpreted in more than one manner; either as evidence against the model or as evidence against our measure of sophistication. If we assume the first to be true, then there will not be any variation between sophistication and take up. However, if we take the latter to be true, then the variation within sophistication that was supposed to be captured by the observable determinant, ‘*stokvel*’, will now be captured by the error component¹⁶. Technically, this need not be of concern to us as long as we are not directly concerned with measuring the effect of sophistication; and we are willing to assume that the unobserved component within sophistication would be uncorrelated with all the other variables in our model. If we are not willing to make this assumption, it may well be biasing our results.

iii. Determinants of Take Up among Treatment 1 and Treatment 2

Before proceeding to compare take up across the two treatment groups, we first test for the balance between the two groups. Table A1 (appendix) compares the means and standard errors among an array of variables and reports the p-values for the t-test of equality of means across assignment groups for each. Two sets of covariates were tested: The top half of the table contains demographic variables, while second part of the table contains the observable determinants

¹⁶ Under the maintained assumption that sophistication enters the model via our presumed channel

which we will be modelling explicitly. None of the variables were found to differ at a 10% level of significance, allowing us except the assumption of balanceness across assignment groups.

Take up of the commitment mechanism, was regressed over both treatment groups. The first two models were fitted using the Linear Probability Model, while the rest were fitted using the Probit Model. Model 3 is our model of choice, since it follows straight from our theory and explicitly tests all our propositions. Interactions for all our variables of interest were introduced in model 4, to ensure that the interaction between food and drink is not proxying for other omitted interactions. Further controls are added in model 5.

Table 4: Determinants of Take Up

Take Up	(1)	(2)	(3)	(4)	(5)
Food	0.031 (0.040)	0.080* (0.045)	0.228* (0.125)	0.354 (0.269)	0.370 (0.272)
Drink		0.256*** (0.056)	0.852*** (0.262)	0.846*** (0.263)	0.908*** (0.269)
Drink * Food		-0.289*** (0.097)	-0.966*** (0.346)	-0.966*** (0.347)	-0.956*** (0.354)
Stokvel			-0.246 (0.190)	-0.163 (0.286)	-0.102 (0.292)
Side Effects			0.303* (0.173)	0.245 (0.191)	0.240 (0.194)
Disclosure			0.293** (0.146)	0.381* (0.201)	0.411** (0.204)
Stokvel * Food				-0.141 (0.383)	-0.154 (0.393)
Side Effects * Food				0.158 (0.205)	0.160 (0.209)
Disclosure * Food				-0.195 (0.293)	-0.207 (0.295)
Male					-0.224 (0.139)
HIV Length					-0.007*** (0.002)
Food Security					-0.111* (0.059)
Constant	0.679*** (0.029)	0.635*** (0.032)	0.095 (0.146)	0.027 (0.185)	0.179 (0.192)
Number of observations	527	527	527	527	527
Adjusted R2	-0.001	0.018	0.035	0.037	0.061

*note: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$*

Since patients were randomly allocated between the two treatment groups, we are able to test our proposition that food increases take up by a simple mean comparison. On aggregate the selection into treatment was only 3 percentage points higher for the group that were offered adherence and food rather than only adherence. The difference is insignificant at a 10% level.

While the addition of food did not have a large effect on take up when measured at the aggregate, it did have a significant effect on drinkers and it also had a significantly different effect between drinkers and non-drinkers. Both of these predictions are in line with our last proposition, which predicted that the addition of food will have a positive effect on take up that will be higher for those who discount the immediate future more severely (with lower β_i 's). Both these claims appear to hold in our data. Not only does food increase take up, but the interaction between food and drinking is also negative. Model 3 shows that the addition of in kind incentives increased uptake for non-drinkers by a larger amount than it did for drinkers. According to model 2, the uptake among non-drinkers was 29 percentage points higher among the group that were offered adherence support and food than those who were merely offered adherence support. The difference is significant at a 10% level.

