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**THE PRIVATE DEMAND FOR INFORMATION
AND THE EFFECTS OF PUBLIC TESTING PROGRAMS:
THE CASE OF HIV**

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**THE PRIVATE DEMAND FOR INFORMATION AND THE
EFFECTS OF PUBLIC TESTING PROGRAMS: THE CASE OF
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by

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and

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ABSTRACT

This paper provides a theoretical and empirical analysis of the impact of private HIV information on sexual activity showing how the private information provided by a public testing program would change the allocation of information generated in absence of the program. The theoretical analysis implies that mainly low-risk HIV-positive and high-risk HIV-negative individuals, which are small groups by definition, will respond to a public testing program, and that the total response to such a program may be small due to the offsetting effects that are masked by aggregating across such risk groups. The result follows from the key insight of our model, that the information 'treatment' varies with the prior probability of infection held by the respondent, thus necessitating disaggregation by categorical characterizations of these unobservable prior probabilities in order to interpret the evidence on these information interventions. We address these implications empirically using a longitudinal survey that imitated a public HIV testing program by actually administering an HIV test as part of the survey as well as recording the respondent's prior knowledge of HIV status, and his or her sexual practices before and after this information intervention. Using this direct evidence on the trade effects of endowing traders with private information, we are able to directly assess the longitudinal impact of such a

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public information intervention. Consistent with the theoretical discussion, we find that inducing private information *increased* the volume of sexual contact by 16 percent for high-risk HIV-negative respondents and had little effect on high-risk HIV-positives. We conclude by discussing the general implications our analysis has for empirical work that attempts to *directly* sample information and information changes in assessing theoretical models of informational effects on market equilibria.

1 Introduction

Information, and in particular the distribution of information amongst agents in a given market, plays a vital role in many models of economic activity. For example, many studies of credit markets argue that information asymmetries by borrowers and lenders reduce the volume of credit exchanged. Similarly, more than half the federal budget, in terms of Medicare health insurance and Social Security savings, is frequently argued to be due to the trade barriers that information asymmetries impose. Yet, it is difficult to *directly* observe or measure the extent and consequences of information asymmetries in decentralized markets. Instead, in empirical assessments of models of asymmetric information we often *infer* the distribution of information in a market from the nature of prices and/or quantities traded, or by construction of proxies for information by subgroups of traders without the information itself actually being observed.² The pattern of information in the market is then recovered from a model of the information distribution given prices and/or quantities or the outcomes of interest.³ In other empirical studies of economic models of information, proxies

²In the asymmetric information auctions literature concerning oil exploration drainage leases (e.g. Hendricks and Porter (1986)) rather tight arguments can be made that firms with tracts adjacent to the tract up for auction have access to seismological data that the "outside" firms do not have. However, the *degree* to which an inside firm's information set is a finer partition than the outside firm's is not measurable. Nor does the pattern of information change in known or observable ways over these sample periods.

³For example, in the literature on asymmetric information models of strikes, (e.g. Kennan and Wilson (1988) and Card (1990)) the joint distribution of the endogeneous outcomes of

for information (usually subgroupings of the market traders which are assumed to have better or worse access to the information under question) are used, but again, the information itself is not actually observed.⁴ Our objective here is to consider the demand side of a market for *information* regarding individuals' HIV statuses, and how changes in that information relate to changes in sexual behavior. We want to make clear that we are *not* considering a market for sexual activity. The unique feature of our study of the impact of information in an economic model is that for a portion of the participants in our data, their information (regarding their HIV status) is changing in a known and quantifiable way. We are thus in a position to evaluate clearly a behavioral response to a change in an individual's information set that has changed during the course of the survey (in this case, the outcome we study is the change over the survey in the number of sexual partners before and after the individuals' information sets have changed).

The central policy issue we study, the role of informational interventions such as public testing programs in altering sexual practices of a population so as to help curtail the spread of HIV, clearly necessitates the development of a model of the behavioral response to a change in information. HIV, the causative agent of wage settlements, strike durations and incidence are studied to infer something about the degree of asymmetric information of the profitability of the firm.

⁴In the asymmetric information strikes literature, Tracy (1986,1987) uses the *observed ex-post* stock market valuation of the firm to proxy for the union's *ex-ante* uncertainty about the firm's profitability.

AIDS, is an asymptomatic disease such that those infected cannot be discerned from those not infected. Nor is infection evident to an individual unless he or she has a blood test to determine his or her infection status. Furthermore, HIV is not a disease that is spread through the air or transmitted by chance (at least in the bulk of transmissions) such as measles, but is spread by actions largely dictated by choice.⁵ Indeed, many of the public health proposals aimed at trying to curb the spread of HIV are aimed at changing transmissive behavior by altering the nature of information about the disease: through education about the prevalence of HIV and the means by which it is transmitted, and by public testing programs aimed at providing information on HIV status to individuals (and in more radical proposals, to their partners) so that they may take actions to limit exposing others to the disease. But standard epidemiological models of disease transmission, since they do not provide a model of the behavioral response to a change in information, provide no means for evaluating the efficacy of these proposals. This is because the allocation of information regarding disease status does not matter for the progression of diseases such as HIV in so far as these models are concerned.⁶ This paper provides a theoretical and empirical analysis of the impact of private HIV information on sexual activity. We do so by first considering the nature of the *demand* for information about HIV, so as to understand who would be affected by a public testing program.

⁵See the general discussion in Bloom and Carliner (1988), Bloom and Glied (1991), and Philipson and Posner (1993) among others.

⁶See Anderson and May (1991) and the references therein.

We find that the *aggregate* response to the information change is close to zero, suggesting little if any behavioral elasticity with respect to a change in an individual's information set. However, the model we construct for an individual level demand for information predicts exactly this result, but it is perfectly consistent with a non-zero *individual level* elasticity with respect to an information change. The aggregate net effect of zero comes from aggregating across sub-populations with different prior beliefs, and thus differing responses to the information learned through the survey. When we disaggregate our results by discrete characterizations of the prior beliefs, such as High and Low risk individuals, we find that those individuals who 'learn a lot' from the survey, i.e. those individuals whose posterior beliefs of infection change a lot upon having their HIV status revealed to them relative to their prior beliefs of infection, do in fact respond in a significant way to that information change. A somewhat surprising result of this is that not all individuals who learn they are HIV positive change their behavior (in this case, their number of sexual partners) in a significant way. Instead only those individuals who thought they were HIV negative, but who learned instead they were HIV positive, appeared to change their number of sexual partners significantly (although this particular group was so small that a definitive statistical claim cannot be made). Another somewhat surprising finding, although completely consistent with the model we propose, is that those individuals who thought they were HIV positive, but who learned through the survey they were HIV negative, *increase* their number of sexual partners as a

result of this information. Somewhat perversely, in the face of a growing HIV epidemic, such an effect of increased sexual contact could potentially enhance the spread of HIV.⁷ In any event, the changes in behavior as a result of this sort of 'information intervention', which emulates a publicly subsidized HIV testing program, indicate that the benefits to a public testing program would be few if any.

The paper may be outlined as follows. Section 2 studies the determinants of the private demand for HIV-testing and the incremental effect that a public testing program would have on that demand. We show that the private demand is a non-monotonic function of the prior belief of infection, corresponding to the fraction of a group that is infected, and therefore only those people who are most unsure demand the information generated by a test. This has the implication that a public testing program affects mostly the extreme risk groups, consisting of low- and high-risk traders. A consequence of this is that only high-risk HIV-negative traders and low-risk HIV-positive traders gain any substantial amount of information from the public testing program, in the sense that the posterior belief after testing is altered compared to the prior belief before testing. If

⁷We should emphasize however, the goal in our paper is *not* to analyze the effects of a public testing program on the *spread* of HIV. As Michael Kremer has pointed out to us, disease dynamics can be driven by the behavior of only a very few infected individuals having a lot of sexual contact. To the extent this occurs in our data, if our intent were to study disease dynamics, we would want to look at effects other than just mean treatment effects of learning, but consider effects at different quantiles.

behavior is information elastic, the greatest response to the public information intervention is therefore in these groups, which are small by definition.

Section 3 provides an empirical examination of these implications. We first describe the dataset, the San Francisco Home Health Study (SFHHS), which is unique relative to many economic datasets used to empirically study economic models of information because it includes *direct* measures on private information (in the crude sense that we know if the respondent does or does not know his or her HIV status before the start of the survey) and behavior. Using these data, we estimate that the effect of a public program endowing individuals with private HIV information is small, in terms of the effect on the *quantity* of sexual trades (number of partners). The *survey itself* mimics a subsidized public testing program by performing blood tests on the respondents through the survey, in addition to asking about private knowledge of HIV infection status and sexual behavior, both before and after this information intervention. The idea is that the *availability* of HIV testing conducted by the survey was devised independently of the private demand for HIV testing (in that the survey frame was designed independently of the demand for HIV tests). Using this survey we are able to test in a *direct* manner (by observing an intervention of an information 'dosage' through the survey) the implications discussed in the theoretical analysis, as well as the implicit trade effects of endowment of private (and so potentially asymmetric) information that such a program involves.

Consistent with the theoretical model outlined above, we find that nega-

tive high-risk individuals are the people most responsive to the public testing mimicked by the survey (only a negligible proportion of the sample falls in the low-risk group that learn through the survey they are HIV positive, as a result we cannot conclude anything with regard to the behavioral response of this group).

