ELSEVIER

The link between public and private insurance and HIV-related mortality

Jayanta Bhattacharya [a], Dana Goldman [b,*], Neeraj Sood [b]

[a] *Center for Primary Care and Outcomes Research, Stanford Medical School, 117 Encina Commons, Stanford, CA 94305-6019, USA*
[b] *RAND, 1700 Main Street, Santa Monica, CA 90401, USA*

Abstract

As policymakers consider expanding insurance coverage for the human immunodeficiency virus (HIV+) population, it is useful to ask whether insurance has any effect on health outcomes, and, if so, whether public insurance is as efficacious as private insurance in preventing premature death. Using data from a nationally representative cohort of HIV-infected persons receiving regular medical care, we estimate the impact of different types of insurance on mortality in this population.

Our main findings are that (1) ignoring observed and unobserved health status misleads one to conclude that insurance may not be protective for HIV patients, (2) after accounting for observed and unobserved heterogeneity, insurance does protect against premature death, and (3) private insurance is more effective than public insurance. The better performance of private insurance can be explained in part by more restrictive Medicaid prescription drug policies that limit access to highly efficacious treatment.
© 2003 Elsevier B.V. All rights reserved.

1. Introduction

Most economic models treat health insurance as a hedge against financial risk from illness. But the benefits from health insurance are not limited to the avoidance of financial risk; additional benefits are derived from insurance's ability to make available medical care that would otherwise be unaffordable (Nyman, 1999). In fact, the public health community

---

prominently cites the unaffordability of health care and consequent poor health outcomes as the primary reason for expanding health insurance coverage (Institute of Medicine, 2002).

This view has motivated many investigations of the link between health insurance and health. In their review of this literature, Levy and Meltzer (2001) found that most of these studies were flawed. There was no causal link between having health insurance and better health—rather, there were associations. Exceptional studies that use randomization or quasi-randomization to address causation have found either no or limited effects on health, depending on the population under investigation and the health measure used. One problem with asking the broader question of how insurance affects health is that it may just be too ambitious. The impact of insurance on health will obviously depend on circumstances; it might be more useful to ask what population can most benefit from insurance expansions (either public or private), and how this relationship changes with medical treatment.

The treatment of human immunodeficiency virus (HIV) provides a useful case study. In 1995, the Food and Drug Administration approved the first protease inhibitor for treatment of HIV. When new viral particles break off from an infected cell, protease cuts long protein strands into the parts needed to assemble a mature virus. These drugs block the protease enzyme, and hence prevent new viral particles from maturing. Clinical trials and observational data have confirmed their clinical usefulness in combination with older drugs (so-called highly active anti-retroviral therapy or HAART) in forestalling HIV-related mortality (Palella et al., 1998). After the introduction of protease inhibitors into clinical practice, age-adjusted death rates from HIV infection fell 25% from 1995 to 1996 and 47% from 1996 to 1997 (Center for Disease Control, 1998).[1]

HAART is expensive, costing on average about US$ 13,000 per year. Thus, it is possible that health outcomes for HIV-infected persons will be very responsive to the availability of insurance. Indeed, in previous work, we found that insurance significantly reduced mortality in HIV+ patients (Goldman et al., 2001). These results suggest that policies to expand insurance coverage to the uninsured HIV+ population could save many lives. That paper treats insurance as a single category for computational ease. However, HIV+ patients can be covered either through private insurance or public insurance (typically Medicaid). Since these insurance types differ in generosity we would expect that they differ in the quality and intensity of treatment.

Several studies have examined the relationship between insurance type and service utilization among HIV+ patients (Fleishman et al., 1994; Horner et al., 1996; Joyce et al., 1998; Shapiro et al., 1999). Most of these studies find that the uninsured or publicly insured HIV+ patients incur lower per diem charges, receive fewer procedures and are less likely to receive expensive drug therapy than privately insured HIV+ patients with similar medical conditions. In addition, a majority of HIV patients covered by public insurance are required to demonstrate a disability to qualify for coverage. Thus, most patients with public insurance obtain coverage only in the advanced stage of disease. These differences in the intensity of treatment and initiation of coverage among publicly and privately insured

---

[1] HAART is a combination therapy involving three types of drugs: nucleoside reverse transcriptase inhibitors, protease inhibitors, and non-nucleoside reverse transcriptase inhibitors. All regimens require at least three drugs, and the vast majority involve at least one protease inhibitor.

