Marital Shopping and Epidemic AIDS

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ABSTRACT

HIV risks decline sharply at age 30 for women in South Africa, long before coital frequencies or pregnancies decrease. I evaluate several prominent behavioral models of HIV, and find that these do not suggest sharply decreasing risks with age. This paper formulates a model of spousal search, and finds that it can generate epidemic HIV prevalence despite low transmission rates because search behavior interacts with dynamics of HIV infectiousness. The implied age-infection profile closely mimics that in South Africa, and suggested behavior matches that reported by South Africans. Condom use in new relationships and transmission rate reductions are both found to be effective policies, and used together eliminate the potential of spousal search to spread HIV. In contrast, ARV treatment is found to have only a minimal effect on the epidemic.

INTRODUCTION

A well-established consensus within the medical literature indicates that the average transmission rate of HIV is extremely low, on the order of 1 in 1000 per sex act (e.g. Gray et al 2001, Quinn et al 2000, Fideli et al 2001), or 8-12% per partner-year. Despite this, antenatal prevalence is 40% in Botswana and 25% in South Africa, suggesting that many have been afflicted by this extremely unlikely event. Unsurprisingly, social scientists, epidemiologists, and

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the popular press have each weighed in on what sorts of behavior could yield such an outcome, with behavioral models ranging from heterogeneous preferences over risk (Kremer 1996) to networks of concurrent sexual partners (Morris and Kretzschmar 1997) to unfaithful husbands (Kristof 2005). An implicit criterion in evaluating each of these models is that high HIV prevalence can be obtained despite low transmission rates.

This paper asks which of these stories could explain a majority of infections, and proposes that the age-profile of infection represents a second useful criterion in assessing behavioral models of HIV. Death registries in South Africa reveal a strong age trend: HIV infection risk declines sharply after age 30 for women (and 35 for men). I open this paper by discussing the age-profile of infection, and illustrate that this decline in risk occurs too early in life to be explicable simply by age-changing preferences, at least if these preferences are correlated with reported coital frequencies or pregnancy rates. I then argue that we can parse most existing behavioral HIV models into those which predict increasing risks with age and those which suggest age-independence (though a spurious age-decline may be generated by the absorbing nature of HIV infection). Intuitively, the sharp decline in risk levels with age seems unlikely to be generated by behavior where risk is age-independent or age-increasing; as a formal test, I calibrate a flexible model of age-independent heterogeneous risk and find that this class of models do not fit the age-death profile in South Africa well, suggesting that age-independent models do not explain how most people contract HIV[1]. As these models dominate age-increasing risk in terms of fitting the observed age profile of HIV infection, rejecting age-independent models rules out this latter class of models as well.

Instead, the age-death profile prefers a model of decreasing risk, with risks of new infection declining around age 30 for women (shortly after the median age of first marriage). This age profile, combined with an empirical trend that longer marital tenures are associated with reduced infection risks, suggests that the process of becoming married may be dangerous. This paper is the first to show that a simple search and matching model of serial monogamy is capable

[1]Elements of these models may all the same have severe epidemiological consequences, as variations in the behavior of a small fraction of individuals may cause huge swings in prevalence. I return to this point in the conclusion.

of generating high prevalence with relatively few partners because short periods of high partner turnover coincide with a brief window of high infectiousness immediately following infection. That is, epidemic AIDS is possible even when the majority of individuals do not engage in high risk behaviors. This paper then argues that a matching model of spousal search fits three additional empirical trends in the data: first, it generates an age-death profile which fits that observed in South Africa across the duration of the epidemic well for both women and men, a close fit which is robust to a large variety of parametric assumptions. Second, it closely predicts both the level and intertemporal variation in the annual number of partners that young adults report. Third, it suggests that spending a long time single should be associated with risk, but that the correlation between pre-marital sexual behavior and HIV should be eliminated for women who have been married for several years, a trend which is confirmed empirically across Africa. Finally, I evaluate the public health implications of Marital Shopping as an important vector for the spread of HIV by examining policies of transmission rate reductions, short-term condom usage, and Anti-Retroviral (ARV) treatment. Short-term condom usage is found to be the most effective of these strategies, and it eliminates the potential of spousal search to create an HIV epidemic when combined with the lower transmission rates associated with the US and Europe.

HIV IN SOUTH AFRICA

Much of the data which could be used to learn about covariates of HIV infection is suspect. Antenatal prevalence data is subject to selection bias, population survey data have high refusal rates, and cause of death statistics may be rigged. Changes in overall death rates in areas where the epidemic hit suddenly and recently, however, are immune to these concerns. Figure one presents deaths by age and gender observed in South Africa, where death registration is relatively complete (Dorrington et al 2001, Statistics South Africa 2005). Epidemic AIDS is a recent phenomenon in South Africa, where antenatal prevalence was about 1% in 1990 (RSA 2008, US Census 2006). This is reflected in the bottom line of each figure, which shows a relatively flat deaths-by-age profile in 1996. The top line, in contrast, tells a different story. By 2002, the number of 30-40 year old women who died had more than tripled in six years. There is a much slighter increase in deaths for 40-45 year old women, and by age 50, the death rate looks very similar to the historical one. For men, the age-death profile also peaks, though this happens five years later and the decline is less sharp. If we knew the precise time path of prevalence in

South Africa, we could infer at precisely which age infection risk peaks. We don't; however, an approximation can be found by subtracting 10 years from the death rates as individuals survive, on average, about 10 years after infection. This approximation suggests that women are at greatly reduced risks of infection after age 30[2].

EPIDEMIOLOGICAL MODELS OF HIV AND AGE

The fact that women in South Africa face risks that decline quickly with age could be driven by several factors unrelated to spousal search. First, this correlation could be driven by some biological factor of HIV transmission which is correlated with age. Second, it could be a consequence of changes in sexual risk that takes place as adults age. Third, it could be the direct outcome of an existing behavioral epidemiological model.

Biological Correlates of Age

For the age-trend in infection observed in South Africa to be a suitable criterion to evaluate behavioral models, it must not determined by a biological component. Other sexually transmitted infections increase the transmission risk of HIV (e.g. Oster 2005) which may have age implications. In fact, the observation that young people are far more likely to contract sexually transmitted disease is not unique to HIV or to Africa. Whether Gonorrhea or Chlamydia in the US (CDC 2005), Human Papilloma Virus in Costa Rica (Castle et al 2005), or Herpes Simplex Virus type 2 (HSV-2) throughout the world (Smith and Robinson 2002), the young are consistently the population who contract STDs. Despite this observation, the presence of other sexually transmitted diseases actually suggests an age-increasing risk profile for HIV infection. There is mixed evidence on whether non-ulcerative STDs affect HIV transmission at all, and it is widely believed that ulcerative sexually transmitted diseases play a much larger role (e.g.

[2]This trend is not unique to South Africa, nor is it a recent observation. Though good mortality records do not exist for other countries in sub-Saharan Africa, authors in individual medical studies have found similar age-death profiles from HIV in Uganda (Sewankambo et al 2000) and the Democratic Republic of Congo (Pictet et al 1998). Anderson et al (1991) provided an early documentation of this age-pattern in an influential review. Despite the long-standing awareness of this trend, previous work has not used it to assess the plausibility of behavioral models to the author's knowledge.

Fleming and Wasserheit 1999). The most prevalent ulcerative STD in Africa and the world is HSV-2 (e.g. Chen et al 2000, Wawer et al 1999). Therefore, the HIV risks which are induced by other STDs should follow the age-profile of HSV-2. As HSV-2 is incurable and non-fatal, the highest prevalence is among the old (documented empirically across Africa in Smith and Robinson (2002)). Thus, STDs as risk-factors suggest that older men and women should be at an elevated risk of HIV infection, not a much lower one.

Age-Changing Preferences

A second possibility is that this age-trend is an immediate outcome of age-changing preferences over coital frequencies. While thirty seems a very young age for a sharp decrease in preferences over coital frequency, two forms of evidence are available to evaluate this possibility, both available in the South African Demographic and Health Survey (DHS). The first is self-reports of sexual behavior, which is presented in Panel A of Table one. For all potential coital frequencies, we see reported sexual behavior increasing until age 40, after which it remains higher than the reported behavior of women in their 20s, which are the ages that the death profile tells us involve peak HIV risk.