Revisiting our first four propositions, we find that all our variables (and interactions) of interest, apart from sophistication, came out significant and with their expected signs (model 3). Although the estimated effect of disclosure had dropped from 36 to 30% it is still significant at a 5% level of significance. Interestingly, *Proposition 1.c* which did not appear to hold in on our smaller sample now holds in our larger sample. The proposition states that the marginal cost (which we measured through side effects) should be positively related to take up. Side effects were found to be significantly, measured at a 10% level of significance, associated with take up in our larger sample. Measured at the mean, those with moderate to severe side effect were roughly 30% more likely to take up the commitment mechanism. As before, the effect of sophistication, measured through 'stokvel' membership was insignificant.

Throughout our study we ignored the possibility that patients may be using their own informal self-commitment devices. However, these *ad hoc* measures appear to be fairly widespread. 60% of the patients in our study reported having a friend or a family member that remind them to take their medication. If effective, *ad hoc* commitment devices could be biasing our results, since those patients who have most to gain may already be getting adherence support elsewhere and in

doing so be less likely to select into our treatment. It doesn't undermine any of our findings though, since it would bias our results to zero. In this sense, the high prevalence of *ad hoc* commitment devices do not only highlight the necessity for more effective commitment mechanism, but may also be driving the insignificance of our sophistication measure.

6. Conclusion

In this paper, we discussed how hyperbolic discounting could lead to underutilisation of ARV-medication. Not all agents will be aware of the time inconsistency between what they desire to do in the long term and what actually end up doing in the short run. The more sophisticated, however, will and will develop a demand for a commitment mechanism that will enable them to fix their future level of long run adherence to a higher trajectory.

We go on to compare the take up choice among different types of HIV patients, drawing data from the baseline wave of an experimentally designed survey that was conducted in and around Bloemfontein, South Africa. As our propositions predicted, patients with higher marginal costs or instantaneous discounting (who have more to gain) were more likely to select into the adherence support program. The inadvertent cost of the commitment mechanism also plays a significant role in determining take up - those who have had disclosed their HIV status to fewer people were more reluctant to select into the program. Our measure of sophistication came up as insignificant.

Finally, we exploit the experimental variation that is brought about by the randomization in our survey design, to test how in kind incentives affect take up. We found that tied in incentives increase take up, but those who are drawn into selection through the additional incentives are likely to gain less through the commitment mechanism itself. The self-selection is compromised even further if incentives are large relative to the direct gain or if they are received in the future - since those agents with higher levels of hyperbolic discounting (who stand to gain more the commitment mechanism) would be the least enthralled by the delayed benefits. Any program that tries to induce adherence through the introduction of tied in incentives is likely to compromise efficiency by distorting the natural self-selection into these programmes.

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Appendix

i. Tables:

Table A1: Summary Statistics of variables, by treatment assignment

	Treatment 1	Treatment 2	P-value
<i>A. DESCRIPTIVE VARIABLES</i>			
African	99% (0.105)	98% (0.138)	0.447
Female	76% (0.429)	76% (0.430)	0.985
Finished School	65% (0.477)	62% (0.487)	0.402
Employed	25% (0.432)	28% (0.450)	0.378
Length of Disease (in months)	19.1 (24.937)	18.4 (25.473)	0.737
Age	37.0 (8.634)	37.7 (8.903)	0.384
Food Secure	44% (0.497)	46% (0.499)	0.661
<i>B. OBSERVABLE DETERMINANTS</i>			
Drink	10% (0.306)	8% (0.273)	0.356
Stokvel	8% (0.275)	11% (0.316)	0.247
Side Effects	31% (0.465)	31% (0.461)	0.835
Disclosed Status	81% (0.393)	82% (0.386)	0.795
Sample size	268	259	633

ii. *Derivations:*

Utility under optimal adherence (x_{1i}^* and x_{2i}^*):