Since we lack a randomized design of the group who is affected by the testing through the survey, we rely on a non-random treatment and control methodology to estimate the effects of the 'information intervention' presented by the HIV testing conducted through the survey. Since the lack of pure randomization suggests possibilities of selection bias, we present results based on *two* different control groups. Both control groups have the property that neither of them should be affected by the 'treatment' of the HIV test administered through the survey. The first control group consists of the individuals who have prior knowledge of their HIV status *before* the first wave of the survey, but who choose to test through the survey as well. Given that they have prior knowledge of their HIV status, they would not be affected by the treatment administered through the survey. However, as this group is not selected at random, heterogeneity arguments could be made that this 'safe sex' group (given their desire for double testing) would have too strong of a trend towards safer sexual practices, and thus drive some of our results for that reason. Thus, we also make use of a second control group, which would be an ideal counterfactual group were it randomly selected (i.e. if the testing component of the survey were denied to

a randomly chosen portion of the respondents). This control group consists of those respondents who completed the questionnaire portion of the survey, but refused to take the blood test administered as part of the Wave I survey. Thus, for this group we have the demographic information (including their sexual orientation), but do not know their HIV status. While it is certainly possible that both control groups will yield results biased in the same way due to their non-random selection, we find it to be strongly suggestive of the robustness of our results in that the results based on both control groups are qualitatively and quantitatively similar (although not as statistically significant in the latter case).

Because there has been increased protection over time with the rise of the AIDS epidemic,⁸ and because the information intervention is not the *only* factor leading to a change in sexual contacts over the time span of our survey, we use those respondents who tested before the survey as a control group for those who learned their HIV status through the survey. In addition, since this control group is not derived through any randomization device, we construct a second control group of those respondents who filled out the questionnaire portion of the survey, but who refused the HIV test component. Thus, although high-risk respondents who learn through the survey that they are HIV-positive dropped approximately 0.9 of a partner after the information intervention (compared with before the intervention), those high-risk respondents who had prior knowledge

⁸See Ahituv, Hotz, and Philipson (1993).

of being HIV positive dropped roughly half this much (0.45 of a partner), with the statistical difference between these two point estimates being negligible. This suggests no behavioral effect of learning that one is HIV-positive in the high-risk group—a finding consistent with the theoretical model described above. Also as predicted, those high-risk individuals who learned they were HIV-negative contrary to their prior beliefs about their infection status experienced a one-tenth of a partner, relative to a significantly negative trend (-0.7 of a partner) in the change in the number of partners for those who tested negative before the survey.⁹

These findings have potentially important implications for the effects of public testing programs on the growth of the HIV epidemic. If high-risk groups are the target of testing programs, then such programs appear to do little in the way of altering their behavior. Instead, the largest response to information we observe in our data comes from those who actually learn something—the positive low-risk and negative high-risk individuals. Indeed, the high-risk negatives subsequently engage in sexual intercourse with a relatively greater number of partners, potentially placing them at greater risk of becoming infected. The model suggests, and the empirical results corroborate, that a public testing program can potentially have unintended effects. The main substantive conclusion from these close-to-ideal data, therefore, is that public HIV testing programs

⁹However, the number of negative high-risk individuals and positive low-risk individuals is small by definition, so the standard errors are relatively larger.

appear to have little benefit, if any, in reducing the risk of further infections for populations similar to this one from San Francisco.

We conclude this paper with a discussion of the shortcomings of our data, and the methodological issues that are generalizable to other studies of demand for information, information asymmetries, and their resultant impact on quantities of goods traded in an implicit or explicit market. Much of the previous empirical work on models of information and their implications for trade effects has been done in the context of the market inefficiencies arising from the presence of asymmetric information between traders (see e.g. Bond (1982), Genesove (1993), and Foster and Rosenszweig (1994)). The key difference of our paper, beyond its substantive focus on AIDS, is that our data include *direct* measures of how individuals' information sets are changing for our portion of our data. Given the large theoretical literature on models of information and its sizable impact on economic thought (notably the implications of trade reductions and market inefficiencies that arise from models of asymmetric information),¹⁰ we are hopeful that future research efforts in this area will attempt *direct* sampling of the information at the heart of these models, as opposed to inferring its existence by looking at patterns of prices and quantities traded. In the conclusion, we discuss the advantages of having *direct* measures on information, and the degree to which some of the lessons we have learned in this particular study on the demand for information can be carried over to markets where the measure

¹⁰See eg. Akerlof (1970) and the literature that followed it.

of information changes cannot be as precise as an HIV test. We consider as well situations where the interest is on studies of *asymmetric* information, and so the data and survey requirements are greater than in our study. We point out some potential pitfalls that direct survey methods may have on the study of such settings. We also acknowledge that while we do have *direct* measures as to how the information sets of the sample respondents is changing longitudinally through the survey, that their response to that 'information treatment' depends upon their unobservable prior probability of infection. Future studies may wish to try to directly sample these priors or at the very least recognize their impact on the effects of information treatments.

2 Private vs. Public Demand for Information

This section discusses a model of the private demand for testing and the incremental belief and trade effects of a public testing program. We formulate the model solely as an *individual* demand for testing, and set aside for now the issue of behavior as arising from a joint bargaining problem for a couple.¹¹ Consider

¹¹We have chosen this route based on the data available to us. Earlier work with a modelling approach that incorporated a setting of imperfect information as to the partner's beliefs of the respondent's HIV status required data on how those partner beliefs changed with respect a change in the respondent's knowledge of his or her own HIV status. Since we lacked those data, the only feature of this richer model we could address with our data was whether the findings were consistent with a pooling or separating equilibrium of the model. We intend the model presented here to be thought of as representing the separating equilibrium of this

an individual who has a prior belief of infection that may be altered through the information provided by a test. Denote the health states by $H \equiv \{0, 1\}$, where 0 indicates not infected and 1 indicates infected. Let the set $Y \equiv \{y_0, y_1\}$ denote the set of feasible sexual behaviors, where the subscripts denote the most-preferred behavior when the infection statuses are known with certainty, so that in the case of HIV, y_0 may denote no sexual contact or partners, and y_1 may indicate engaging in sexual contact with a partner. Denote the feasible set of test results by $T \equiv \{0, 1\}$ for negative and positive tests. Let the health-dependent utility function be denoted $U(y, h)$, indicating the utility of the individual under health state $h \in H$ behaving according to $y \in Y$, where, *ceteris paribus*, better health and unprotected behavior are preferred (i.e., $U(y, 0) > U(y, 1)$ and $U(y_0, h) > U(y_1, h)$). If $P(H = h, T = t)$ denotes the probability of the individual being of health status h and receiving the test result t , then the prior belief of infection is denoted $p \equiv P(H = 1)$, and the belief conditional on a positive and negative test is denoted $p_1 \equiv P(H = 1|T = 1)$ and $p_0 \equiv P(H = 1|T = 0)$ respectively. Given any prior belief of infection p the expected utility of a given behavior is

$$V[y, p] \equiv pU(y, 1) + (1 - p)U(y, 0).$$

If $y(p)$ denotes the most preferred behavior under infection belief p , it is easily shown to involve protection if and only if the risk is higher than a given threshold richer model, or where there is complete altruism between partners, so no bargaining problem is present.

level

$$y(p) = y_1 \Leftrightarrow p \geq \bar{p},$$

where $\bar{p}$ is the cutoff-belief above which an individual will not engage in transmissive behavior defined by $V[y_1, \bar{p}] = V[y_0, \bar{p}]$. In the special case of perfect knowledge of infection status, the decision rule reduces to choosing transmissive behavior when negative and avoidance behavior when positive, that is, y_0 when $p = 0$ and y_1 when $p = 1$. An individual's expected utility without a test is given by

$$V_N \equiv V[y(p), p] = pU(y(p), 1) + (1 - p)U(y(p), 0).$$

Her expected utility after testing positive is

$$V[y(p_1), p_1] = p_1U(y(p_1), 1) + (1 - p_1)U(y(p_1), 0),$$

and after testing negative

$$V[y(p_0), p_0] = p_0U(y(p_0), 1) + (1 - p_0)U(y(p_0), 0).$$

Thus, at the time of testing, taking into account the results of testing positive or negative, the expected utility of testing is

$$V_T \equiv P(T = 1)V[y(p_1), p_1] + P(T = 0)V[y(p_0), p_0].$$

The value of information comes from the value to the individual of behavioral change upon the result of the test. It is straightforward to show that the net benefit of testing satisfies

$$\begin{cases} V_T - V_N = p[U(y_1, 1) - U(y_0, 1)] & p < \bar{p} \\ V_T - V_N = (1 - p)[U(y_0, 0) - U(y_1, 0)] & p \geq \bar{p}. \end{cases}$$

This may be explained as follows. The individual chooses transmissive behavior when negative and avoidance behavior when positive. If he does not test, he engages in transmissive behavior if he believes he is likely to be negative, $p < \bar{p}$. If he tests, he engages in transmissive behavior only if he tests negative. Therefore, the chance that he is doing the wrong thing when not testing is the probability of being positive, p , and the benefit of switching to avoidance behavior upon testing positive is $U(y_1, 1) - U(y_0, 1)$. Similarly, the chance of doing the wrong thing when believing himself to be at high-risk is the chance of being negative, $(1 - p)$, and the benefit of switching behavior upon a negative test result equals $U(y_0, 0) - U(y_1, 0)$. In summary, the benefit to the individual of screening comes from the expected benefit to the individual of behavioral change.

