Social Learning, Selection, and HIV Infection

Evidence from Malawi

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ABSTRACT

This paper examines social learning regarding HIV infection, using HIV test results and sibling death data from Malawi. In the analysis, we compare hypotheses on social learning, selection. and common factors. Empirical results show that young women are less likely to be HIV-infected if they observed prime-age deaths among their siblings, whereas HIV infection is found to be positively related to prime-age sibling deaths among older women. This supports the social-learning hypothesis. Notably, schooling reinforces the social-learning effect of sibling deaths on HIV infection in women regardless of age. The above findings are robust to age (cohort) effects and unobserved location factors.

Keywords: social learning, HIV infection, siblings, Malawi

1. INTRODUCTION

It is increasingly recognized that certain behaviors, such as high-risk sexual conduct, increase the probability of HIV infection. Once an individual is infected with HIV/AIDS, he/she will experience serious health problems with substantial economic burdens, most often leading to death. Due to the irreversibility of infection, agents cannot learn of HIV consequences from their own experiences, but instead must learn from the experiences of others in order to optimize their behavior. Therefore, social learning and social networks affect the agent's perceptions and behavior (e.g., Kohler, Behrman, and Watkins 2007; Helleringer and Kohler 2005).[1]

The literature shows that social learning is also important in some empirical contexts, such as technology adoption and schooling investments (e.g., Foster and Rosenzweig 1995; Munshi 2004; Yamauchi 2007; Conley and Udry 2004). In particular, learning about HIV is comparable to learning in human capital investment (Yamauchi 2007), where irreversibility excludes the possibility of learning-by-doing. In the case of schooling investment, agents cannot learn of returns from their own investments, since, in addition to being irreversible, the investment occurs over a relatively long period.

This paper aims to empirically verify the social-learning hypothesis using HIV test result data from Malawi. For this purpose, we use the prime-age death (age 15-49) of siblings (reference group) to investigate the agents' learning, assuming that siblings' deaths convey important information on the relationship between the siblings' behavior and HIV infection and AIDS.[2] We hypothesize that agents learn from deaths (or illness) of their siblings, subsequently changing their behavior in order to lower their own HIV infection risk.[3]

The use of siblings' death information allows us to exclude the possibility of direct transmission between agents and their reference groups. Even though agents may learn from their neighbors, it is also possible for them to become infected directly from their neighbors through sexual interactions, making the neighbor group inappropriate for use as a reference group. Although siblings do not necessarily live in the same location and are not necessarily physically close, their social proximity is high during their formative years. This setting is suitable for social learning, with the caveat that they may share (often unobserved) family-fixed characteristics that could increase the positive correlation of their behavioral outcomes.

When seeking to identify social learning in the context of HIV infection, we must also consider other possibilities beyond sibling interactions. First, a dynamic selection process is involved in HIV infection among siblings. For example, those who take higher risks in their sexual activities are more likely to become HIV-infected and die of the disease. Given that there are innate differences in risk preference, siblings can have different infection probabilities, with less risk-averse agents will be more likely to become infected and therefore die of AIDS, compared to the more risk-averse agents. Thus, if the women in our sample are risk averters while those who died of the disease were risk lovers, we should observe a negative correlation between prime-age death (from AIDS) among siblings and HIV infection. However, this selection process is likely to take a rather long period, and such a negative association would be due to dynamic selection ex post. Therefore, it is highly likely that we would observe this association in the sample of older women but not in young women.

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[1] Recent empirical studies examine the formation of perceptions on AIDS. For example, see Bernardi (2002), Kengeya-Kayondo et al. (1999), London and Aroyds (2000), Smith and Watkins (2005), and Watkins (2004).

[2] Information on sibling's illness is also potentially important to the agent's decision making on sexual behavior, thus determining HIV infection risks. However, the data we use (the Demographic and Heath Survey, Maternal Mortality Module) does not capture this information, as it is mainly designed to investigate child mortality among the respondent's siblings.

[3] Manski (1993) clarified the importance of identifying a reference group in empirical studies of social learning and reflection problems. Foster and Rosenzweig (1995), Munshi (2004) and Yamauchi (2007) assumed that agents learn from neighbors in the same village, based on geographical proximity. Conley and Udry (2004) used information flow channels (from their survey data) to carefully identify paths through which agents learn. In HIV/AIDS research, Kohler et al. (2007) and Helleringer and Kohler (2005) attempt to identify social networks through which an agent's perceptions are formed.