However, sexual behavior data is notoriously difficult to measure, and women in particular have been shown to misreport badly (Gersovitz et al 1998). Therefore, we may be concerned that these differences simply reflect differences in reporting bias, and pregnancy may be preferred as a biomarker. Panel B of Table one reports pregnancy rates by age. Indeed, pregnancy rates do decline with age after age 30 as reported in column one. However, natural declines in fecundity also begin around age 30 (te Velde and Pearson 2002). Table one shows births per year divided by the probability of conception for that age found in two studies, Templeton et al (1996) and van Noord-Zaadstra et al (1991)[3]. As Panel B of Table one shows,

[3]Templeton considers all in-vitro fertilizations in Britain from 1991-1994, while van Noord-Zaadstra considers all artificial inseminations in two fertility clinics in the Netherlands during the clinic-specific period when fresh (rather than frozen) semen was used (1973-1980 for one clinic, 1973-1986 for the other), and restricts analysis to married women whose husbands were azoospermic and who had never previously given birth or received an artificial insemination. Sample sizes are tiny in both studies for women over 40, so the adjustment may be unreliable for these groups.

the actual births per year are in between what the two estimates would predict if sexual behavior remained constant, at least for women under 40. These data do not support the hypothesis that women's age-specific preferences over coital frequency are insulating them from their husbands' behavior at older ages, nor that women begin to prefer lower coital frequencies at an early age[4].

Existing Behavioral Epidemiological Models of HIV

Because average HIV infectiousness is so low, models which achieve high HIV prevalence with feasible amounts of sexual behavior often rely on fundamental heterogeneity between individuals. The class of models which has dominated the economics literature revolves around individuals having heterogeneous preferences of sexual risk or variety (e.g. Kremer 1996, Philipson and Posner 1993). In Kremer's specification, heterogeneous individuals optimize a rate a partner change. A high risk group seeks out many partners, while lower risk individuals only occasionally search for new partners, and partnerships form at random. These high risk individuals are overrepresented in the pool of available partners due to their greater frequency of partner search, increasing the chance of drawing a partner with above population-average risk.

This model belongs to the class of age-independent risk models. While different individuals prefer different risk levels and some may be likely to become infected earlier in life than others, any uninfected individual will face an identical probability of infection in each period she remains uninfected. Another age-independent model emphasizes sexual networks of concurrent partners (Morris and Kretzschmar 1997). Once HIV is introduced to a network, it can spread quickly throughout the network, exposing all members to high risk. As such, all members of a network are exposed to very similar risk levels, regardless of age. Presumably, individuals are heterogeneous with respect to the size and turnover rate of their networks, so we may expect this model to produce very similar age predictions to the preference-based model[5].

[4]This analysis cannot rule out age-changing preferences over sexual variety, a possibility discussed at greater length below.

[5]One may imagine many ways by which network formation may covary with age. However, the assumptions needed for networks to create age-decreasing risks are strong. All members of a network are at similar levels of risk, so if network members are both young and old then we have age independence. If networks of partners are stratified by age, one could imagine a situation where uninfected older individuals match separately from more infected younger people.

Can an age-independent risk model with heterogeneous types generate an age-death profile like South Africa's? In principle, a two type model might: high risk types become quickly infected and die out while still young, while low-risk types remain safe. However, this sort of explanation makes two extreme assumptions: first, there are no middle risk types, who likely survive their youth and continue to be probabilistically infected later in life; and second, that the epidemic is in its steady state. Self-reports of the number of sexual partners in the last year from 14-22 year olds in South Africa, though subject to the concerns discussed in the previous section, seem to contradict the first assumption[6]. As reported in columns two and four of table two, young adults in South Africa report a triangular distribution of annual partners, with the majority being low-risk. More importantly, epidemic stability is necessary as this age-independent explanation revolves around the high-risk older women having already attrited from the population, which would not have happened at the onset of the epidemic (as in South Africa's case).

Age-Independent Model Calibration

To evaluate whether an age-independent model is capable of generating the age-death profile observed in South Africa, I calibrate a model with up to three types. Each type is indexed by an annual risk level so that one is at high risk, one at low risk, and one at negligible risk. It can be shown in a similar calibration (results available from the author) that if the majority of at-risk people are the low-risk type, only a bad fit can be generated in the steady-state. Here, I illustrate that the epidemic is too young in South Africa to generate an age-death profile like the one observed, regardless of the distribution of these three types. Let $\rho_g(y)$ be the infection risk of group g in year y, f_g be the fraction of individuals who belong to group g, $X(k)$ be the survival rate into sexual activity at age k, and $S(t)$ be the survival rate t years after infection. Then, the number of deaths at age a would be

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However, the dynamics of these networks are very difficult to justify. For example, if everyone in one network matches only with people born before January 1, 1970 then such a network could exist, but not if everyone matches with people who are within one or two years of their age.

[6]The South African Data Source for this table is wave 1 of the Cape Area Panel Study, which is described below.

$$D(a,2002) = POP * \sum_{g=1}^{2} \sum_{\tau=0}^{a-k} \sum_{k=0}^{a-1} \left(X(k) - X(k+1)\right) f_g \times \left[\prod_{y=2002-(a-k)}^{2002-\tau} \left(1 - \rho_g(y)\right) \right] \times \rho_g(2002-\tau)\left(S(\tau) - S(\tau+1)\right)$$

where POP is the population of reproductive-aged women in South Africa who are at some risk of infection. POP is a free parameter, which I choose so that the peak of the two distributions are equal. To insure that the high risk and low risk group are different, I allow ρ_1 to reach a peak incidence of between 0 and .2 (.2 is the highest considered as this is the annual risk from unprotected sexual activity with an infected spouse among highly sexually active young couples (Gray et al 2001)), while I restrict ρ_2 to be between 0 and .02 to create a low-risk group. Two percent per year is a high level of "low" risk; it creates more than a 50% lifetime chance of infection. I further assume that $\rho_g(y)$ increases linearly from the first year of the epidemic (t_0) until it reaches peak risk, at which point it remains at that level until 2002[7]. This creates a vector of four parameters, $(\rho_1, \rho_2, f_1, t_0)$ which are free in this model; I then evaluate the best possible fit of this age-independent risks model by choosing these parameters to minimize sums of squared deviations between the actual deaths data and that suggested by the model. I further examine the epidemic at a variety of ages. Fifteen years is about right for South Africa in 2002, as antenatal prevalence rates were about 1% in 1990. (RSA 2008, US Census 2006).

Figure two shows that a 35 year old epidemic can fit the age-death profile quite well -- the intuition that, if everyone at risk is at high risk, the risky die while young leaving behind safe older people holds. However, when the infection is young, it cannot. In fact, around year 15, the highest risk is amongst the oldest groups, in contrast to what we observe in the data. In other words, if people belong to one of three risk types, and those risk types remain relatively constant over lifetimes, the no matter how much risk each of those types contain, or what fraction of the population belongs to each of those types, we should observe far more older people being infected in early stage epidemics than we actually do observe in South Africa. The reason for

[7]Results are not sensitive to making this piece-wise linear specification more flexible.

this is simple, and not driven by the stylized assumptions that make this calibration tractable: at the onset of an epidemic, older individuals of all risk levels are being exposed for the first time. Therefore, if risks are independent of age, the first cohort exposed includes risky people of all ages and should feature deaths at all age groups. In South Africa, the epidemic is simply too new for an age-independent risk model to be a strong candidate explanation for most infections.

Marriage as a Risk Factor

Given that age-independent models of heterogeneous risk are unable to generate South Africa's decline in risk with age, we can be certain that age-increasing models are poor approximations to the behaviors by which most people contract HIV. As argued above, this suggests that the presence of other sexually transmitted diseases do not serve as a stand-alone story for the age-death profile in South Africa. Another story which suggests age-increasing risks is the possibility that men bring HIV risk into marriage through extra-marital affairs. Kristof argues this case, writing "The stark reality is that what kills young women [in Africa] is not promiscuity, but marriage. Indeed, just about the deadliest thing a woman in Southern Africa can do is get married" (2005). However, since older (and more often married) women are even safer in South Africa than they would be if marriage had a neutral effect on risk, we can rule out the cheating husbands story as being the mechanism by which most women become infected.