The private sector demand for screening comes at a cost denoted C , including, in addition to the direct price of testing, the time devoted to seeing the doctor (shoe-leather cost) or aversion toward the test itself (psychic costs). Screening is demanded if the value of information offsets this cost,

$$V_T - V_N \geq C.$$

Figure 1 illustrates the benefit and costs of screening and how the individual's

prior belief about infection determines the demand for testing.¹²

[FIGURE 1 INSERTED HERE]

The benefit of screening is peak-shaped and the cost of screening is flat with respect to the individual's perceived probability of infection. This has the implication that the prior has a non-monotonic effect on the demand for a screening. Those individuals with subjective probabilities near the middle of this diagram (i.e., where the peak of the benefit schedule exceeds the cost) demand a private screening. Hence, it is individuals in the middle who get tested privately because those are the ones most unsure about their infection status, and thus they are the most unsure about the correctness of their behavior without screening. People who are more sure of their infection status are more sure that they are doing the right thing, y_1 if $H = 1$ and y_0 if $H = 0$, and hence have a lower demand for testing. In the special case of being sure about one's infection status before the test, there is obviously zero demand for testing.

¹²Gertler, Sturm, and Davidson (1995) consider a model of the demand for supplemental Medicare insurance in which the benefit schedule is similar to ours. They construct a structural model in which information shifts both the mean and the variance of the expected benefits distribution. Their information index is a constructed proxy measuring survey response knowledge of Medicare benefits, before and after an 'information intervention' consisting of educational workshops provided to respondents on their Medicare benefits.

2.1 The Effects of Public Screening

In Figure 1, the demand for private screening comes from individuals in the range between the lower and upper bounds $[L, U]$, which represents those individuals who are more unsure of their infection status. The lower and upper bounds can be shown to satisfy

$$L = C/[U(y_1, 1) - U(y_0, 1)] \quad \& \quad U = 1 - C/[U(y_0, 0) - U(y_1, 0)].$$

The effects of a *public* screening program that subsidizes testing by an amount s may therefore be represented by the interval $[L_s, U_s] \supseteq [L, U]$ corresponding to the beliefs of testers under the reduced testing costs $C - s$ of the program.

We label this (lower) cost of subsidized testing as C_s , and it is shown in Figure

1. As the cost of screening is made smaller by such a program, the set of people affected by a public screening program is comprised more and more of those with relatively more extreme priors, close to 0 or very close to 1. In the extreme case of mandatory testing or a full subsidy, $s = C$, everyone tests, so that $[L_s, U_s] = [0, 1]$. To consider the effect that a public screening has on behavior, suppose the population is specified by a distribution function $F(p)$ over prior probabilities of infection. We assume that an individual's subjective belief of infection corresponds to the objective frequency (i.e. prevalence),¹³ in his risk group, so that the value $F(p)$ may also be interpreted as the fraction of

¹³By assuming that p percent of individuals who have subjective beliefs p are indeed infected, we ignore the important effects of mandatory screening which occur under misperceptions of risk group membership.

individuals in risk groups with prevalence lower than p .

The behavioral effect Δ of the program is then given by the prior specific effects $\Delta(p)$ aggregated up, as in

$$\Delta = \int \Delta(p) dF(p) =$$

$$\int_{[L_s, L] \cup [U, U_s]} [y(p) - y_1]p + [y(p) - y_0](1 - p) dF(p) =$$

$$\int_{[L_s, L]} [y(p) - y_1]p dF(p) + \int_{[U, U_s]} [y(p) - y_0](1 - p) dF(p).$$

This aggregate effect may be interpreted as follows. Since a public screening program only impacts those individuals who do not test privately, the program has an effect only on those individuals who do not test *and* who are doing the wrong thing (i.e., individuals engaging in transmissive behavior when positive and avoidance behavior when negative). Thus, the program only affects negative high-risk individuals (in $[U, U_s]$) and positive low-risk individuals (in $[L_s, L]$). The first term in the last equation therefore represents the behavioral change of positive individuals who do not demand a test privately. These individuals' low belief of infection induces them to engage in transmissive behavior, and the test induces p percent of each risk class to alter their behavior. The second term represents the behavioral change of negative individuals who do not demand a test privately. These individuals' high belief of infection induces them to engage in avoidance behavior, and the test induces $1 - p$ percent of each risk class to

alter their behavior. The behavioral change thus comes from individuals who are not 'doing the right thing,' consisting of low-risk positives who do not protect and high-risk negatives who do protect.

We illustrate in Figure 2 the effects of testing for the continuum of the prior beliefs of infection. This figure is drawn under the assumption that the change in behavior is binary (eg. adopt protective behavior if one learns he positive, and adopt transmissive behavior if one learns he is negative). In our context, where we focus on the particular behavioral variable of the number of sexual partners, we may think of this binary behavioral change as adding or dropping a partner. This figure, as is the case with Figure 1, is drawn under the assumption of symmetry. If individuals are distributed with beliefs uniformly on $[0, 1]$, then it is visually clear from the diagram that the aggregate effect will be exactly 0, even though individuals are information elastic. Under the more realistic assumption that individuals are distributed with disproportionate mass near $p = 0$, and allowing for non-binary responses, the aggregate response may not be exactly 0, but the diagram does indicate how aggregate effects near 0 are consistent with underlying behavioral responses that are information elastic.¹⁴

¹⁴In order to make the exposition of the model clear, we have focused on the case of symmetry, (i.e. the benefit schedule peaks at 0.5, the change in behavior when an individual learns she is positive is of the same magnitude (but opposite sign) as when she learns she is negative, etc.), although the fundamental points of the model (individual-level behavior which is information elastic consistent with small aggregate effects of testing, opposite signs of the change in behavior for low-risk positives and high-risk negatives, essentially 0 changes for the other groups, etc.) remain unchanged.

[FIGURE 2 INSERTED HERE]

For a partially subsidized public testing program (i.e. the subsidy level s lying in the interval $(0, C)$), the figure depicts the increase or decrease in protection induced by the program as a function of the risk level. Note for those individuals with prior beliefs p lying in the intervals $[0, L_s]$, $[L, U]$ and $[U_s, 1]$ there is no effect on behavior of the public testing program. This is because for the group of people with p in $[L, U]$ there is a *private* demand for the test before the start of the survey. Whereas for those people with p in either $[0, L_s]$ or $[U_s, 1]$ there is no demand for a test even with the lower, subsidized cost of testing under the survey. We make use of these 'non-affected' groups to construct appropriate control groups which we discuss in Section 3 below. Thus only for those people with p in either $[L_s, L]$ ('low-risk' individuals) or $[U, U_s]$ ('high-risk' individuals) is there a change in behavior induced by the program. Among the low-risk individuals there is an increase in protection made up of a fraction p of positives whose protection rises by one unit, $y(p) - y_1 = 1$, so that the risk-specific effect is $\Delta(p) = (p)1 + (1 - p)0 = p$. Among the high-risk individuals there is a decrease in protection made up of a fraction $1 - p$ of negatives whose protection falls by one unit, $y(p) - y_0 = -1$, so that the risk-specific effect is $\Delta(p) = (p)0 + (1 - p)(-1) = p - 1$. The size of the trapezoid

Based on our work below, we would expect the peak of the benefit schedule to lie near 0.04 (the mean HIV prevalence in our data), and the drop in the number of partners from learning positive to be somewhat larger in magnitude terms (in terms of just point estimates) than the increase in the number of partners from learning negative.

above the abscissa minus the size of that below it makes up the overall effect Δ . The trapezoid to the left is the aggregate increase in protection by low-risk positives, and the trapezoid to the right is the aggregate reduction of protection by high-risk negatives.

Figure 2 illustrates several points of interest. The sign of the effect in protection is different, dependent on whether a non-demander is high- or low-risk. Consequently, the *unconditional* effect Δ , aggregating up over *all* risk classes, may be close to zero if the sizes of the two trapezoids in the figure are approximately equal.¹⁵ Furthermore, the change in behavior is close to zero for high-risk positives and low-risk negatives since they gain little information from getting a test result which confirms their prior beliefs. The individuals who *learn* something from the public program, a prerequisite for any behavioral effect, are those whose test results do not coincide with their priors: high-risk negatives and low-risk positives.

3 Empirical Analysis

This section uses the theoretical model presented in Section 2 above to aid in the construction of appropriate control groups by which we can estimate the behavioral effects of a public testing program using a longitudinal dataset on sexual behavior. The dataset's key feature is that it samples *directly* whether

¹⁵For example, if beliefs are uniform and losses from misbehaving are symmetric, then the aggregate change in behavior Δ is exactly zero.

or not the respondents have tested previously for HIV. Thus, for a portion of the sample (those who have not tested previously, but who choose to be tested through the survey), their information sets are changing in a known and observable fashion. This feature stands in contrast to previous empirical studies of the trade effects of incomplete information, in that we have *direct* data on a change in information.

3.1 The Dataset

The dataset we used comes from the San Francisco Home Health Study (SFHHS), collected by the AIDS Prevention Center at the University of California at San Francisco (UCSF).¹⁶ The SFHHS is an epidemiological study designed to yield data on the prevalence of AIDS and related risk factors in multi-cultural neighborhoods. The respondents of the baseline survey were interviewed to obtain information about their behavior, attitudes, and beliefs relevant to AIDS. The sampling frame was defined to be unmarried males and females, ages 20-44, who were also residents in San Francisco census tracts with substantial proportions of blacks and Hispanics (those geographically adjacent to the Castro District).