Second, as discussed above, unobserved family-fixed factors may determine HIV infection among siblings. In other words, if the family factors are significant, HIV outcomes will be positively correlated among siblings but less so across families. Therefore, our empirical analysis of social learning in HIV infection critically depends on how and whether we can identify social learning against the above two alternative scenarios.

In our empirical analysis, we directly use information on HIV test results among Malawian women, under the assumption that HIV infection ultimately represents a behavioral consequence that reflects diverse individual choices in this high-HIV prevalence setting.[4] In 2004, antiretroviral treatment was not generally available to the public in Malawi.[5] Given that the life expectancy of an HIV-infected agent is fairly low in the absence of treatment, one study shows that women without antiretroviral treatment survive only 43.7 months (Holmes et al. 2006). Women found to be HIV-infected in 2004 are unlikely to have been infected before the infection of siblings who died of the disease. In other words, respondents who were HIV-positive in the 2004 survey must have been infected within a relatively short period prior to the survey. Conversely, assuming that survival time after HIV infection is relatively consistent for all siblings in the absence of treatment, a sibling's death means that the sibling was infected earlier than the agent.

To verify our hypothesis, we also examine condom use and extra marital affairs. The information on these choices can contain measurement errors, and therefore does not provide a precise measurement of behavioral consequence; accordingly, we use it as supporting evidence, but focus our main analysis on directly examining HIV infection.

The paper is organized as follows. Section 2 discusses our data and empirical strategy. We use data on HIV infection in a subsample of women, drawn from the Demographic and Health Survey in Malawi (2004). Section 3 summarizes our empirical results. First, we find that young women are less likely to be HIV-infected if they observed prime-age deaths among their siblings. This supports the social learning hypothesis. However, HIV infection is found to be positively related to prime-age sibling deaths among older women. Second, schooling reinforces the negative effect of sibling deaths on HIV infection, regardless of age. This suggests that human capital helps agents learn from the prime-age deaths of siblings. These findings are robust to age (cohort) effects and unobserved location factors. Concluding remarks are mentioned in the final section.

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[4] Marston, Harriss, and Slaymaker (2008) analyze non-response biases in the HIV test data, but conclude that such bias is not significant in the DHS. This may potentially bias our estimates to an unknown degree.

[5] Of the estimated 150,000 people who needed antiviral treatment in June 2005, an estimated 18,000-23,000 (12-15 percent) were receiving it.

2. EMPIRICAL STRATEGY

This study uses data drawn from the 2004 Malawi Demographic and Health Survey (DHS). The DHS surveys, conducted in various developing countries since the mid-1980s, are nationally representative surveys designed to collect information on marriage, fertility, family planning, reproductive health, child health, and HIV/AIDS. The survey focuses on reproductive-age women aged 15-49. For our purposes, the DHS survey is a uniquely large database containing information on HIV test results among a subsample of respondents; we use this subsample herein.

The survey asks respondents to list their siblings and indicate whether or not each sibling is alive at the time of the survey. Table A.1 shows distributions of number of siblings and age in the study population. The population is relatively young with median age of 24. The average number of siblings is 5 to 6. The available data include additional information on each sibling's current age (if alive) and year of death and age at death (if deceased). For the purpose of our study, the birth-death records of respondents' siblings are particularly important for estimating their prime-age deaths. We compute the mortality rate from the death records of the respondents' siblings.[6]

Since the respondents are, by definition, alive at the time of the survey (2004), the mortality rate estimates could be biased downward if survival probabilities are positively correlated among siblings. Conditional on an agent's survival at the time of the survey, the likelihood of her sibling's survival is higher than that of an agent who had already died by the time of the survey.

In our analysis, we use siblings as a reference group. This has the advantage of allowing us to exclude direct transmission of HIV between agents and the reference group. Neighbors can also be used as a reference group in some cases, but we cannot exclude the possibility of sexual transmission of HIV from this group. Accordingly, we herein focus on siblings as the reference group from which the agents learn about HIV.