This story is so pervasive that it is worth reviewing the evidence which has led to it. There is clearly some risk associated with a long term partnership, for both men and women[8]. However, a lack of long-run panel data, combined with mortality-related attrition and low HIV transmission rates has left us without a good sense of how that risk compares to the risks of pre-marital behaviors. Much of the evidence on extra-marital risk has come through anecdotal and ethnographic channels, which are hard-pressed to answer questions of relative magnitudes. Even if a large fraction of adults have extra-marital partners, risks may be low if the existence of a spouse prevents high frequency sex with these partners. Empirical evidence on marital risk has been mixed, with Clark (2004) finding that married teenage girls have higher prevalence than their unmarried peers, concluding that marriage is risky. In contrast, Bongaarts (2006) finds that

[8] De Walque (2007) documents that in a large fraction of infected couples, only one member (as often the woman as the man), is infected.

marriage is less risky than being sexually active and single, and Glynn et al (2001) document that married women who avoided premarital sex have lower prevalence than their married peers who did not.

The idea that married women may have higher prevalence than unmarried women is consistent with the idea that a year of marriage might be less risky than a year of being sexually active and single if the process of becoming married is dangerous, but staying married is relatively safe. Individual-level data with HIV status is not publicly available for South Africa, preventing a direct examination of this hypothesis. However, in all of the high prevalence countries with publicly-available DHS data including serostatus, there is suggestive evidence that it is indeed the process of becoming married which garners risk. If becoming married carries much risk, then we should observe surviving couples being more likely to test jointly negative if they have been married for long enough that preexisting infections should have resulted in death. In the appendix, I show that monogamous couples who have been married for at least 10 years are substantially (and statistically significantly in every country save Lesotho) more likely to test jointly negative than couples who have been married 0-5 years, a trend which is robust to flexible age and spousal age controls and in some cases represents truly substantial risk reductions. Across Africa, there is evidence that the process of becoming married, but not being married, is risky.

SPOUSAL SEARCH

The process which leads to marriage has changed substantially in sub-Saharan Africa over the last 50 years, just as it has elsewhere in the developing world. A large body of research has emphasized globally that traditional, kinship-based marital institutions are being replaced by western notions of courtship, where individuals must identify for themselves suitable partners to marry (e.g. Ghimire et al 2006, Thornton et al 1994; for African examples see Mukiza and Ntozi 1995, Smith 2000). Recent research has more directly emphasized the parallels between Western notions of courtship and premarital behaviors in Africa, where researchers have documented that young people engage in sexual relationships as part of a search for long term monogamous relationships with psychologically compatible partners (Clark et al 2010) and that the likelihood of marriage is important in the choice of sexual behavior among premarital partners (Clark et al

2009)[9].

Model

The process of searching for a compatible spouse, where compatibility is hard to observe, can be easily understood through adapting Jovanovic's (1979) classic model of job turnover. The idea is simple: if there is some unobserved and unpredictable component to the quality of a relationship between two people, then individuals need to "try out" a relationship to learn its quality. Once a person learns the quality of a current relationship, she can compare that quality against how well she could expect to do by ending that relationship and looking for a new partner. Formally, suppose individuals live for T periods. Each period, individual i receives utility $\theta_{ij} + q_j$ from a match with partner j, where $\theta_{ij} \sim F(\theta)$. q_j is the observable component of quality which is known before the relationship, and θ_{ij} is match-specific and unobservable. She faces a choice at the end of the period: to stay with partner j or to draw partner j', with whom $\theta_{ij'}$ is unknown but $q_{j'}$ is known[10]. Match quality evolves stochastically according to $H(\theta)$. Individuals match assortitatively on the q_j; in practice, this observed element drops out of the optimization problem as it is constant among any mutually acceptable suitor. Therefore, each individual solves the dynamic programming problem with Bellman equations

$$V_t(\theta_{ij}) = \max_{\{stay,leave\}} \left\{ \theta_{ij} + q_i + \beta E\left[V_{t+1}\left(\theta'_{ij}\right) | \theta_{ij} \right], E\left[\theta_{ij'} + q_i + \beta V_{t+1}\left(\theta'_{ij'}\right) \right] \right\}$$

[9]The African studies cited here are from Nigeria, Uganda, Kenya, and Malawi. I am not aware of a similar documentation in South Africa, though as researchers frequently hypothesize that it is exposure to Western culture and the development process which leads to this shift we may expect the courtship and marriage paradigm to be at least as important in South Africa as in other parts of Africa.

[10]Individuals in this model draw only one partner at a time. While age-independent concurrencies perform poorly against the age-risk profile, search with multiple concurrent draws would have similar predictions to this model, with even more severe epidemic prevalence implications.

where β is the discount rate and θ'_{ij} is the random variable representing next year's match quality with partner j.

To solve this model, individuals solve backwards. In the second to last period, each person faces a choice between staying with their current partner (for who θ_{ij} is known, and θ'_{ij} can be predicted with some accuracy), or finding a new one. Therefore, she stays with the current relationship if $E\left[\theta'_{ij}|\theta_{ij}\right] > E\left[\theta'_{ij'}\right]$, or if her current relationship is better in expected value than she could do by drawing a new one. Otherwise, she ends her current relationship and finds a new partner, indexed j', from whom she receives match-specific utility $\theta_{ij'}$ in her last period of life. The previous period, the choice is slightly more complicated; here, she stays if the expected value of the current relationship -- including the option value of being able to end that relationship in the following period -- is larger than the expected value of a new relationship, again considering the possibility that that relationship could be terminated after one period. Iterating back to the first period leads to the well-known solution for search models, where optimal behavior is to form a sequence of reservation qualities, $\bar{\theta}_t$, where individuals stay in any relationship where $\theta_{ij} > \bar{\theta}_t$. In this specification, $\partial\bar{\theta}_t/\partial t < 0$, so that as individuals age, reservation qualities lower. This means that the probability of a good relationship turning sour enough to dissolve after a certain age becomes low -- this fact creates "marriage" behavior. In my preferred specification, match evolution is slow, so that the first period provides a fairly accurate measure of the quality of a relationship; this creates behavior which mimics the observed 9 year time-span from sexual initiation until marriage in South Africa. This evolution can be interpreted as truly evolving utility from a partner or as a learning process. In any matching model, a large fraction of relationships are rejected immediately, as reservation qualities are always bounded below by the expectation of the quality of a new match. As a result, individuals have clusters of several short relationships in between a few much longer ones; this will turn out to have disastrous consequences for the HIV epidemic. I define marriage as occurring at the date when a person begins to match with her (ex post) last partner. Men and women match randomly with someone else who is actively searching for a new partner. After 20 years, individuals quit

searching, with a payoff of 10 years' worth of utility at the current match quality[11]. Men and women are identical in this model; this assumption is supported by de Walque (2007), who observes that both women and men are the only infected member of a large fraction of couples with only one infection.

Integrating HIV into the Matching Model

The average rate of infection per partnership year (PPY) has been documented through longitudinal studies in Uganda and Zambia. By following sero-discordant couples (where only one member is HIV-positive) through time, medical researchers observe transmission rates. The studies in Rakai, Uganda, are particularly compelling as participants report minimal condom use despite counseling. These studies estimate average infection rates of about 12% PPY (Gray et al 2001, Quinn et al 2000), which corresponds to an approximately 1 in 1000 infection rate per contact. Transmission in Lusaka, Zambia, where higher condom usage was reported, was lower (8% PPY) (Fideli et al 2001).