The survey sample was a stratified two-stage sample of all households within the

¹⁶For details of the sample design, see 'Sampling Methods and Wave 1 Field Results of the San Francisco Home Health Study,' Survey Research Center Technical Report, University of California, Berkeley (the initial project site of the SFHHS). See also the paper by Fullilove et al (1992) which compares the sampled population to the population covered by the sampling frame, using Census tract data.

designated census tracts. All eligible persons in each selected housing unit were taken into the sample. Interviewing for the baseline survey began in April 1988 and finished in July 1989, and interviewing for the second wave was initiated one year later. For details of the sample, particularly with regard to the target population and the specific census tracts within the frame, see Fullilove et al. (1992).

The summary statistics for the types of respondents in our constructed sample are given in Table 1 below. Since our sample is representative of a region of higher risk than the US as a whole, we offer the reader a comparison of some of the variables in our data with those for the entire US. To that end, we report where possible the means for comparable variables from the National Health and Social Life Survey (NHSLS) conducted by National Opinion Research Center (NORC) at the University of Chicago (see Laumann et al. (1994)), which is intended to be a nationally representative sample of sexual attitudes and practices.

TABLE 1 INSERTED HERE

The SFHHS obtained very detailed information from these respondents from the standpoint of evaluating the trade effects of incomplete information. In particular, the measures were far more detailed than those of previous empirical studies of incomplete information, most importantly in that they include *direct* measures on the respondents' beliefs. The observables of interest in the SFHHS include the sexual (disease transmissive) activity of the respondent with the

respondent's partner(s), the respondent's knowledge of the HIV status of his or her own self and partner(s), and the *actual* HIV status of the respondent as measured by blood samples taken as part of the survey.¹⁷ The survey slightly undersampled males relative to the US population estimates, and 14 percent of the sample self-reported their sexual orientation as homosexual, defined here as those respondents with a partner of the same gender, indicating a large degree of over-representation of homosexuals in our sampled population relative to the US population. However, we present results largely broken down along this line of stratification, so this over-sampling is not of concern in our context. Indeed,

¹⁷The survey questions relating to the respondent's knowledge of his or her own HIV status are the following two questions: 1. Have you ever had your blood tested for infection with the AIDS virus? 2. (conditional on answering 'yes' to Question 1) Do you know what the result(s) of your test(s) (was/were), or didn't you find out your result(s)? Interestingly, while the survey asked whether the respondent knew his or her HIV status, they did not ask what that status was *through the survey instrument*. Thus, there exists the possibility of contamination of this control group (although we present results below based on a second control group for whom this source of bias should not be present). In particular, some of those who *knew at some point in the past* they were HIV negative, but who tested positive through the survey (and this is the first time they learn this), we would classify in the group Know HIV positive, when in fact they thought they were HIV Negative. Based on the observed prevalence of HIV in the population, however, we estimate using our data the number of such individuals is quite small in our control group. This 'contamination' of this particular control group would tend to bias *down* the estimate of the change in the number of partners for those who we classify as Know HIV positive, thus biasing *up* (toward zero) our difference-in-difference estimate of learn HIV positive. Again, however, we point out that the likely magnitude of this bias is quite small (certainly relative to the sampling error of these estimates).

it is highly desirable, since homosexual respondents are such a small fraction of the population. A total of 44 percent of the respondents were white, 25 percent black, and 25 percent Hispanic in our sample, indicating an over-representation of minorities. This could be a concern in our context, if race/ethnicity affects sexual practices, apart from affecting prior probabilities of infection status.

The behavioral outcome (Y) with which we are concerned is the total number of partners with whom the respondent has been sexually active with in the previous 12 months. Approximately 78 percent of the respondents had one or more sexual partners, while 38 percent had two or more partners. The health status variable (H) of concern is the HIV status of the respondents. The overall HIV prevalence among respondents was 4 percent, with prevalence being 30 percent among homosexual respondents, and less than 0.8 percent among heterosexual respondents. Finally, the combined response rate for screening and interviewing of SFHHS respondents was 61.8 percent, which is lower than the response rate for the NHSLS, but relatively high to comparable surveys involving information as confidential as HIV status and sexual behavior.

A central aspect of the SFHHS is that the survey administered to the respondents an HIV blood test, so a respondent learned information about his or her own HIV status just by participating in the survey.¹⁸ The interviews were

¹⁸Of the 1770 members of the Wave I sample, 254 participated in the questionnaire portion of the survey, but refused to participate in the blood test portion of the survey. 1369 of the 1770 respondents participated in *both* the questionnaire and blood test portions of the survey. Of these 1369, 833 learn for the first time their HIV status as a result of the survey. Of the

conducted face-to-face in the home of the respondent, and the HIV testing took place directly after the interview as a blood test administered by the interviewer. Therefore, there was virtually no lag time between administration of the questionnaire and the HIV test. This is important for interpreting the retrospective responses of behavior of the untested individuals in wave 1, since we want to capture their sexual practices at exactly the same time that we record their HIV result. Also, it is important that the survey was conducted in the respondent's household, *without* the initiation of the respondent, since this lowers both the 'shoe-leather' and psychic costs of HIV testing for the respondent.

Notice that if compliance with the blood testing component of the survey were perfect, then we could interpret the results as the effect of a *mandatory* public testing program, because the sampling frame was devised independently of the (private) demand for blood testing. This 'program effect', however, would not coincide with the causal effect of the 'information intervention' itself, since the pre-existing demand for information in the sampled population was not randomly assigned conditional on the covariates. We content ourselves with measuring the program effects of the information intervention rather than trying to draw causal inferences from the effect of changing respondents' information sets. Since participants *may* refuse the blood testing component of the survey, we interpret this intervention as a *subsidy* to the cost of testing (so the cost lowers to the line denoted by C_s in Figure 2), since the surveyors contacted

833 who learn, 814 learn they are HIV Negative, and 19 learn they are HIV positive.

the respondents and went to their homes independent of their explicit demand for a blood test. Thus, our results are perhaps more appropriately construed as an analysis of a public subsidy program for HIV testing that operates in conjunction with a private demand for testing, effectively reducing the costs associated with a private demand for testing.

We construct two types of control groups which we use to 'bound' the effect of the information intervention using the model presented in Section 2 above. The first control group consists of those individuals who were tested for HIV prior to the inception of the survey, and so would not be impacted by their 're-testing' through the survey. In looking at Figure 3, we see that this group is comprised of those individuals for whom the benefit exceeded the *private* cost of testing (thus leading them to test prior to the survey), and so have prior probabilities of infection lying between L and U in Figure 3.¹⁹

[FIGURE 3 INSERTED HERE]

The second control group we propose consists of those individuals who fill out the questionnaire portions of the survey, but who decline the HIV blood test component. In terms of the model we presented above, this consists of the union of those people with prior probabilities very close to zero or one,

¹⁹ Although, of course since testing reveals to them precisely whether they are HIV positive or negative, this group may be thought of as mass points at 0 and 1 at the time of the survey. In that sense then, Figure 3 may be thought of as a depiction of the 'initial conditions', but not representative of the position of their priors of this first control group at the time of the survey.

and whose benefits to testing lie below even the lower subsidized cost of testing through the survey C_s . Thus this group consists of those respondents with prior probabilities in $[0, L_s]$ and $[U_s, 1]$ who do not value testing beyond its cost even when subsidized. The treatment group consists of those individuals for whom the private cost of testing exceeds the benefit, but that the cost of testing through the survey (the 'subsidized cost' C_s) is lower than the benefit. Note that this group is 'sandwiched' between these two control groups. This is important, since the model predicts that the 'information treatment' administered to those who learn of their HIV status through the survey will vary by their initial prior probability of infection. Thus, if the intent of the control group is to try to 'hold constant' the prior probability of infection, then these two control groups will do so in different ways. In particular, the 'sandwich' effect should allow us to bound the treatment effects. If however, the (unchanging for both control groups) prior probability of infection is unrelated to the *change* in sexual behavior over the two periods (but perhaps the *level*), then we would expect that the two control groups would yield the same treatment effect.

The most unique aspect of the data we use to study the effects of the impact of a change in a person's information set (in this case, concerning his or her HIV status) is for those people who were uninformed of their HIV status before the survey (i.e., were not *private* demanders): by participating in the survey, their information set is altered. This gives us the opportunity to *directly* observe the impact of a change in an economic agent's information set on their resultant

behavior in a longitudinal setting. In particular, we know before the testing whether the agent has prior knowledge of his or her HIV status. As the discussion in the previous section indicated, the *aggregate* behavioral effect of this testing program is expected to be close to zero when people are symmetrically distributed with respect to their prior beliefs of infection. As a result, and as suggested by Figure 2, we disaggregate individuals based on what we infer their prior (or subjective) probability of infection to be. We have experimented with a logit model which uses a variety of demographic and sexual orientation characteristics of the respondents to predict their (observed) HIV status. We then used the predicted probabilities of infection from this logit to infer whether a respondent would think of him or herself as low or high risk (defined as above or below the median predicted probability in the sample). However, the dominant risk factor in that version of the analysis was clearly sexual orientation.²⁰ Thus for clarity and simplicity, we use instead the dominant risk factor of sexual orientation to proxy for the unobserved prior probability of infection, and classify homosexuals as 'high-risk' and heterosexuals as 'low-risk' based on the prevalence of HIV in the respective populations.²¹ We present all results bro-

²⁰Indeed, at the time of this survey, the incidence of HIV amongst heterosexuals and homosexuals in our sample is vastly different. The incidence for heterosexuals is less than 1 percent, while for homosexuals it is 30 percent. Along no other observable dimension is this difference in incidence so stark.