We use HIV status as a measure of an agent's behavioral consequence (outcome). HIV infection status has the benefit of smaller measurement errors compared to other behavioral choices, such as condom use or partner choice. However, the available information on current infection status does not allow us to identify the age at which the agents became infected. Given the fact that many infected individuals die of AIDS within several years of infection, we take current infection status as reflecting cumulative behavioral choices over a few years prior to the survey.

In the empirical analysis, we estimate the following equation,

$$y_{ij} = \alpha + \beta D_i + \sum_a \gamma_a I(a_{ij} = a) + \mu_j + \varepsilon_{ij},$$

where y_{ij} is HIV infection status ($y_{ij} = 1$ if infected, or 0 otherwise) of agent (woman) i living in location j, D_i is the proportion of prime-age deaths among her siblings (see below), a_{ij} is her age, μ_j is the location-fixed effect, and ε_{ij} is an error term.

D_i is defined as

$$D_i = \frac{\sum_{k=1}^{S_i} I\left(d_{i,k} = 1, a_{i,k}^d \in \text{prime age}\right)}{N_i^s} \in [0,1],$$

where $d_{i,k} = 1$ if i's sibling k is dead and 0 otherwise, $a_{i,k}^d$ is the age at which sibling k died, and N_i^s is the total number of siblings belonging to i.

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[6] Similarly, Ueyama and Yamauchi (2008) used the prime-age mortality rate constructed from the sibling data to analyze the impact of prime-age mortality on marriage age among young woman.

By definition, if $N_i^s = 0$, D_i cannot be defined. We set $D_i = 0$ in this case, but it would also make sense to distinguish between the two cases: $D_i = 0$ when $\sum_{k=1}^{S_i} I\left(d_{i,k} = 1, a_{i,k}^d \in \text{prime age}\right) = 0$ and $N_i^s = 0$, since the situation of $N_i^s = 0$ creates no information.

As we described in this section, social learning from the reference group of siblings implies a negative effect of sibling deaths on HIV infection ($\beta < 0$). This prediction contrasts to the positive correlation that can arise from family-fixed factors ($E\left[\varepsilon_{ij} D_i\right] > 0$). Therefore, if social learning is important, we are estimating the upper bound on the effect of prime-age sibling death on HIV infection status.

Potentially, unobserved fixed components (beside family-fixed effects), as well as common or correlated shocks, can have similar effects on both HIV infection and sibling deaths, causing a positive correlation between sibling death and HIV infection, thereby creating an upward bias. To partially mitigate this potential problem, we include location-fixed effects to control for common or correlated shocks specific to each location. For example, we control for information locally available in the village, and common behavioral patterns in the village.

In our setting, however, we cannot control for individual-fixed components in the empirical analysis, as we do not have information on dynamic changes in HIV infection status or changes in prime-age deaths among siblings. If such a factor is correlated with siblings' deaths, this factor may bias our estimates. The nature of HIV infection and AIDS death means that the length of time an agent stays infected is likely to be bounded. This implies that the current HIV infection status reflects a relatively recent change of status from negative to positive. To check robustness of the above point, we also examine the effect of "recent" sibling deaths in the past 10 years on current HIV infection status.

As noted previously, a process of natural selection in the dynamics of HIV infection among siblings may lead to a negative correlation between HIV infection and prime-age sibling death. However, since this selection process would take a rather long period to screen out those who are likely to get infected, we are likely to observe such a negative correlation only among older women. Therefore, it is important to note possible age heterogeneity.

In the estimation, we include age-fixed effects to control for possible cohort effects. Since the infection process is cumulative in the sense that risky action at one time can cause an irreversible transition to HIV-positive status, we expect HIV infection to increase with age (see Figure 1).