However, the average infection rate may not be a sufficient statistic to understand the dynamics of HIV transmission. PPY rates do not describe within-individual or between-individual heterogeneity in infectiousness, and there is substantial evidence that both are important for HIV. Many studies (e.g. Gray et al (2001), Quinn et al (2000), Pedraza et al (1999)) have documented the correlation between viral load and infectiousness. In terms of viral load, we can divide a person's HIV infection into three broad periods. Acute infection lasts for the first two to three months. The body has not developed an immune response to HIV, and viral load is high. In this period, HIV may be 10 times as infectious as it will be later (Pilcher et al 2004, Wawer et al 2005). Next, in latent infection (the next eight years or so), the body's immune response keeps viral load extremely low. Finally, the body's immune system starts to

[11]In a more sophisticated model, individuals may consider the state and future path of the HIV epidemic in making their spousal choices. However, if individuals have rational beliefs about HIV, the distortion in behavior is minimal. As the risk from drawing a new partner is extremely close to the risk from staying with the current partner when you are still early in a relationship, individuals are only very marginally willing to lower their reservation in response. Given that the rational difference is extremely small, and that I have no way to assess what South Africans actually believe about the transmission rate or the future path of the epidemic, I abstract from this analysis.

fail at containing the virus and the infection becomes mature. Starting about 2 years before death, viral loads begin climbing. As viral loads increase, AIDS breaks out causing death within about a year without medical intervention (e.g. Katzenstein 2003). People in the longitudinal studies described above are primarily in the second phase of infection, which is why low average transmission rates are consistently observed. In the epidemiological model most similar to this one, Koopman et al (1997) assume individuals transition randomly between high and low-turnover states and match non-randomly with others in the same state. Considering homosexual infection in the US, and assuming very risky behavior, they find that shutting down acute infection may end the epidemic entirely[12].

Therefore, it is important to take into account the dynamics of HIV infection. I specify that if an individual of gender g matches with a partner is in phase k of infection, they face probability ρ_k^g of infection. Once infected, individuals die according to the survival function in UNAIDS (2004). Two years before death, individuals enter mature infection (following Wawer et al 2005). I do not allow individuals whose partners have died from HIV to find a new match, unless they would have resumed searching from the relationship's evolution in any event. In each cohort, a small percentage (δ) enter already infected with HIV, with $\delta/6$ entering in acute infection[13]. This consistent injection of HIV can be interpreted as infection from all sexual behaviors other than spousal search. Individuals who are initially infected do not behave differently from other individuals.

Each period corresponds to a month. Latent transmission rates are those measured by

[12]Koopman et al's model is a more traditional epidemiological model than the one described here and as such differs strongly in focus. There, transitions between phases of high and low sexual activity are not brought about by any behavioral process but rather outcomes of exogenous (and calibrated) chance. Similarly, systematic matching between individuals in different phases is specified rather than the outcome of a behavioral process like spousal search. This makes it more difficult to derive policy predictions like those employed in this paper, as the motivations for risky behavior are not identified. Koopman et al also assume a higher transmission rate and an order of magnitude more partners than those employed here, with individuals in their model accumulating a staggering 2 new partners per month.

[13]1/6 was chosen as I find from simulations that about 1/6 of single, HIV-positive individual-months are spent in acute infection for those who don't enter already infected. Varying this fraction has only a small impact on results.

Gray et al (2001) for young couples (slightly above average rates due to the greater coital frequency of young couples) whereas acute and mature transmission rates follow Pilcher et al (2004) and Wawer et al (2005)[14]. The distribution over θ is uniform and chosen for simplicity; changing to other simple distributions alters lifetime numbers of partners and HIV prevalence only slightly, as reservation qualities endogenously adjust downwards when good matches become more scarce. Men and women are identical in this model, and as such have similar simulated data; I present only the results for simulated women.

Simulating the Model

To simulate this model, I first solve the dynamic programming problem numerically (distributional assumptions and summary statistics from the simulated data are given in the appendix). Then, I simulate N men and N women in the first cohort, each of whom randomly draws a partner from the simulated opposite sex and receives a draw of partner quality. At the end of the period, they decide whether to stay in the relationship or draw a new one based on the solution of the dynamic programming problem (if either individual prefers to end the relationship, it ends). If they stay, their match quality evolves and they face the same decision in the subsequent period. If they leave, they receive a new partner from the pool of other individuals who choose to search and a new draw on θ. The outcome of this random process is that individuals stay single for about 8.5 years, which is close to South Africa's empirical data (the median South African woman becomes sexually active at 17 and married at 25). Over the 20 years of sexual activity, the median person has 11 partners. Eleven partners over 20 years is low relative to many epidemiological models, which typically assume that individuals accumulate at least 1-2 new partners per year (e.g. Morris and Kretzschmar 1997, ASSA 2005, Oster 2005) and sometimes assume much more (e.g. Koopman et al 1997). Every ten months, a new cohort of N individuals of each sex enters the pool of searching singles and similarly draws a partner and a

[14]It is natural to suspect that coital frequencies decline as people become increasingly sick in mature infection. Wawer et al (2005) follow transmission rates and coital frequencies approaching death, and find that coital frequencies do decline gradually over 6-30 months before death, from a rate of 10 per month earlier in infection to a rate of 6 per month 6 months before death. However, transmission rates increase faster than coital frequencies decline, and Wawer et al document that transmission happens about 3.65 times as frequently over months 6-30 prior to death. I use that number in this calibration.

match quality. I simulate the model for 40 years; I do not explicitly model homophily in age so that entering cohorts are treated the same as single individuals in existing cohorts (though of course their age is different, resulting in a different solution to the search problem). With the entry of the 10th cohort, the HIV epidemic enters, and all living individuals face instantaneous probability δ of contracting HIV[15]. Following, each new cohort enters with prevalence δ, and HIV transmission occurs as HIV-negative partners of HIV-positive individuals face random draws against the transmission rate. There are 2 sources of randomization in this model: the match process and the HIV epidemic. Once the match has been solved, the spread of HIV happens in a way which is quick computationally. All results presented are for 50 realizations of the match, each averaged over 10 realizations of the epidemic.

RESULTS

Table three reports the fraction of fully exposed women who will become infected for each δ. Marital shopping acts as a multiplier of 5-7 on inputted infections, so that if each percentage point of inputted infections results in about 6% of people becoming infected. In other words, each person who enters the spousal search period infected will cause about two to two and a half other single people to become infected, and all of them go on to infect the person they eventually marry. Thus, if 1% of the population enters the spousal search period infected, we end up with a prevalence rate similar to that in Kenya or Tanzania. With just 3% infected by all sexual behaviors other than spousal search, we are at South Africa's epidemic prevalence rates. The reason for this is basic behavior generated by matching models (and hence fairly insensitive to parametric assumptions). Individuals have clusters of several very short relationships which are

[15]Since cohorts must build up over time, HIV entry is delayed to minimize the effects of time dynamics in homophily in age (simulations suggest that this time-delay has a slight and conservative effect on HIV prevalences). In fact, the role of age-specific homophily in the spread of HIV is somewhat unclear -- if people match non-randomly with others in a similar age-category, this may lead to more explosiveness in the HIV epidemic as people in high turnover phases with greater likelihood of acute infection match systematically with others in the same phase. This observation makes the role of homophily in the spread of HIV an important avenue for future research, particularly as it interacts with "relative risks" messages (Dupas 2007) which persuades girls that older men have higher prevalence (and hence that younger boyfriends are safer). Fortunately, the lower pregnancy rates found by Dupas (2007) for girlfriends of young and presumably high-turnover boys suggest that lower fertility demand may overwhelm this concern.

easily rejected in between a few much longer ones. Therefore, when they enter a new relationship, they are likely to have just left another very short one. In turn, this means they are much more likely to have just been infected, remain in the acute phase of infection, and hence more likely to infect their new partner. Prevalence among the first 6 cohorts reaches 80% of its eventual peak (not presented here). Thus, with a tiny fraction of the population entering the spousal search phase of their lives infected, marital shopping can create an epidemic quickly.