²¹We should be clear in the use of the term 'risk' in this context. The term is meant to imply that, based on the *observed* prevalence rates in the sample, a given homosexual respondent is at greater 'risk' of being HIV positive than is a given heterosexual respondent.

ken down by this grouping since the nature of the 'information intervention' depends upon each person's initial prior probability of infection (in addition to what test result each person actually obtains). Breaking our sample into these discrete high prior and low prior probability of infection subgroups gives us a 2 by 2 table of 'treatment effects'.

3.2 Empirical Results

We used the first two waves of the SFHHS (conducted 12 months apart) to construct data that measured the quantity (total number of partners) and quality (extent of sexual protection given sexual contact) of sexual trades that were taking place before and after the blood testing component of the survey. We make use of two control groups: the first uses those respondents who had knowledge of their HIV status before the survey since their information sets would be largely unchanged by the blood test administered through the survey. The second control group we make use of is of those respondents who turned down the blood test administered through the survey, but who fill out the questionnaire portion of the survey. In both cases, we study the impact of the testing intervention separately for Homosexual and Heterosexual respondents (proxying for high and low risk of infection respectively), to avoid reporting just an aggregate effect that would mask important differences in behavioral changes by risk group (in particular, differences that are roughly equal and of opposite sign).

TABLES 2A & 2B INSERTED HERE

In Tables 2A and 2B we present the means of the variables used in the analysis for our 'treatment' and 2 control groups broken out by the risk classes (Homosexual and Heterosexual) for comparison. Table 2A presents the means for the Homosexual and Heterosexual respondents who are HIV positive (for completeness in Tables 2A and 2B we report means for those respondents who refuse the HIV test, and so their HIV status is unknown). In Rows 2 and 3 we present the means of the *level* of the outcome variable of interest, the number of sexual partners in the past 12 months. In Row 4 we present the *change* in the number of partners over the two waves. While the means of the levels are quite different for Homosexuals who learn they are HIV positive than for those who knew prior to the survey they were HIV positive, we see that the difference in the change in the number of partners is within a standard error of each other (-0.86 vs. -0.45). Those homosexuals who learn through the survey they are HIV positive are slightly more likely to be white and low education/income than those who knew this ahead of time, or those who refused the test altogether. In addition, they are slightly younger. The differences along observable demographic characteristics is not great, and in analysis we do below, we control for what observable differences that *do* exist. Although this is not an incredible rich set of controls for the outcome we study, we hope that the slight differences along observable dimensions is related to no more than slight differences along unobservable dimensions, in so far as our outcome variable in

concerned.

TABLE 3 INSERTED HERE

In Table 3 we present the unconditional effects of providing respondents with private information has on quantity and quality of trade, conditional on the sexual orientation of the respondents.

The advantage of presenting the results in this format first are that the reader can see the relevant cell sizes (given in brackets below the point estimates and the standard errors in parentheses for each cell), and underlying point estimates. In Tables 4 and 5 below, we reproduce these results in a regression format in which we can readily include covariates to control for observed heterogeneity. Table 3 concerns the quantity effects (i.e., the change between the two waves in the number of sexual partners) that learning one's HIV status through the survey has relative to those who knew their status before the survey. The first two rows of the table present the mean change in partners for those who *learn* their HIV status through the survey. The second two rows present the mean change in the number of partners for those who *know* their HIV status prior to the start of the survey (our first control group). The bottom two rows present the 'difference-in-difference' estimates which are simply the difference in the estimates in the first and third rows and then the second and fourth rows respectively. These effects are the mean effect of *learning* one is HIV positive (row 5) or HIV negative (row 6) relative to knowing this information prior to the survey. The two columns of the table do this exercise separately for homosexuals and heterosexuals.

The point estimates imply that those homosexuals who learn they are HIV positive through the survey (a total of 19 sample respondents) drop 0.86 partners on average in the 12 months between the two surveys. Even with such a small cell size, this estimate is significantly negative at conventional significance levels. However, when we compare this negative estimate with the mean change for those who knew they were HIV positive before the survey commenced (-0.45) we find that the bulk of this -0.86 is potentially *not* due to the effect of *learning* that one is HIV positive, but instead perhaps reflecting an overall trend towards safer sexual practices for individuals in a high risk group. The difference in these two changes (the difference-in-differences) estimate of -0.41 is well below its standard error ($t=0.63$) and insignificantly different from 0, thus reflecting that for the homosexual population the effect of learning HIV positive does not generate a significant change in behavior. This result is consistent with the theoretical model outlined above, in that those in the high prevalence of HIV group (homosexuals) would have prior probabilities of infection that would induce them to behave more like they are HIV positive than negative. Thus *learning* that one is HIV positive does not tend to generate large changes in behavior.

Similarly, if we look at the effects of learning one is HIV negative for the homosexual group, we see smaller in magnitude negative trends in the change of the number of partners, but we see a stronger negative trend for those homosexuals who know before the survey they are HIV Negative. As a result, the

difference-in-difference estimate of the effect of learning that one is HIV negative for the homosexuals, we see that this knowledge is actually associated with an *increase* in the total number of partners (0.80 with s.e.=0.35). This rather surprising finding can also be interpreted in the context of the model outlined above, since for homosexuals, learning that one is HIV negative generates a rather large change in information. If sexual behavior is elastic with respect to information on HIV status, then we should expect to see greater changes in behavior for the Homosexuals who learn they are HIV negative than learn they are HIV positive. While the point estimates of the effect of learning one is HIV positive is roughly half that of the effect of learning one is HIV negative through the survey, the statistical significance of this latter *positive* estimate ($t=2.3$) is substantially greater. This rather surprising finding can be interpreted in the context of the model presented above.

It is important to note that the low cell frequencies in Table 3 of in the cells where we would expect to see the most 'action' are inherent in this setup. In particular, alternative means of classifying risk (instead of using a single indicator of risk, but using a logit to yield predicted probabilities of infection in a multivariate setting), will still yield relatively small numbers of observations in these cells. However, the greatest behavioral response to the endowment of information will most likely fall in those cells, since it is for those types of respondents that the observed blood test result (i.e., the posterior probability of infection) is altered most from the prior probability of infection. If priors

are set by the respondents in accord with average posterior probabilities, as would be the case when beliefs are consistent with objective frequencies, then the greatest changes in beliefs occur among low-risk individuals who learn they are HIV-positive and high-risk individuals who learn they are HIV-negative. This is because they were 'doing the wrong thing,' in terms of behavior based on prior beliefs that turned out to be different from the truth.

INSERT TABLE 4 HERE

Table 4 examines the robustness of the 'difference-in-difference' results to the addition of covariates to account for *observed* heterogeneity. The first and third columns of Table 4 reproduce the 'difference-in-difference' estimates from Table 3. The coefficients on what we will call the 'learning effects' (Rows 3 and 4) are the 'difference-in-difference' estimates from Table 3. Again, we see the estimates that are greatest *in magnitude* are the Learn Negative effects for the Homosexual group, and the Learn Positive effect for the Heterosexual group, consistent with the theoretical model outlined in Section 2. However, only the Learn Negative effect for the Homosexual group is statistically significant (the large Learn Positive effect for Heterosexuals being identified off of only 8 individuals, as shown in Table 3). The bottom line from Table 4 is that the addition of demographic indicators for race/ethnicity and age add little to the explanatory power of the model (bear in mind the dependent variable is *differenced* thus helping to account for the low R-squared of the regressions in Table 4 and those presented below as well). In addition, in columns 3 and 6

of Table 4 we include indicator variables for having 'low education' and 'low income' and being covered by medical insurance to the model. Again the R-squared changes little, and the point estimates on the learning effects changes negligibly.

However, the results in Table 4 may also be explained by some form of unobserved heterogeneity. Since we lack randomization of individuals into a 'treatment' (offered the HIV test) and 'control' groups (a subset of the sampled population from whom the offer of free, in-home HIV testing is withheld), we will necessarily be subject to the criticism that our point estimates are biased due to some form of selectivity. For example, in the present context, a plausible explanation of our results, apart from any true 'learning' effect would be that our control group which is composed of individuals who tested prior to the survey, are a 'safer sex' group of people. The fact that they tested prior to the survey is simply one dimension by which their attitudes toward risk manifest themselves. In the face of a growing HIV epidemic, they may also be engaging in other dimensions of safer sex, such as reducing their number of sexual partners, greater prophylactic usage when engaging in sexual contact, etc. As a result, relative to our 'treatment' group, those who did not have an HIV test before the survey, we may be subtracting off too large of a negative trend in the change in the number of partners over time, thus generating the results we obtained in Table 4, but which have nothing to do with the model we proposed to explain those results. Unfortunately, the data set does not contain plausible instruments

which could affect the desire to obtain an HIV test, but also meet the exclusion restriction of not affecting the outcome of the change in the number of sexual partners over the intervening 12 months. Thus, we present instead results based on a *second* control group, which are qualitatively *very* similar to the results we obtained in Tables 3 and 4, but slightly less statistically significant. Indeed, earlier work based on different cuts of the data are also quite consistent with the results shown here, indicating to us that the results we present here are certainly representative of the overall pattern of results in the data.