Figure 1. Age-fixed effect estimates in HIV infection equation
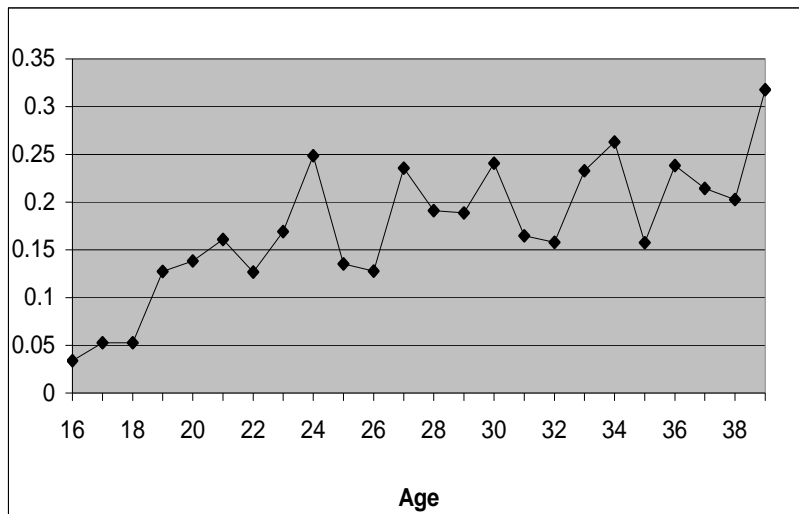
3. EMPIRICAL RESULTS

Table 1 shows our main findings on the effect of prime-age sibling death on HIV infection in the population of women aged 15 to 39, normalized by the proportion of siblings who died in their prime. In the estimation, we include all age dummies with location-fixed effects.

Table 1. The effects of prime-age death among siblings on HIV infection

Sample women of 15 to 39. Dependent: =1 if HIV infection, and =0 otherwise

			Age <=25	Age >26
Proportion of prime-age death among siblings	0.1176**	-0.1574*	0.3145	-0.2081
	(1.65)	(1.92)	(1.15)	(0.90)
* Age 26 or above		0.4060***		
		(3.46)		
Squared proportion of prime-age death			-0.8837*	0.6581*
			(1.67)	(1.79)
No sibling	0.0563	0.0601	0.0792	0.0627
	(1.07)	(1.15)	(1.07)	(1.79)
Age dummies	Yes[a]	Yes	Yes	Yes
Location-fixed effects	Yes	Yes	Yes	Yes
Number of observations	2,428	2,428	1,367	1,061
R squared (within)	0.0571	0.0622	0.0564	0.0303

Notes: Absolute t-values are in parentheses, using robust standard errors with location clusters; *** significant at 1 percent, ** significant at 5 percent, and * significant at 10 percent. There are 64 observations with no sibling.
[a] Age dummies and location-fixed effects are included.

We see a positive but insignificant effect of prime-age sibling death on HIV infection (Table 1, column 1). From this estimation, we also capture age effects on HIV infection status. Figure 1 shows age-fixed effect estimates, demonstrating that HIV infection in the sample increases monotonically when agents are in their twenties and stays more or less constant when agents are in their thirties.

To capture potential age heterogeneity (Table 1, column 2), we include an indicator that takes the value of one if the respondent is age 26 or above, and interact this with the measure of prime-age sibling death. Interestingly, we see that sibling death significantly decreases the likelihood of HIV infection among young women (aged 25 or less), while it increases the likelihood of infection among older women (aged 26 or above). Preliminary analyses confirm that this age threshold at age 26 explains the heterogeneity; HIV infection among siblings has a greater effect on the behavioral choices made by the younger group compared to the older group. Sibling death significantly decreases on the likelihood of HIV infection among young women aged 26 or less.

Columns 3 and 4 of Table 1 split the sample at age 26. We find a clear contrast in the parameter estimates between the two groups. In the group of women aged 25 or less (Column 3), the effect is concave, showing a negative effect once the proportion of prime-age sibling death is large (above 0.1844). In contrast, the effect is convex among women aged 26 or above, showing a positive effect when the proportion of prime-age sibling death is above 0.1739.

As shown in Figure 1, the age of 26 appears to be a threshold in HIV infection behavior. HIV infection clearly increases up to the age of 26 and plateaus thereafter. This is consistent with our finding that social learning is important among women younger than 26. Our findings support the notion that when agents are young and potentially exposed to HIV infection risks, the value of information from sibling deaths is high and the behavioral reaction to sibling deaths becomes significant.

The above results support social learning, since we think social learning is important when women are young. Our results do not support the selection hypothesis. The fact that we could not control

for family-fixed effects (as well as individual-fixed effects) implies that the above estimates are at best an upper bound for the true effect.