To explore the importance of acute infection, Rows 3 and 4 of table three report prevalence results if the acute infection transmission rate, ρ_1 were 0. The results are strong: acute infection is responsible for a large fraction of the infections caused by spousal search, including about a third of infections for δ<0.05. Now, δ is multiplied by 4-5 rather than 5-7, so that each initially infected person only causes about one to one and half other infection before both people infect their spouses. This thought experiment is interesting for two reasons. First, it supports data from Switzerland (Yerly et al 2001) which has shown that over 1/3 of infections are attributable to acute infection (Wawer et al 2005 report similar findings in Uganda). Secondly, it is implementable. A policy of wearing condoms for the first three months of each relationship would stop new infections caused by acute infection. While a preference for condoms in short relationships is familiar and intuitive, it represents a sharp divergence from the current message being spread by many NGOs, who promote condom use throughout marriage (e.g. Ali et al 2004). This policy is evaluated more explicitly below.

Theoretical predictions and data can be used to determine the age-profile of infection and death suggested by spousal search with minimal reliance on parametric assumptions. More specifically, matching models imply that infection risk is more or less constant for the uninfected single[16]. When married, in contrast, it declines exponentially -- since no new infections are

[16]This is true to a gross approximation. With most specifications, the declining reservation quality with age is relatively slight up until very near mortality; this has consequences for marginal relationships only (though the odds of marriage can be boosted by not terminating during a few stochastically marginal periods). As I show below when I turn off transmission at the beginnings of relationships, much of the single risk enters through "bad" relationships that are easily rejected; this behavior is hardly affected by declining reservation quality with age. For example, in the preferred specification, about 71% of relationships last only one month in years 1-18 of a 20 year lifespan (s.d. 0.015) based on 2500 observations of simulated lifespans, and there is no statistical age trend across these years.

brought into a marriage, your risk at any point in time is the product of the transmission rate, the risk that your spouse entered the marriage already infected, the chance that you entered the marriage uninfected, and the probability that you haven't caught it from your spouse yet. Similarly, the time path of risk suggests the relative fraction of infections occurring to the single and married. The algebraic identities suggested by these predictions are straightforward if tedious, and are contained in the appendix.

Using the South African DHS, I estimate Kaplan-Meier hazard functions into marriage and into sexual activity to estimate search and marriage behavior, with the assumption that being unmarried but having had sex is reflective of being single and searching[17]. As the DHS only provides data on women, I estimate the men's hazard function into marriage by taking means by age of the fraction married from the September 2001 South African Labour Force Survey, and I alternatively assume that the hazard function into sexual activity is the same for men as it is for women, or that it is shifted to five years older as South African men marry five years later than women on average. As described above, I know the risk profiles for the single and for the married if I know the time path of the epidemic and the relative prevalence rates for newlyweds and singles. Therefore, to construct the age-death profile, I need only the time paths of the prevalence for single people and for newlyweds. These are taken from simulations; reassuringly, the figures below are robust to reasonable changes in both of them as it is the empirical distributions of singlehood and marriage as well as the theoretical risk predictions which produce the close fit.

Figure three presents this comparison. Here, I make the further assumption that the number of deaths by age in 1996 represents the number of non-AIDS deaths in 2002, and I set the peak of simulated AIDS deaths equal to the empirical peak. That is, the height of the curves is rigged; however, that is the only point which is expressly fit and the shape of my predicted

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[17]These create an integer problem as you are only sexually active for some fraction of the year in which you first have sex. Because the age at which you first have sex is the absolute earliest you could start risky search, I assume that young adults begin searching (and hence reach the full single risk level) in their first full year of sexual activity. However, the potential for heightened pre and post-nuptial coital frequencies suggest that married individuals may start on the married risk rates in the year in which they get married; the figures presented reflect this assumption. Resolving the integer problem in different ways results in very similar fits.

age-death curve is derived from the theory and the data. As the reader can observe, the fit is very close[18]. I very slightly underpredict older women's deaths. Recalling that this model assumes perfect monogamy, extramarital sex may create this difference; however, the quantity of infections that this non-monogamy produces is tiny in comparison to the number of monogamous infections. For men, the data lie in between the age death curves created by assuming that men have the same sexual initiation as women and that assuming that their sexual initiation is five years older. Still, the fit is overall very close, and distinguishable from the fit for women. Men marry five years later than women and over a broader range of ages, and that is precisely how they die.

There is no reason to expect that the marital shopping model would only fit the epidemic in 2002. Figure four estimates the model for every year, 1997-2002. For men, I take the average of the predicted results from the two assumptions on sexual initiation. For both men and women, at all years of the epidemic, we find a close fit (once again, only the peak of each year is fixed). Moreover, some of the time dynamics in the age-profile of infection are preserved between the model and the data, not least being the age-peak from early years which was absent in the heterogeneous risk model. Regarding the female age-prevalence profile, we see that at the onset of the epidemic, peak deaths occurred in the 25-30 age group, with a similar rate among 30-35 year olds, whereas by 2002 30-35 year olds experienced more deaths. This is predicted by the marital shopping model.

Marital Shopping or Preferences over Variety?

In the marital shopping model people are motivated by the utility payoff of finding a high quality partner. They undergo search in their youth which is characterized by brief periods of high partner turnover in between longer periods with a single monogamous partner. Ultimately, they find a high quality match, and marry. A behaviorally similar model (and one which would generate an identical age-profile construction as that used above) would be that individuals prefer to have a high rate of partner turnover in their youth, until they exogenously prefer to have a single partner at heterogeneous ages. It is doubtless true that both changing preferences over

[18]In fact, the fit outperforms that of the current state of the art epidemiological model for South Africa, the ASSA AIDS model, with sum of squared deviations of about half the magnitude.

variety and the presence of a high quality partner play a role in marriage decisions. A few distinctions are worth noting: first, spousal search generates higher HIV prevalence than a changing preference model with an identical number of partners. This follows as the short periods of high turnover are a major contributor to the potential of spousal search to spread HIV. If partner turnover is relatively uniformly distributed over time, this potential is attenuated. Further, if preferences over variety decline in a smooth and monotonic manner, then the age-predictions are a worse fit than those created by a spousal search model. The spousal search model presumes that individuals are of constant risk over their ages of singlehood, and already fully explains the number of infections among the very young. Finally, as discussed below, marital shopping (unlike preferences over variety) has the implication that there should be a high variance in intertemporal choices in sexual partners, which is supported by self-reported sexual behavior data. However, it remains true that a model where individuals transition randomly between preferences for high and low partner turnover rates in a monotonic way and exogenously prefer a single partner after a particular age would be behaviorally equivalent to the one presented above, and hence have similar predictions[19]. As with any model, this one is indistinguishable in data from behaviorally equivalent, exogenously changing preferences. However, many policy predictions remain similar in either case.

Spousal Search, Sexual Behavior, and Risk

In spousal search, a large number of partners is simply an indication of bad luck in finding a good match in a given year rather than an indication of underlying preferences. Thus, if this model is relevant, we should observe relatively little serial correlation in number of partners. Moreover, most people will have isolated years where they have several partners, but most years are spent with a single partner as most time is spent with either a long-term boyfriend or girlfriend or with a spouse. Therefore, a second prediction of spousal search is that nearly everyone only has a single partner in a given year, most of the time.

As discussed above, sexual behavior data is problematic and known to be biased with strong underreporting. It is not clear whether bias changes over time, so that it is not clear

[19]Excepting the eventual marital behavior, such a model is very similar to that described in Koopman et al (1997)

whether bias could itself account for a low level of serial correlation in the number of partners. The Cape Area Panel Study (CAPS)[20] (Lam et al 2006), a random sample of 4758 young adults aged 14-22 in 2002 who live in the Cape Town Metropolitan Area, however, can provide suggestive evidence. Here I examine the self-reports of CAPS respondents who had had sex at least once by 2002 and the same respondents three years later in 2005 on the number of sexual partners from the previous 12 months. I then compare the scatter plot of these young adults to that generated by 2500 observations of simulated data. Since CAPS respondents are younger than a random selection of simulated adults from the model, I choose for each simulated observation one of the first 5 years at random to represent that simulated person's behavior in 2002 and then compare the number of partners that observation had in that year with the same observation 3 years later. I then overlay the two scatter plots for easy comparison, weighting each observation so that the sample sizes are identical.