We consider now this control group which consists of the considerable number of people who filled out the questionnaire portion of the survey, but who refused the blood test component of it (we call this group the Refusers). There are 254 such people in our data who have completed the questionnaire, which is roughly 14 percent of the overall sample. This group, as well as those who tested before the survey, should be unaffected by the 'treatment' of the HIV test administered through the survey. However, they like those who tested before the survey, are a non-random subset of the overall sample, and so we are not immune from arguments that our results here are also driven by self-selectivity. While we acknowledge this potential flaw, we argue that unobserved heterogeneity arguments like those presented in the previous paragraph would not likely apply to this control group. In particular, it is difficult to see why those individuals who refused an essentially free test (apart from the psychic costs of testing), could be seen as a group exhibiting preferences toward 'safe sex' more

so than those people in our treatment group. For this, and a variety of other reasons, we report results based on this second control group in Table 5, which indicate that qualitatively, the results from Tables 3 and 4 are robust.

INSERT TABLE 5 HERE

The 'learning' effects in Table 5 are again given by the interaction terms in Rows 2 and 3.²² We again see, as in Table 4, that the effect of learning one is HIV positive for the homosexual respondents, leads to a slight decline in the change in the number of partners that is within one standard error of zero. Consistent with the *positive* growth in the number of partners for the homosexuals who learn through the survey they are HIV negative found in Table 4, we find a positive effect using this second control group as well, although the magnitude is about 60 percent of that in Table 4. The standard error is roughly the same, and the t- statistics on these coefficients are roughly 1.5, which is insignificant at conventional significance levels. However, the consistent pattern of the results obtained from using both control groups, and given the rather small size of the overall survey, lead us to conclude that the results in Table 5 are broadly supportive of the results in Table 4.

²²Notice that there is no main effect for HIV Positive in these regressions as in Table 4, since we do not have the HIV status for those respondents who refused the blood test administered by the survey.

4 Conclusion

Our results have important implications for the possible effects of public HIV testing programs which would enhance the testing for HIV either through subsidizing the cost of HIV testing or through requiring testing through certain channels. Such programs are often proposed as an instrument to combat the spread of HIV and AIDS. Proponents of these programs have in mind that those who learn they are HIV-positive will take subsequent action so as to limit the spread of the disease. Such an analysis, however, ignores the possibility that individuals have private assessments of the probability that they are infected, and that these prior beliefs help formulate their sexual practices and behavior. If individuals who test through a public testing program are largely those individuals who considered themselves to be likely carriers of HIV to begin with, then the testing program may do little to alter their behavior. Such individuals may well be better served through HIV awareness programs and education programs oriented towards prevention, although that is an open question for further research. These analyses also ignore the impact of testing on those individuals who learn they are HIV negative. We found here a tendency for these individuals to *increase* their number of sexual partners. This presents the possibility that a public testing program could perversely enhance the spread of HIV, although we are not in a position to ascertain the likelihood of that happening. In any event, the benefits of subsidized testing, in terms of significantly lowering the amount of sexual contact for those who learn they are HIV positive, do not

appear to be present in our data.

We take away from this particular exercise several lessons for future empirical work attempting to directly observe information or its changes, and its impact on market behavior.

First, in our case, while the *change* in information for different people in our setting *was* observable, its impact was not. In particular, unless everyone in the market has the same prior beliefs, the same change in information to two people will result in different changes in behavior if they have different prior beliefs. Thus, we still have to rely on the somewhat artificial device of resorting to groupings of subpopulations to proxy for prior beliefs. In this case, since we are dealing with prior beliefs of HIV infection, we point to the dramatic differences in infection rates for homosexuals and heterosexuals, and use the proxy of sexual orientation for the high and low prior probabilities of being HIV positive. Thus future work needs to take into account that in situations where prior beliefs vary over the population, simply having access to observable information changes is not enough to avoid ad hoc assumptions in creating proxies. In short, this is because the information treatment will be different for people with differing, unobservable prior beliefs. Future work should try to identify settings where priors are more homogeneous, or when they differ, where such differences can be measured.

In addition, we recognize that in most markets stark differences in information changes, such as we have with people learning they are either HIV positive

or negative (and nothing in between), are not discernable. For example, when people apply for a bank loan a credit check can determine if they are 'good' or 'bad' risks, although a fail-safe test for such riskiness does not exist. Furthermore, there are gradations of riskiness and it is not a simple binary indicator as is the case with HIV status. Thus, empirical assessments which use some measure of observable information need to take into account the reliability of measurement device (which in our case is virtually 100 percent).

Finally, models of *asymmetric* information concern not just an individual level change in an agent's information set, but also the degree to which that information gets passed on to other agents in the market. Empirical assessments of such models need to try to measure *directly* the correlation between the change in an individual's information set with the change in the market's perception of that individual. Otherwise, only conclusions regarding whether the market is best characterized by a pooling or a separating equilibrium appear to be recoverable from only individual level data. In our setting, such assessments could be made if we contacted our respondents' partners and surveyed *them* as to whether or not they thought the *respondent* was HIV positive or negative. By focusing attention on those respondents who learned their HIV status through the survey, and seeing if their partners' beliefs as to their HIV status changed as a result of the testing done through the survey, we could measure directly the correlation of the partners' beliefs with the respondent's updates in HIV status. In that case, we could make direct inferences as to the degree of asymmetric

information in our setting by using *direct* measures of observable information. We acknowledge, however, the parallel to other markets would be difficult to carry out in practice.

There is, however, a difficulty with such a *two-sided* (i.e. sample the respondent and his or her partner(s)) sampling scheme. In contrast to the one-sided sampling scheme used in collecting our data, where only the respondent was sampled, with no identifiable link to his or her partner(s) (even though some of them may have been sample participants as well), a two-sided sampling scheme may run into incentive problems. In particular, if the respondent knows that the survey is two-sided, and so the interviewers will be explicitly surveying the people he names as his partners, he may have an incentive to respond in the same way to the interviewer as he would to his partner's queries. In particular, if he has lied to his partner, then two-sided sampling may create incentives for him to lie to the interviewer as well, whereas one-sided sampling may not. Thus there is the possibility of Hawthorne effects, whereby the sampling process itself affects the population sampled, apart from the intervention of interest. In devising surveys to capture the degree of asymmetric information in a market, we urge researchers to give careful consideration to the possibility of such Hawthorne effects that may arise from direct two-sided sampling schemes.

Informational effects on markets are well characterized by a voluminous theoretical literature. But empirical work on these models usually treats the information under question much like a residual in an econometric model, in that it is

unobserved, but together with the independent variables, can be used to explain the dependent variable completely. Unfortunately, for most explicit markets we cannot construct a test (like an HIV test in our context) to yield a perfect discrimination of each market trader's 'type'. But if surveys are designed for the explicit purpose of testing these theoretical models, we can begin to amass more *direct* evidence of the role that information imperfections play in determining market behavior and market inefficiencies.²³ We are hopeful that the sampling needs discussed briefly in this conclusion are helpful in the design of such surveys and in their implementation.

²³For example, the survey used in Ashenfelter and Krueger (1994), conducted at a twins festival, was designed explicitly for estimating a return to schooling with unobserved family components, and allowed for a correction to an exacerbated measurement error problem. The survey was designed to sample *directly* 2 or 3 key components in the debate over the magnitude of the economic return to schooling. In particular, through a rather clever survey design, they could estimate a reliability ratio for measured schooling, a component not observable with cross-section data (or even longitudinal data, except under strong assumptions). The combination of these variables was not available in other datasets.

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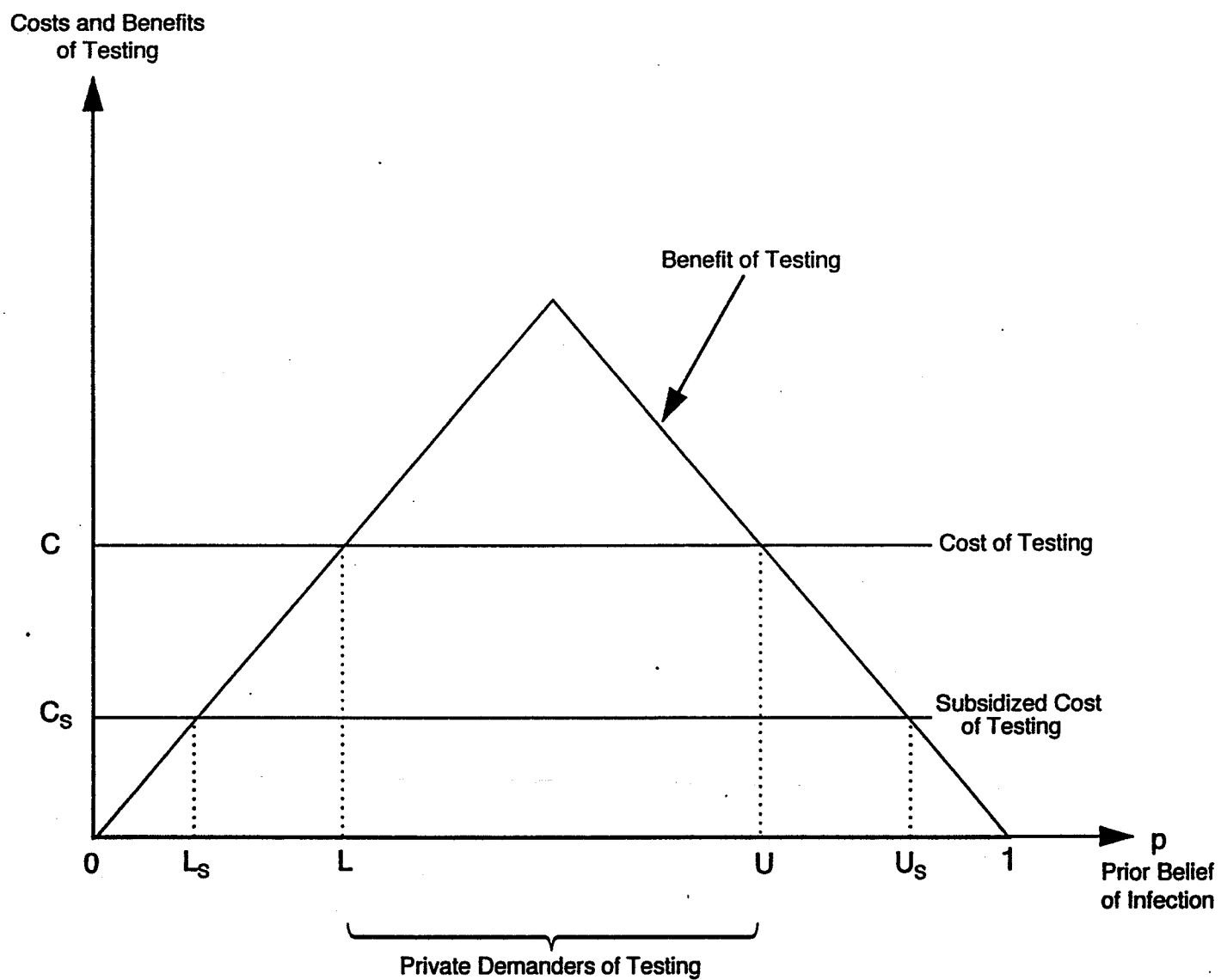