Table 2 shows how education affects the sibling-death effect on HIV infection. In Column 1, we include an indicator that takes the value of one if the respondent completes higher than primary education, and is zero otherwise. We find that schooling has a negative but insignificant effect on HIV infection.

Table 2. Education effect on social learning

Sample: women of 15 to 39. Dependent: = 1 if HIV infection, and = 0 otherwise

Proportion of prime-age death among siblings	0.1134	0.1533**	-0.0564
	(1.61)	(2.00)	`(0.65)
Schooling completed higher than primary	-0.0489*	-0.0313	-0.0348
	(1.85)	(1.08)	(1.20)
Proportion of prime-age death among siblings * Schooling higher than primary		-0.3963**	-0.7025***
		(2.03)	(3.12)
Proportion of prime-age death among siblings * Age 26 or above			0.3036**
			(2.46)
Proportion of prime-age death among siblings * Schooling higher than primary * Age 26 or above			0.7088**
			(2.08)
No sibling dummy	0.0529	0.0541	0.0561
	(1.02)	(1.05)	(1.10)
Age dummies	Yes[a]	Yes	Yes
Location-fixed effects	Yes	Yes	Yes
Number of observations	2,428	2,428	2,428
R squared (within)	0.0594	0.0614	0.0679

Notes: Absolute t-values are in parentheses, using robust standard errors with location clusters; *** significant at 1 percent, ** significant at 5 percent, and * significant at 10 percent. There are 64 observations with no sibling.
[a] Age dummies and location-fixed effects are included.

In Column 2, we interact the education indicator with the proportion of prime-age deaths among siblings. Interestingly, prime-age sibling death decreases the likelihood of HIV infection among educated agents, while sibling death increases the likelihood of infection among uneducated agents. This is consistent with the hypothesis put forth by Schultz (1975) on the ability to deal with disequilibria. That is, the more educated can efficiently learn about HIV from their siblings who died in prime age.

We further interact the education and age-26-or-above indicators with the proportion of prime-age sibling deaths (Table 1, column 3), and find that sibling death decreases the likelihood of HIV infection among educated women. Similar to the findings shown in Table 1, we find that sibling death increases the likelihood of HIV infection among women aged 26 or above, while the education effect is weaker (insignificant) among older women.

As shown in Table 3, we next investigate reported condom use and extramarital affairs, as an additional measure of observable behavioral choice. Columns 1 and 2 show the results for condom use, which is positively associated with prime-age sibling death and educational attainment. In contrast to our above finding, the interaction of education and prime-age sibling death is not significant.

Columns 3 and 4 show that the death of prime-age siblings does not affect the likelihood of extramarital affairs, but education is positively related to the likelihood of affairs. However, education has a negative effect on the likelihood of extramarital affairs when the respondent observed the prime-age death of a sibling. This is consistent with our findings with regard to HIV infection status.

Table 3. The effects of prime-age death among siblings on HIV prevention

Sample: women of 15 to 39

	Condom use		Extramarital sex	
Proportion of prime-age death among siblings	0.1010**	0.1494***	0.0105	0.0542
	(2.01)	(2.84)	(0.21)	(1.03)
Schooling completed higher than primary		0.1148***		0.0853***
		(3.07)		(2.87)
Proportion of prime-age death among siblings * schooling higher than primary		-0.3941		-0.3741
		(1.46)		(1.63)
No sibling dummy	0.0562	0.0652	-0.0144	-0.0085
	(1.21)	(1.37)	(0.59)	(0.36)
Age dummies	Yes[a]	Yes	Yes	Yes
Location-fixed effects	Yes	Yes	Yes	Yes
Number of observations	1,934	1,934	2,428	2,428
R squared (within)	0.0525	0.0728	0.0228	0.0328

Notes: Absolute t-values are in parentheses, using robust standard errors with location clusters; *** significant at 1 percent, ** significant at 5 percent, and * significant at 10 percent. There are 64 observations with no sibling.
[a] Age dummies and location-fixed effects are included.

In Table 4, we show estimation results on HIV infection using an alternative definition of prime-age sibling death by restricting death cases in the 10-year period prior to the survey (1994-2004) to capture relatively recent sibling deaths.[7] This restriction is important because the current HIV infection status (dependent variable) implies relatively recent infection (otherwise, the respondent would have died in our study setting, where proper treatment was not available prior to the survey period). The results are similar to the above findings, and remain robust.