Figure five shows the results of this exercise. Here, simulated data is represented by diamonds while actual data is represented by squares, and the size of each point is proportional to the frequency it occurs in the data[21]. One deviation from the model takes place insofar as the model does not allow individuals to transition out of sexual activity, and there are a few individuals in CAPS who have 0 partners in a year after they have had sex at least once. These observations aside, the fit is quite remarkable. In both CAPS and simulated data, the overwhelming majority of observations have one partner in 2002 and again one partner three years later in 2005. In both CAPS and simulated data, there are also substantial fractions who have more than one partner in one of the two years -- however, it is extremely infrequent to observe individuals who have many partners in both years. The overwhelming majority of individuals with several sexual partners in either one of the two years had only one partner in the other. This is striking, particularly as any models which rely on fundamental heterogeneity would suggest the opposite -- if some people simply prefer more variety, one would expect this

[20]The Cape Area Panel Study Waves 1-2-3 were collected between 2002 and 2005 by the University of Cape Town and the University of Michigan, with funding provided by the US National Institute for Child Health and Human Development and the Andrew W. Mellon Foundation. The data set is described in further detail in the appendix.

[21]There are 6 (out of 2500) simulated observations and 0 actual observations which are obscured by the legend in the upper right quadrant.

preference to be serially correlated; in practice reported behavior is not. I also impose a regression line for both simulated and actual data to describe the relationship between partners in 2002 and partners in 2005. The constant term is slightly larger in the simulated data; however, the estimated slopes are both indistinguishable from 0 and from each other. Young adults in Cape Town report sexual behavior which is strikingly similar to that suggested by marital shopping.

The model of courtship and marriage developed here would seem appropriate for the US and Europe, too, and so it would be reassuring if young adults in both contexts reported similar sexual behavior. Columns one and three of table two report tabulations of numbers of partners in the last year using U.S. adolescents from the National Survey of Family Growth (NSFG), while columns 2 and 4 report similar calculations using the CAPS teenagers (all data is described in the web appendix). Across cultures, the annual numbers of reported partners appear identical. To the extent that adolescent males are only partnering with adolescent females, we know that one of these two groups is misreporting. Strikingly, even the bias in male versus female reporting appears to be consistent between the two continents[22].

Finally, if spousal search is dangerous, then those who spend a longer time single should be at greater risk of HIV infection. That is, a longer time period spent single indicates less luck in finding a match, and should be correlated with the amount of risk borne. Data to test this is not available for South Africa; however, in the Web Appendix, I use DHS data to illustrate that length spent single is correlated with HIV status across Africa, and that it is only correlated for those who have been married recently enough to have survived whatever infections were suffered during their singlehood. That is, for people who have been married less than 10 years, a long singlehood is strongly predictive of HIV status, while for individuals who have been married for more than 10 years, it is not. This suggests that the long singlehood creates this risk,

[22]An external additional test of the model would be to input transmission dynamics of a different sexually transmitted disease in a different context and evaluate the age profile. Unfortunately, transmission probabilities of most sexually transmitted diseases are little understood and cases are often undocumented. Gonorrhea in the US, however, provides a good case study (though extremely low prevalences may cause some concern over selectivity). I complete this analysis in the appendix; once again, male and female patterns are differentiable from each other and do resemble infection rates, though the fit is less close than in the HIV case, possibly due to low and non-random prevalence.

rather than things like preferences which may be correlated with singlehood but would persist into marriage. While caution must be taken with this analysis as those who have been married longer may be an imperfect control group, this represents additional suggestive evidence that spousal search is dangerous.

POLICY DISCUSSION

This paper considers three policies to reduce the spread of HIV. The first is for individuals to use protection for the first three months of each relationship. The majority of relationships in this model are short in tenure, as most partnerships are quickly found unacceptable, which means that using condoms at the beginning of relationships may prevent transmission from most lifetime partners, in addition to eliminating the role of acute infection. I consider what happens when fraction λ of the population follows this policy. Panel A of table four observes the mean lifetime prevalence across cohorts for a variety of λ, in each case assuming $\delta = .03$, which relates to the South African epidemic[23]. For each λ, the overall prevalence is reported and then decomposed into the prevalence for those who follow this strategy and the prevalence for those who do not. The strategy of wearing condoms in short relationships is very effective for those who follow it -- in each case, inputted $\delta's$ are multiplied by 3-4.5 for the people following this strategy and 4-5.5 for those who do not. We see strong evidence of externalities here; as some individuals protect themselves, epidemic progression is limited, and everyone faces lower risks. Moreover, if everyone uses protection in short relationships, we see a tremendous fall in epidemic prevalence -- now, each new infection is responsible for about two additional infections. In a model like this, spouses will almost deterministically become infected, so that each inputted infection now causes about half of an additional couple to become infected (rather than 2-2.5 couples in the baseline case).

A second potential policy is to reduce the transmission rate of HIV, either through reducing STI prevalence (e.g. Oster 2005) or through male circumcision (e.g. Potts et al 2008). Using the marital shopping model, we can observe the effect of reducing transmission rates on prevalence, while remaining agnostic about the best mechanism to achieve such a result. A

[23]Other $\delta's$ have their multiplier reduced similarly under this policy.

review of medical studies (presented in the web appendix) suggest that transmission in the US and Europe is on the order of 50%-100% as efficient as in Africa[24], so I take the midpoint and assume that a 25% transmission rate reduction is accomplishable through biological means. The Western transmission rates rows of Table four repeat the marital shopping model, but reduce transmission risks in all stages of infection to 75% of the previous levels. The first row indicates what can be expected if transmission rates are reduced by 25% without accompanying behavioral change. In this case, the multiplier changes from between six and seven to just over four, or one extra couple being infected. Clearly, this is an effective strategy, about as effective as compelling half of individuals to use protection in their short relationships; which of these approaches is more feasible is an empirical question[25]. Table four also explores the consequences of reducing transmission rates in parallel with short-term condom use. The results here are strong. In this model, there is basically no way to reduce the multiplier below two -- individuals who enter spousal search already infected will with very high probability infect their eventual spouse. When we reduce the transmission rate to Western levels and compel individuals to use protection in their short relationships, we find that very few individuals other than spouses ever become infected. If Americans have these transmission rates and follow this strategy of protection in short relationships, it may explain why spousal search does not create an HIV epidemic in America despite otherwise similar sexual behavior. Strategies which seek to reduce

[24]African PPY transmission rates are 8-12%, which contrasts with US and European rates of 5-10%. This similarity in the face of lower condom use, more circumcision, and higher STI prevalences led Gray (2001) to reject the hypothesis that HIV-1 subtypes in Africa are more infectious. Still, these similar PPY transmission rates contrast sharply with the large differences in Oster (2005), who uses a per-partnership transmission rate (Downs and Di Vicenzi (1996) advocate the per-partnership measure as HIV infections do not appear to be binomial in contacts due to heterogeneity in infectiousness). This approach represents one approximation; it is imperfect as individuals do become infected several years into the described studies. No matter how transmission rates are estimated, a wide variety of point estimates are received, with per partnership rates exhibiting a particularly high variance. This is due to factors such as differences in lengths of the studies, differences in coital frequencies, differences in the fraction of study participants who report perfect condom usage, and small sample sizes, as well as different biological transmission rates. Because of this, the most recent studies, all African, prefer per-partnership-year or per-coital act transmission rates, which this study follows.

[25]If only a partial reduction in transmission rates is achieved, using biology to treat the epidemic is less effective. Reducing transmission rates by 12.5% (50% of what is feasible) reduces the multiplier by one (available from the author).

transmission rates through treatment of sexually transmitted diseases and male circumcision can effectively combat the epidemic, as can short intervals of protected sex, and the pursuit of both strategies in tandem may be tremendously effective.