Figure 1: Private and Public Testing

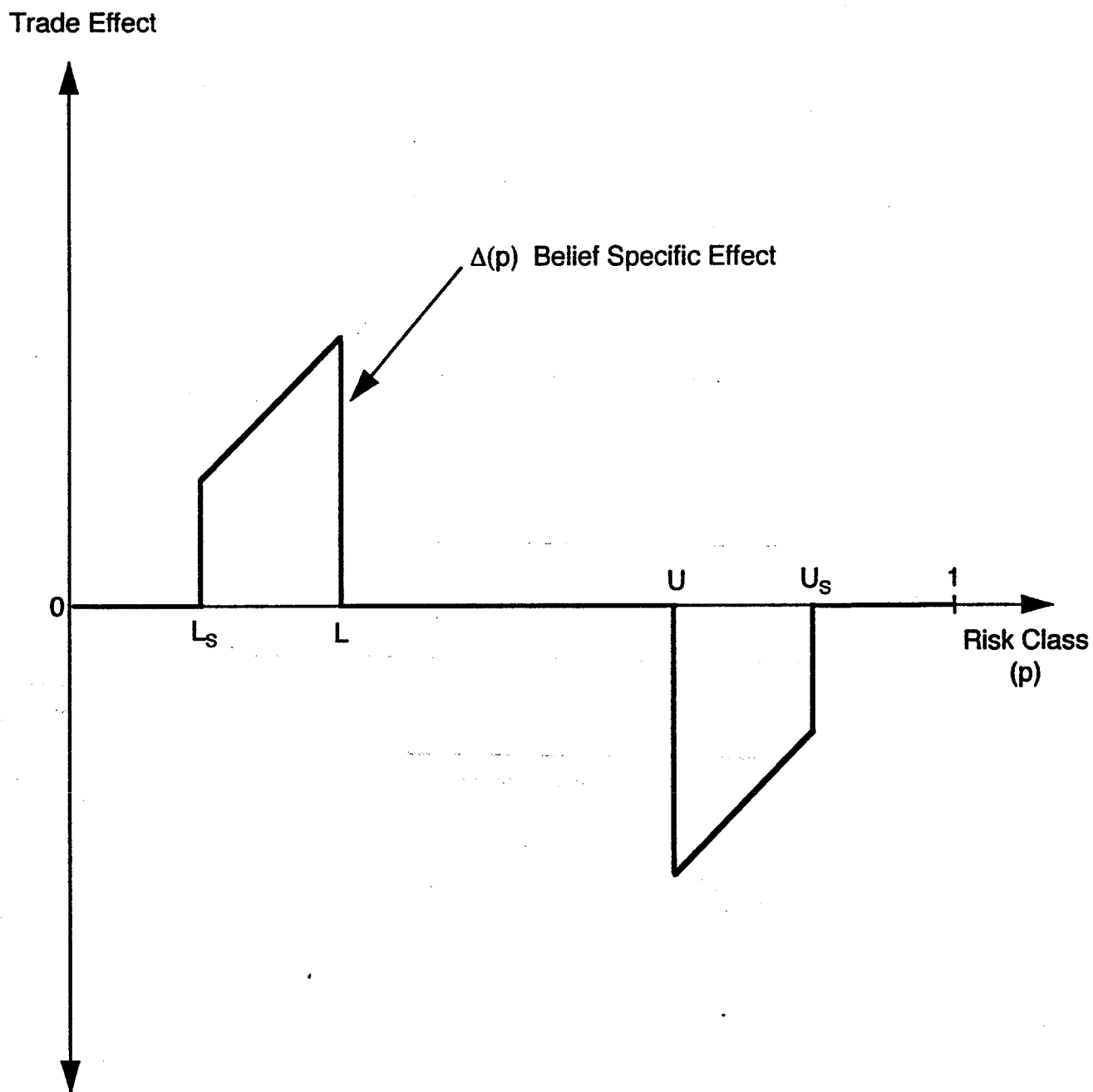


Figure 2: Effects of Public Testing as a function of Risk Class

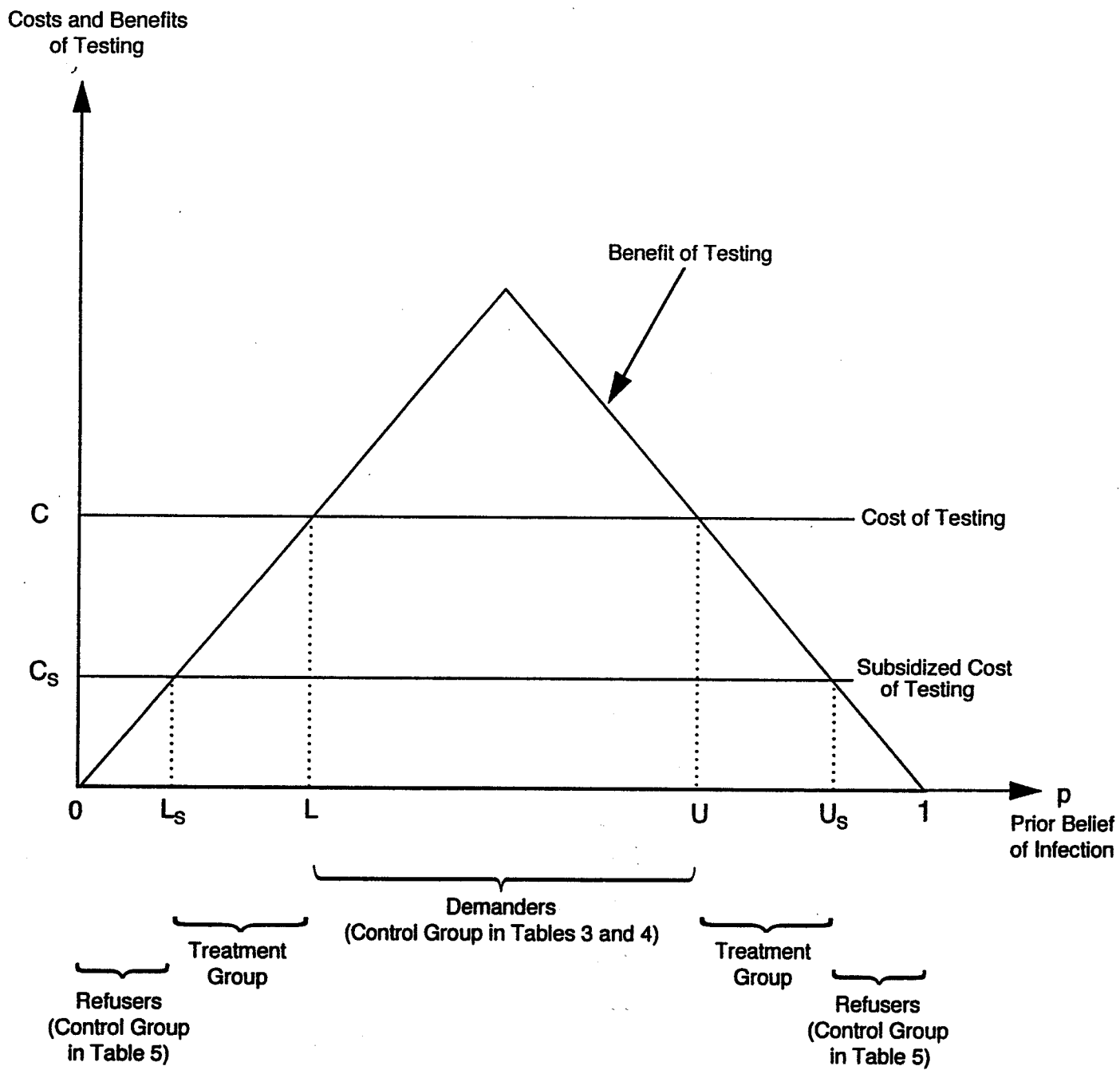


Figure 3: Private and Public Subsidized Testing Treatment and Control Groups

Table 1
Comparison of the means in the SFHHS and the US Population¹

Variable	SFHHS Sample Mean ²	NHSLS ³
Male	0.47	0.55
Homosexual	0.14 (0.35)	0.02 ⁴
Age	30.0 (6.79)	36.4
[Range:	20-44	18-59]
White	0.44	0.77
Black	0.25	0.13
Know HIV Status	0.22	
HIV Positive	0.04	
Total Number of Partners Wave 1	2.24 (1.96)	
Total Number of Partners Wave 2	1.99 (1.73)	
Ever Use Condom During Sex Wave 1	0.52	
Ever Use Condom During Sex Wave 2	0.59	
Learn HIV Pos.	0.02	
Learn HIV Neg.	0.76	
Know HIV Pos.	0.03	
Know HIV Neg.	0.19	
Response Rate	62%	80%
Number of obs.	1110	3432

¹ The US Population figures come from a random sample of American men and women called the National Health and Social Life Survey (NHSLS) on sexuality conducted by Laumann, Gagnon, Michael and Michaels, at the University of Chicago, 1994.