Table 4. The effects of prime-age death among siblings in previous 10 years on HIV infection

Sample: women of 15 to 39. Dependent: = 1 if HIV infection, and = 0 otherwise

Proportion of prime-age death among siblings	0.1251	-0.1805***	0.1215	0.1617	-0.0770*
	(1.60)	(2.11)	(1.56)	(1.89)	(0.85)
* Age 26 or above		0.4876***			
		(3.75)			
Schooling completed higher than primary			-0.0493*	-0.0345	-0.0394
			(1.86)	(1.18)	(1.35)
Proportion of prime-age death among siblings * Schooling higher than primary				-0.3529	-0.6593***
				(1.56)	(2.87)
Proportion of prime-age death among siblings * Age 26 or above					0.3709***
					(2.70)
Proportion of prime-age death among siblings * Schooling higher than primary * Age 26 or above					0.8134**
					(1.98)
No sibling dummy	0.0552	0.0589	0.0518	0.0527	0.0546
	(1.05)	(1.12)	(1.00)	(1.02)	(1.06)
Age dummies	Yes[a]	Yes	Yes	Yes	Yes
Location-fixed effects	Yes	Yes	Yes	Yes	Yes
Number of observations	2,428	2,428	2,428	2,428	2,428
R squared (within)	0.0569	0.0633	0.0593	0.0607	0.0688

Notes: Absolute t-values are in parentheses, using robust standard errors with location clusters; *** significant at 1 percent, ** significant at 5 percent, and * significant at 10 percent. There are 64 observations with no sibling.
[a] Age dummies and location-fixed effects are included.

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[7] We were able to define sibling death in the 5 years prior to the survey, but preliminary analysis revealed that this mortality rate is small enough to reduce the statistical power of our work.

4. CONCLUSION

We herein show that young women in Malawi are less likely to be HIV-infected if they observed prime-age deaths among their siblings, which supports the social learning hypothesis. However, HIV infection is found to be positively related to prime-age sibling deaths among older women. Our results indicate that young agents learn about the risks associated with HIV from the deaths of their siblings and change their behavior accordingly. The above findings are robust to age (cohort) effects and unobserved village-level factors.

Notably, schooling reinforces the social-learning effect of sibling deaths on HIV infection regardless of age cohorts, implying that education increases the efficiency of learning. In our analysis, education itself does not directly decrease HIV infection, but rather affects it through social learning effects related to sibling death. Therefore, our analysis indicates that education not only directly changes knowledge about HIV/AIDS through schooling, but also changes learning efficiency, thus helping agents alter their behavior to avoid HIV infection.

Our study also showed two observable implications consistent with stylized facts in developing countries. First, HIV infection increases at the initial stage, probably because of lack of knowledge and information on the nature of HIV/AIDS, but as the information accumulates, the infection rate starts decreasing. Second, at the initial phase of the AIDS epidemic, education, which is correlated with other socioeconomic characteristics, is positively associated with HIV infection. However, our results suggest that the effect of education on HIV infection becomes negative as the information accumulates and education helps agents decipher the optimal reaction to the risk of HIV infection. Whether the above findings from Malawi can also be confirmed in other populations is an interesting question, but beyond the scope of this paper.

APPENDIX: A SIMPLE MODEL

We herein describe a simple model used to relate new information created through death of siblings (reference group) to social learning. Notably, we can exclude direct transmission of HIV from the reference group. Agents learn from the successes and failures of different behavioral choices with regard to HIV infection. Death incidence in the reference group signals an action taken by the individual in the reference group. Agents learn from deaths in the reference group to optimize their behavior, as they cannot learn from their own experiences (experiments).

Assume that there are two types of actions, $x = a,b$. Action a is safe behavior, while action b is the risk-taking choice. The utility cost of action is $c(a) > c(b) > 0$. Both actions are observable.

The causal relationship from action to result is as follows. $\Pr(z = death|x = a) = p_1$, and $\Pr(z = death|x = b) = p_2$ where $p_1 < p_2$. However, agents do not know the probabilities. For simplicity, assume that $p_1 = 0$.[8] In case of death, agents have a negative utility, $u(death) \ll 0$. We also assume, for simplicity, that $u(alive) = 0$.