Finally, Panel B of table four considers the implications of anti-retroviral (ARV) treatment for the epidemic. ARV treatment both extends life and reduces transmission rates, creating in principle an ambiguous effect on the epidemic and leading to mixed findings in the epidemiological literature (Cohen et al 2007). Standard medical practice is to treat individuals either just before or at the onset of AIDS; hence, in calibrating I assume that life is extended for between three to seven years in between latent and mature infection, and transmission rates are reduced to between 0 and 50% of that latent infection rate[26]. As Panel B illustrates, ARV treatment has only a limited effect on the epidemic, resulting in at most a 15% reduction in prevalence or potentially a very small increase. The reason for this is simple: by the time people are nearing the end of their HIV infection, they are in general married, and no longer accumulating many new partnerships. This suggests that using ARVs to provide a similar window of low infectiousness earlier in the infection's course may be much more effective, and similar simulations (not presented here) reveal that extending life by reducing infectiousness in the first years of infection may be associated with substantial reductions in epidemic prevalence[27]. This heightens the immediacy of current medical studies which are testing the value of treating HIV positive individuals long before the onset of AIDS (HPTN 2010). It also highlights the importance of the underlying behavioral modeling: models which do not account for the life history of sexual risk would overstate epidemiological implications of current ARV practice.

CONCLUSIONS

[26]These transmission rate numbers are justified by the review in Cohen et al (2007), which also suggests that the true transmission rate under treatment is likely closer to the 0 estimate than the 50% of latent rates; the durations of treatment are meant to provide a fairly broad range given the availability and institutional constraints in Africa.

[27]Results are not presented as the large number of assumptions renders them fairly speculative. In particular, there is little research on the natural course of infection which would result from a window of treatment early in the infection. Implications for drug resistant strains may also be severe and quite negative, but are beyond the scope of this analysis.

Serial monogamy with high turnover is sufficient to create and maintain high prevalence levels, and can blow up infection from other sources to much higher levels. Simple dating can create this behavior, if we believe that there are idiosyncratic, unpredictable components to the quality of a relationship and that individuals prefer spending more time with better matches. In contrast with many existing behavioral models which can achieve high prevalence with low transmission rates, the matching model is consistent with the observed age-death profile. Moreover, matching also supports empirical evidence on the importance of acute infection for the HIV epidemic without requiring extremely high numbers of partners.

If individuals use protection in only their short relationships, prevalence rates fall dramatically, with 50% usage being as effective as a reduction in transmission rates to Western levels. This highlights an important choice for public policy: should policy makers emphasize using condoms in new relationships at the cost of using them in older ones? Some individuals may find three months of condom use more palatable than a lifetime of protection, and a tremendous amount of risk would be averted[28]. Indeed, this message contrasts strongly with the message adopted by many public health groups, who encourage condom use throughout marriage. As many individuals doubtless hope to have unprotected sex at some point in their lives, it would be dangerous if they felt they had to make an all or nothing choice because they did not understand the relative risks of pre-marital and marital sex. Moreover, when short-term condom use is combined with reduced transmission rates, marital shopping ceases to be an effective mechanism to transmit HIV, suggesting two clear goals for policy. This also suggests that public health campaigns should target young and single men and women, with a message stressing the risks in new relationships.

An implication falling out of the medical literature is that testing campaigns will be hard to sell. Antibody-based tests are by far the cheapest and those predominantly in use both in Africa and in the United States. These tests cannot pick up acute infection, and therefore misdiagnose HIV when it is most infectious, because the body has not yet developed an immune response. Since much of the risk that a person faces with a new partner is caused by acute

[28]Adolescent girls in rural Kenya have been shown to respond to information on relative risks of different partners by changing their sexual behavior (Dupas 2007) so this message of targeted risk seems likely to be one that individuals can understand.

infection, demanding that a partner get tested before intercourse is not fully protective--moreover, if a negative result encourages choosing against condoms, then it could actually make sex more dangerous. This may pose a major challenge to testing initiatives.

Finally, I show in this paper that serially monogamous dating and marriage behavior acts as a multiplier on low prevalence rates from outside sources, and simulated evidence suggests that spousal search could cause 5/6-6/7 of all infections. Another consequence of this multiplier is that small changes in the number of these outside infections can lead to huge differences in the prevalence rate. A number of papers (e.g. Oster 2005) including this one have documented the similarity in reports of sexual behavior between Africa and the United States, and this paper has reemphasized how similar behavior with differences in transmission rates and short term condom use can explain the different prevalence levels. However, a few pivotal people may well behave differently and become infected through other methods, and both the aggregate statistics which are central to this paper as well as traditional econometric analysis are ill-suited to identifying these small samples, perhaps explaining the divergence between these results and the reports of ethnographers. Respondent-driven sampling methodologies (e.g. Heckathorn 1997) may be an asset in understanding the behavior of this pivotal minority.

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Table 1: Coital Frequencies and Birth Rates by Age

Panel A: Reported Coital Frequencies			
age	% intercourse last week	% intercourse last 2 weeks	% intercourse last month
15-19	8.09	12.52	19.13
20-24	22.58	32.69	44.63
25-29	34.12	46.49	58.26
30-34	40.59	53.4	64.36
35-39	40.29	53.01	63.57
40-44	36.57	51.47	62.31
45-49	30.85	43.69	54.97

Panel B: Birth Rates			
age	%birth in last year	adjusted % births Templeton rate	adjusted % births van Noord-Zaadstra rate
15-19	7.08	N/A	11.91
20-24	15.3	15.3	15.39
25-29	15.3	15.3	15.57
30-34	12.56	13.59	14.98
35-39	8.91	11.99	20.09
40-44	2.63	7.69	11.48
45-49	0.41	2.79	N/A

Notes

1 The Data Source is the South African DHS.

2 Adjusted % births adjust for declines in fecundity using rates from two medical studies

Table 2: Number of partners, last 12 months (sexually active adolescents only)

	(1)	(2)	(3)	(4)
Number of Partners				
0	12.37	13.48	6.83	12.39
1	48.41	43.64	67.77	70.65
2	20.00	19.04	14.15	12.13
3	9.08	10.37	6.03	3.18
4	3.77	5.45	2.33	0.95
5	2.32	2.35	1.45	0.52
6	1.35	1.18	0.48	0.09
7+	2.71	4.49	0.96	0.09
N	1035	935	1244	1162
Data Source	NSFG Males	CAPS Males	NSFG Females	CAPS Females

Notes

1 Tabulations of number of partners in the last year for sexually active 14-22 year olds in 2 surveys

2 NSFG is the National Survey of Family Growth; CAPS is the Cape Area Panel Study. Both are described in the appendix.

Table 3: Prevalence Results

		(1)	(2)	(3)	(4)	(5)	(6)
Seed Infection Rate (δ)		0.01	0.02	0.03	0.04	0.05	0.06
Acute Transmission Rate (Monthly)	20%	0.071	0.130	0.181	0.223	0.265	0.299
		(0.006)	(0.007)	(0.008)	(0.007)	(0.007)	(0.009)
	0%	0.049	0.094	0.135	0.171	0.208	0.241
		(0.004)	(0.004)	(0.006)	(0.005)	(0.005)	(0.006)

Notes

1 Presents simulated infection rates from 50 realizations of the match, each averaged over 10 realizations of the epidemic.

2 Delta is the fraction of individuals who enter the epidemic already infected

Table 4: Policy Simulations

		(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Short Term Condom Use and Transmission Rate Reductions							
Fraction adopting short term condom use		0	0.1	0.25	0.5	0.75	1
African Transmission Rates	Full Sample	0.181	0.168	0.151	0.127	0.109	0.094
		(0.008)	(0.007)	(0.006)	(0.005)	(0.004)	(0.004)
	Protected Rate		0.139	0.129	0.115	0.103	
			(0.011)	(0.007)	(0.005)	(0.004)	
	Unprotected Rate		0.172	0.159	0.140	0.125	
			(0.007)	(0.007)	(0.006)	(0.008)	
Western Transmission Rates	Full Sample	0.126	0.119	0.111	0.098	0.089	0.080
		(0.005)	(0.005)	(0.004)	(0.004)	(0.003)	(0.003)
	Protected Rate		0.102	0.098	0.091	0.085	
			(0.009)	(0.006)	(0.004)	(0.003)	
	Unprotected Rate		0.122	0.115	0.107	0.100	
			(0.005)	(0.005)	(0.005)	(0.007)	
Panel B: ARV treatment							
		0.169	0.186	0.159	0.186	0.150	0.185
		(0.008)	(0.008)	(0.007)	(0.008)	(0.006)	(0.008)
Survival length		3 years		5 years		7 years	
ARV transmission rate		0%	50%	0%	50%	0%	50%