² Sample means as of the first wave, 1989, standard deviations for non-binary variables are in parentheses.

³ These are the weighted means for the NHSLS sample given in their Appendix B.

⁴ In keeping with the definition from the UCSF, we use the response from the NHSLS which is the "self-identification" response, rather than previous experience with same gender sexual situations (this number would be about 5% had we used that definition). In addition, using the Laumann et. al Table 8.2 we compute that this figure is about 0.03 for men and women aged 18 to 39.

Table 2A
Means for Treatment and Control Groups by Sexual Orientation

Variable	Homosexual			Heterosexual		
	Learn HIV +	Know HIV +	Refuse Test	Learn HIV +	Know HIV +	Refuse Test
Number of Obs.	14	31	44	5	3	210
Number of Partners, Wave 1	3.14 (0.81)	4.42 (0.69)	1.93 (0.46)	3.60 (1.69)	1.33 (0.67)	1.45 (0.11)
Number of Partners, Wave 2	2.29 (0.65)	3.97 (0.67)	1.54 (0.34)	2.60 (0.68)	1.33 (0.67)	1.40 (0.10)
Change in Number of Partners	-0.86 (0.40)	-0.45 (0.51)	-0.39 (0.38)	-1.00 (1.52)	0.00 (1.15)	-0.05 (0.11)
White	0.79	0.68	0.66	0.40	0.33	0.30
Black	0.07	0.10	0.11	0.60	0.00	0.34
Hisp	0.07	0.13	0.11	0.00	0.67	0.25
Age	31.0 (1.68)	35.0 (0.94)	32.9 (0.84)	31.4 (2.46)	34.7 (3.84)	29.3 (0.47)
Low Education	0.29	0.13	0.09	0.06	1.00	0.30
Low Income	0.43	0.29	0.27	0.04	1.00	0.58
Medical Insurance	1.00	0.97	0.93	0.80	0.67	0.85

Notes: Standard errors for the non-binary variables are in parentheses.

Table 2B
Means for Treatment and Control Groups by Sexual Orientation

Variable	Homosexual			Heterosexual		
	Learn HIV -	Know HIV -	Refuse HIV Test	Learn HIV -	Know HIV -	Refuse HIV Test
Number of Obs.	67	44	44	747	252	210
Number of Partners, Wave 1	0.79 (0.22)	2.70 (0.49)	1.93 (0.46)	1.66 (0.06)	1.89 (0.11)	1.45 (0.11)
Number of Partners, Wave 2	0.90 (0.25)	2.00 (0.41)	1.54 (0.34)	1.53 (0.06)	1.65 (0.11)	1.40 (0.10)
Change in Number of Partners	0.10 (0.16)	-0.70 (0.31)	-0.39 (0.38)	-0.13 (0.06)	-0.23 (0.10)	-0.05 (0.11)
White	0.73	0.80	0.66	0.42	0.44	0.30
Black	0.12	0.07	0.11	0.26	0.23	0.34
Hisp	0.10	0.07	0.11	0.24	0.26	0.25
Age	31.0 (0.75)	33.3 (0.84)	32.9 (0.84)	29.5 (0.25)	29.9 (0.42)	29.3 (0.47)
Low Education	0.15	0.09	0.09	0.37	0.38	0.30
Low Income	0.49	0.43	0.27	0.65	0.63	0.58
Medical Insurance	0.97	0.93	0.93	0.85	0.85	0.85

Notes: Standard errors for the non-binary variables are in parentheses.

Table 3
The Effect of Subsidized Testing on
the Change in the Number of Sexual Partners

Total Partners Wave 2 - Total Partners Wave 1		
	Homosexual	Heterosexual
Uncertain HIV status before the survey		
Learn:		
HIV Positive	-0.86 (0.40) [14]	-1.00 (1.52) [5]
HIV Negative	0.10 (0.16) [67]	-0.13 (0.06) [747]
Known HIV status before the survey		
Know:		
HIV Positive	-0.45 (0.51) [31]	0.00 (1.15) [3]
HIV Negative	-0.70 (0.31) [44]	-0.23 (0.10) [252]
"Difference-in-Differences"		
Estimate of the Effect of Public Testing:		
Learn:		
HIV Positive	-0.41 (0.65)	-1.00 (1.91)
HIV Negative	0.80 (0.35)	0.10 (0.11)

Notes: Standard errors are in parentheses, and number of observations for each cell are in brackets.

Table 4
Regressions For the Effects of Subsidized Testing on the
Change in the Total Number of Partners:
(Uses those who know their HIV status prior to the survey as the control group)

Independent Variable	Dependent Variable = Total Partners Wave 2 - Total Partners Wave 1					
	(1)	Homosexual		(4)	Heterosexual	
		(2)	(3)		(5)	(6)
Intercept	-0.70 (0.29)	2.33 (4.83)	1.13 (5.03)	-0.23 (0.10)	-2.34 (1.10)	-2.45 (1.14)
HIV Positive	0.25 (0.45)	0.20 (0.46)	0.24 (0.47)	0.23 (0.90)	0.22 (0.90)	0.16 (0.90)
Learn HIV Pos. (Learn*HIV Pos.)	-0.41 (0.62)	-0.34 (0.64)	-0.37 (0.65)	-1.00 (1.13)	-1.04 (1.13)	-0.98 (1.14)
Learn HIV Neg. (Learn*HIV Neg.)	0.81 (0.37)	0.83 (0.39)	0.84 (0.39)	0.11 (0.11)	0.11 (0.11)	0.11 (0.11)
White	----	-0.41 (0.65)	-0.40 (0.65)	----	-0.27 (0.20)	-0.26 (0.20)
Black	----	-0.54 (0.80)	-0.68 (0.82)	----	-0.06 (0.21)	-0.09 (0.21)
Hisp	----	-0.20 (0.80)	-0.26 (0.82)	----	-0.06 (0.21)	-0.08 (0.21)
Age	----	-0.19 (0.29)	-0.14 (0.29)	----	0.16 (0.07)	0.16 (0.07)
Age Squared (x 10)	----	0.03 (0.04)	0.03 (0.04)	----	-0.03 (0.01)	-0.03 (0.01)
Low Education	----	----	0.18 (0.49)	----	----	0.06 (0.12)
Low Income	----	----	0.43 (0.35)	----	----	0.05 (0.11)
Medical Insurance	----	----	-0.06 (0.86)	----	----	-0.06 (0.15)
R-Squared	0.04	0.05	0.06	0.002	0.01	0.01
Number of Observations	156	156	156	1007	1006	1006

Note: Standard errors are in parentheses.

Table 5
Regressions For the Effects of Subsidized Testing on the
Change in the Total Number of Partners:
(Uses those who refuse the HIV test through the survey as the control group)

Independent Variable	Dependent Variable = Total Partners Wave 2 - Total Partners Wave 1					
	(1)	Homosexual (2)	(3)	(4)	Heterosexual (5)	(6)
Intercept	-0.39 (0.28)	1.95 (4.97)	1.08 (5.07)	-0.05 (0.11)	-3.44 (1.13)	-1.41 (1.37)
Learn HIV Pos. (Learn*HIV Pos.)	-0.47 (0.57)	-0.41 (0.58)	-0.58 (0.60)	-0.95 (0.70)	-1.04 (1.13)	-0.95 (0.70)
Learn HIV Neg. (Learn*HIV Neg.)	0.49 (0.36)	0.57 (0.37)	0.54 (0.38)	-0.08 (0.12)	0.11 (0.11)	-0.03 (0.12)
White	----	-0.59 (0.67)	-0.55 (0.67)	----	-0.27 (0.20)	-0.42 (0.20)
Black	----	-0.61 (0.81)	-0.88 (0.82)	----	-0.06 (0.21)	-0.09 (0.20)
Hisp	----	-0.69 (0.83)	-0.75 (0.84)	----	-0.06 (0.21)	0.01 (0.21)
Age	----	-0.15 (0.31)	-0.10 (0.31)	----	0.16 (0.07)	0.23 (0.08)
Age Squared (x 10)	----	0.03 (0.05)	0.02 (0.05)	----	-0.03 (0.01)	-0.04 (0.01)
Low Education	----	----	0.87 (0.54)	----	----	-0.06 (0.12)
Low Income	----	----	0.00 (0.37)	----	----	0.09 (0.12)
Medical Insurance	----	----	-0.08 (0.92)	----	----	0.23 (0.15)
R-Squared	0.03	0.05	0.07	0.002	0.02	0.03
Number of Observations	125	125	125	962	961	961

Note: Standard errors are in parentheses.