For agents, the question is whether p_1 is sufficiently lower than p_2 (gain is sufficiently large or not), given that $c(a) > c(b)$. In other words, risk-neutral agents learn about probability difference, $p_1 - p_2$. Assume that agent's prior on $E_0[p_1 - p_2] = 0$ at the initial stage, i.e., agents do not know the difference in the death probability that the two actions can make.

For simplicity, we ignore intertemporal preference and strategic interactions among agents. Thus, the description below is regarded as that of social learning from a previous generation.

Assume that agents have heterogeneity in taste, $v_j \sim F(v)$, that affects the agent's expected utility. The agent will choose action a if

$$E_0[p_1 - p_2]u(death) - (c(a) - c(b)) + v_j = -(c(a) - c(b)) + v_j > 0.$$

Thus, they choose a if $v_j > c(a) - c(b)$, and b otherwise. Those who have v_j below $c(a) - c(b)$ choose action b, and some of them die with probability p_2. If nobody chooses b (e.g., the lower bound for v_j is above $c(a) - c(b)$, and nobody dies of AIDS), then it is impossible for an agent to learn of p_2 and the probability difference, $p_1 - p_2$.

At the second stage, agents form the unbiased estimate, $\hat{p}_1 - \hat{p}_2$, by

$$\hat{p}_1 = \frac{1}{N_a} \sum_i I(z_i = death \,|\, x_i = a)$$

$$\hat{p}_2 = \frac{1}{N_b} \sum_i I(z_i = death \,|\, x_i = b)$$

where $E[\hat{p}_k] = p_k$ and $Var(\hat{p}_k) = \frac{1}{N_k} p_k(1 - p_k)$, $k = 1, 2$. Under the assumption that $p_1 = 0$, we have $Var(\hat{p}_1) = 0$.[9]

In the second stage, agents can estimate $p_1 - p_2$ to form the expected utility. They will choose action a if

$$[p_1 - \hat{p}_2]u(death) + v_j > c(a) - c(b),$$

where the first term on the left-hand side is strictly positive since $u(death) \ll 0$ and $p_1 = 0$. New information created through deaths will tend to make agents choose the safe action a under the reasonable range of v_j. If there is no death, agents cannot estimate p_2 (and $p_1 - p_2$), and therefore some of agents will choose action b.

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[8]We can relax this assumption, but the main result remains robust.
[9]This assumption can be relaxed.

An implication from the above model is that the mortality rate increases at the initial stage, but decreases thereafter if social learning works at the second stage. The crucial assumption in the above story is the observability of action: agents can learn of the causality from action choice to the consequence of HIV/AIDS.

Table A.1. Number of siblings and age in the study population

	Frequency	Percent	Cumulative
Number of siblings			
0	64	2.64	2.64
1	95	3.91	6.55
2	158	6.51	13.06
3	225	9.27	22.32
4	294	12.11	34.43
5	377	15.53	49.96
6	364	14.99	64.95
7	324	13.34	78.29
8	199	8.20	86.49
9	168	6.92	93.41
10	82	3.38	96.79
11	46	1.89	98.68
12	23	0.95	99.63
13	5	0.21	99.84
15	1	0.04	99.88
16	1	0.04	99.92
18	2	0.08	100.00
Age			
15	96	3.95	3.95
16	120	4.94	8.90
17	81	3.34	12.23
18	123	5.07	17.30
19	125	5.15	22.45
20	156	6.43	28.87
21	119	4.90	33.77
22	182	7.50	41.27
23	113	4.65	45.92
24	121	4.98	50.91
25	131	5.40	56.30
26	102	4.20	60.50
27	103	4.24	64.74
28	108	4.45	69.19
29	83	3.42	72.61
30	103	4.24	76.85
31	61	2.51	79.37
32	77	3.17	82.54
33	68	2.80	85.34
34	79	3.25	88.59
35	74	3.05	91.64
36	62	2.55	94.19
37	38	1.57	95.76
38	55	2.27	98.02
39	48	1.98	100.00
Total	2,428	100.00	

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