$$\begin{aligned}
U_t(x_{1i}^*, x_{2i}^*) &= (-c_i x_{1i}^*) + \beta \delta \left(b_i x_{1i}^* - \frac{1}{2} \kappa_1 (x_{1i}^*)^2 - c_i x_{2i}^* \right) + \beta \delta^2 \left(b_i x_{2i}^* - \frac{1}{2} \kappa_1 (x_{2i}^*)^2 \right) \\
&= \left(-c_i \left[\frac{b_i}{\kappa_1} - \frac{c_i}{\beta \delta \kappa_1} \right] \right) + \beta \delta \left(b_i \left[\frac{b_i}{\kappa_1} - \frac{c_i}{\beta \delta \kappa_1} \right] - \frac{1}{2} \kappa_1 \left[\frac{b_i}{\kappa_1} - \frac{c_i}{\beta \delta \kappa_1} \right]^2 - c_i \left[\frac{b_i}{\kappa_1} - \frac{c_i}{\delta \kappa_1} \right] \right) + \beta \delta^2 \left(\left[\frac{b_i}{\kappa_1} - \frac{c_i}{\delta \kappa_1} \right] - \frac{1}{2} \kappa_1 \left[\frac{b_i}{\kappa_1} - \frac{c_i}{\delta \kappa_1} \right]^2 \right) \\
&= \left(\frac{c_i c_i}{\beta \delta \kappa_1} + \frac{\beta c_i c_i}{\kappa_1} - \frac{1}{2} \frac{c_i c_i}{\beta \delta \kappa_1} - \frac{1}{2} \frac{c_i c_i}{\kappa_1} \right) - \left(\frac{b_i c_i}{\kappa_1} + \frac{\beta \delta b_i c_i}{\kappa_1} \right) + \left(\frac{1}{2} \frac{\beta \delta b_i b_i}{\kappa_1} + \frac{1}{2} \frac{\beta \delta^2 b_i b_i}{\kappa_1} \right) \\
&= \frac{c_i^2}{2\delta\beta\kappa_1} (1 + \delta\beta\beta) - \frac{b_i c_i}{\kappa_1} (1 + \beta\delta) + \frac{b_i^2}{2\kappa_1} (\beta\delta + \beta\delta^2)
\end{aligned}$$

Utility under reneged adherence (x_{1i}^+ and x_{2i}^+):

$$\begin{aligned}
U_t(x_{1i}^+, x_{2i}^+) &= (-c_i x_{1i}^+) + \beta \delta \left(b_i x_{1i}^+ - \frac{1}{2} \kappa_1 (x_{1i}^+)^2 - c_i x_{2i}^+ \right) + \beta \delta^2 \left(b_i x_{2i}^+ - \frac{1}{2} \kappa_1 (x_{2i}^+)^2 \right) \\
&= \left(-c_i \left[\frac{b_i}{\kappa_1} - \frac{c_i}{\beta \delta \kappa_1} \right] \right) + \beta \delta \left(b_i \left[\frac{b_i}{\kappa_1} - \frac{c_i}{\beta \delta \kappa_1} \right] - \frac{1}{2} \kappa_1 \left[\frac{b_i}{\kappa_1} - \frac{c_i}{\beta \delta \kappa_1} \right]^2 - c_i \left[\frac{b_i}{\kappa_1} - \frac{c_i}{\beta \delta \kappa_1} \right] \right) + \beta \delta^2 \left(\left[\frac{b_i}{\kappa_1} - \frac{c_i}{\beta \delta \kappa_1} \right] - \frac{1}{2} \kappa_1 \left[\frac{b_i}{\kappa_1} - \frac{c_i}{\beta \delta \kappa_1} \right]^2 \right) \\
&= \left(\frac{c_i c_i}{\beta \delta \kappa_1} + \frac{c_i c_i}{\kappa_1} - \frac{1}{2} \frac{c_i c_i}{\beta \delta \kappa_1} - \frac{1}{2} \frac{c_i c_i}{\beta \kappa_1} \right) - \left(\frac{b_i c_i}{\kappa_1} + \frac{\beta \delta b_i c_i}{\kappa_1} \right) + \left(\frac{1}{2} \frac{\beta \delta b_i b_i}{\kappa_1} + \frac{1}{2} \frac{\beta \delta^2 b_i b_i}{\kappa_1} \right) \\
&= \frac{c_i^2}{2\delta\beta\kappa_1} (1 + 2\beta\delta - \delta) - \frac{b_i c_i}{\kappa_1} (1 + \beta\delta) + \frac{b_i^2}{2\kappa_1} (\beta\delta + \beta\delta^2)
\end{aligned}$$