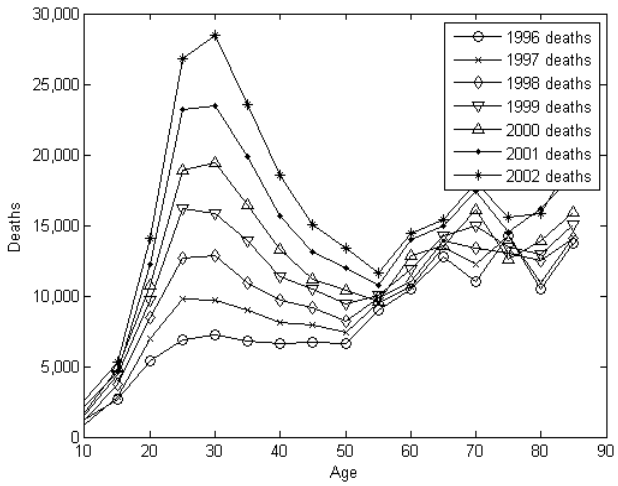
Notes

1 Presents simulated infection rates from 50 realizations of the match process, each averaged over 10 realizations of the epidemic.

2 δ, the fraction of individuals who enter spousal search infected, =0.03

Figure 1: Deaths by Five-Year Age Group, 1996-2002

(a) Women's Deaths



(b) Men's Deaths

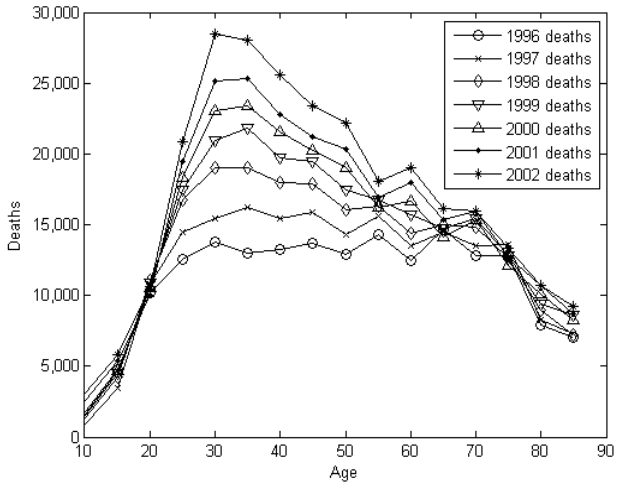


Figure 2: Best Fits of Age-Independent Risk Models

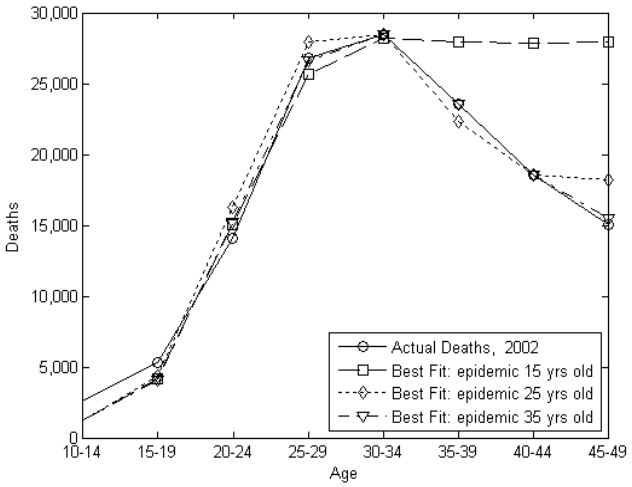
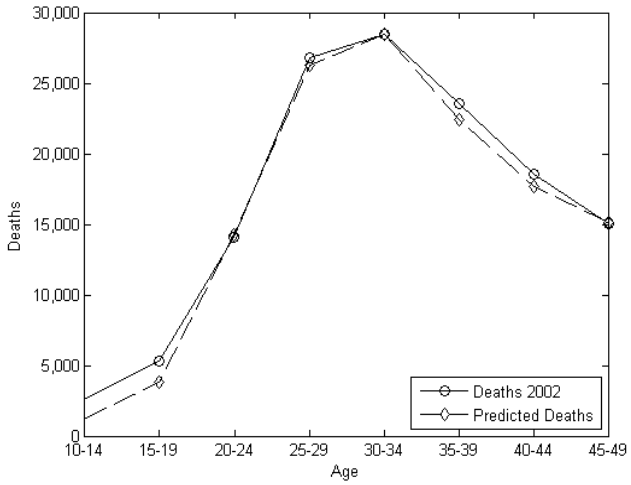


Figure 3: Predicted and Actual Deaths, 2002

(a) Women's Deaths



(b) Men's Deaths

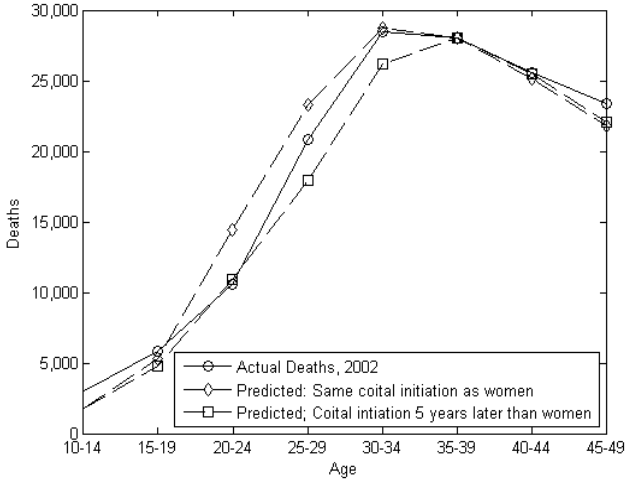
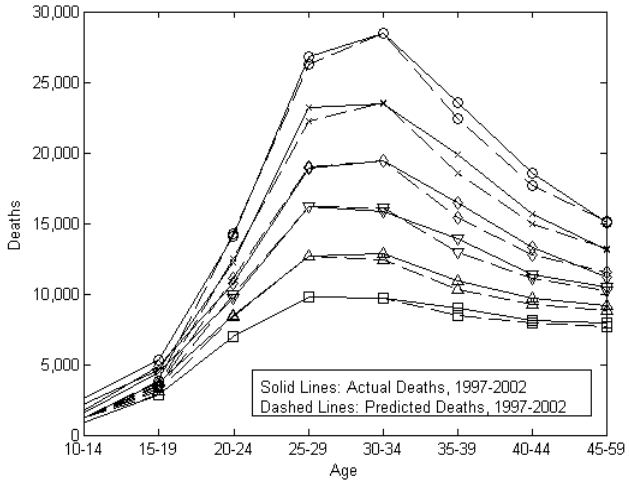


Figure 4: Predicted and Actual Deaths, 1997-2002

(a) Women's Deaths



(b) Men's Deaths

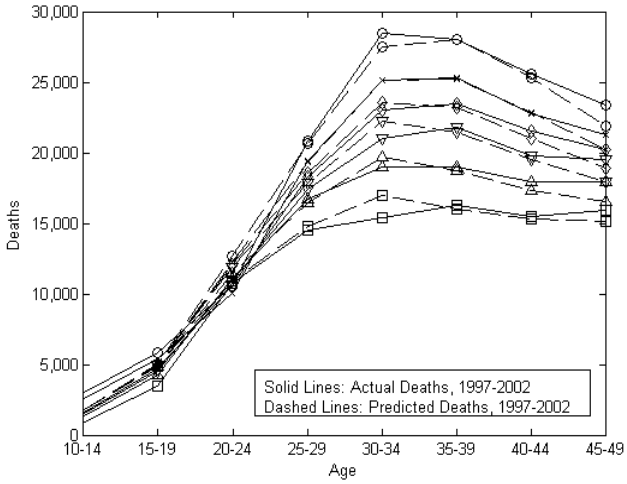


Figure 5: Simulated and Actual Annual Partners in Cape Town