Actual net loss in value due to reneging:

$$\begin{aligned}
V_{anti} &= U_t(x_{1i}^*, x_{2i}^*) - U_t(x_{1i}^+, x_{2i}^+) \\
&= \left[\frac{c_i c_i}{2\delta\beta\kappa_1} (1 + \delta\beta\beta) - \frac{b_i c_i}{\kappa_1} (1 + \beta\delta) + \frac{b_i b_i}{2\kappa_1} (\beta\delta + \beta\delta^2) \right] - \left[\frac{c_i c_i}{2\delta\beta\kappa_1} (1 + 2\beta\delta - \delta) - \frac{b_i c_i}{\kappa_1} (1 + \beta\delta) + \frac{b_i b_i}{2\kappa_1} (\beta\delta + \beta\delta^2) \right] \\
&= \frac{c_i c_i}{2\delta\beta\kappa_1} (1 + \delta\beta\beta) - \frac{c_i c_i}{2\delta\beta\kappa_1} (1 + 2\beta\delta - \delta) \\
&= \frac{c_i c_i (\beta\beta - 2\beta + 1)}{\kappa_1 2\beta} \\
&= \frac{c_i^2 (\beta - 1)^2}{\kappa_1 2\beta}
\end{aligned}$$

Perceived net loss in value due to reneging:

$$\begin{aligned}
V_{pnl} &= U_t(x_1^*, x_2^*) - U_t(x_1^+, x_2^+ - s(x_1^+, x_2^+)) \\
&= (-c_i x_{2i}^+) + \beta \delta \left(b_i x_{2i}^* - \frac{1}{2} \kappa_1 (x_{2i}^*)^2 - c_i (x_{2i}^+ + s(x_{2i}^+ - x_{2i}^*)) \right) + \beta \delta^2 \left(b_i (x_{2i}^+ + s(x_{2i}^+ - x_{2i}^*)) - \frac{1}{2} \kappa_1 (x_{2i}^+ + s(x_{2i}^+ - x_{2i}^*))^2 \right) - U_t(x_{1i}^+, x_{2i}^+) \\
&= U_t(x_1^*, x_2^*) - \beta \delta c_i s (x_{2i}^+ - x_{2i}^*) + \beta \delta^2 b_i s (x_{2i}^+ - x_{2i}^*) + \beta \delta^2 \left(-\frac{1}{2} \kappa_1 (+2x_{2i}^+ s(x_{2i}^+ - x_{2i}^*) + s(x_{2i}^+ - x_{2i}^*)^2) \right) - U_t(x_{1i}^+, x_{2i}^+) \\
&= -\beta \delta c_i s c_i \left[\frac{(1-\beta)}{\beta \delta \kappa_1} \right] + \beta \delta^2 b_i s c_i \left[\frac{(1-\beta)}{\beta \delta \kappa_1} \right] + \beta \delta^2 \left(-\frac{1}{2} \kappa_1 \left(+2 \left[\frac{\beta \delta b_i - c_i}{\beta \kappa_1} \right] s c_i \left[\frac{(1-\beta)}{\beta \delta \kappa_1} \right] + s^2 c_i^* \left[\frac{(1-\beta)}{\beta \delta \kappa_1} \right]^2 \right) \right) \\
&= \frac{c_i^2}{\kappa_1} \left(-s(1-\beta) + \frac{s(1-\beta)}{\beta} - \frac{s^2(1-\beta)^2}{2\beta} \right) \\
&= \frac{c_i^2 (\beta - 1)^2}{\kappa_1 2\beta} (2s - s^2)
\end{aligned}$$