HIV+ patients leads naturally to questions about how these insurance types differ in their efficacy in preventing premature HIV related deaths.

In this paper, we identify the causal effects of different types of insurance on mortality by analyzing observational data on a nationally representative sample of adult HIV+ patients receiving care in the US. Measuring the efficacy of insurance in observational data is complicated by the fact that insurance decisions might be correlated with unobserved differences in the health status of patients. To account for this endogenity of insurance choices, we jointly estimate insurance decisions and mortality and allow for arbitrary correlation between insurance and mortality in a parametric setting. We identify the 'true' effect of insurance on mortality using state-policy variables that affect the ease with which patients' can obtain public insurance and a state-level variable for firm size that affects the likelihood of obtaining employer provided insurance. These instruments are clearly related to patient insurance status, but should not directly affect death rates (except through insurance).

The next section describes the data used for this analysis. We then present our joint model of insurance and mortality and provide the details of our identification strategy. Following that, we present results from a reduced form model that treats insurance as exogenous and compare them to the results from our structural model. The final sections discuss the conclusions and limitations of our analysis.

2. Data

We use data from a nationally representative study of HIV+ patients in care—the HIV Costs and Services Utilization Study (HCSUS). The HCSUS employed a multi-stage national probability sample design to identify HIV+ patients over 18 years old, who made at least one visit for regular care in the 48 contiguous states in January or February 1996. It does not include HIV+ patients whose only contact with the health care system was through military, prison, or emergency department facilities, or those who have not received any treatment for HIV. The HCSUS collected three rounds of interviews: baseline, a first follow-up and a second follow-up with a baseline response rate of 68% (Duan et al., 1999). The first follow-up interview was conducted with 2466 subjects between December 1996 and July 1997; and the second follow-up was conducted with 2267 subjects between August 1997 and January 1998. Most of the attrition between waves is due to mortality. Towards the end of data collection for the baseline survey, HAART entered clinical practice and disseminated widely as the first follow-up survey was in the field. Thus, we use the first follow-up sample of 2466 subjects for our analysis.

We use analytic weights to adjust the sample to the reference population. A respondent's analytic weight, which may be interpreted as the number of people in the population represented by that respondent, is the product of three patient-specific quantities—the sampling weight, the multiplicity weight, and the non-response weight. The sampling weight adjusts for over-sampling (of women, for example); the multiplicity weight adjusts for patients who could potentially enter the sample via multiple providers; and the non-response weight adjusts for differential cooperation (Duan et al., 1999). All analyses presented in this paper use these weights.

Table 1
Insurance, use of HAART, and AIDS

Insurance status	Sample (%)	HAART by first follow-up (%)	AIDS (%)
None	18	32	21
Public	52	34	53
Medicaid only	28	33	47
Medicaid and medicare	16	33	58
Medicaid and private	2	42	57
Medicare only	4	43	64
Medicare and private	2	53	62
Private (only)	30	50	34

Source: HCSUS first follow-up ($N = 2466$).

Health is measured using mortality 1 year after the interview date. It is based on data collected by HCSUS interviewers in the field, as well as from Equifax Inc., a credit company that also tracks deaths in the United States. The key predictors are insurance type. Table 1 shows the proportion of respondents on HAART by sources of insurance coverage. The majority of HIV+ patients obtain coverage through either private insurance or Medicaid −30% of the respondents are covered by private insurance only, 28% are covered by Medicaid only, 16% are covered by both Medicaid and Medicare, and 18% are uninsured. However, there are sharp differences in the use of HAART by source of insurance coverage. HIV patients with private insurance are more likely to be on HAART than patients with any public insurance coverage, despite the higher prevalence of AIDS (more advanced disease) among patients with public insurance. To account for differential use of HAART, we consider the effect of three types of insurance—private insurance only, (any) public insurance, no insurance.[2]

3. Empirical model of insurance and mortality

Let m_i^* represent an index function that measures the mortality propensity for HIV+ patient i 1 year after interview. We model this mortality propensity as a function of the insurance status of the individual, observed covariates, and unobserved error:

$$m_i^* = c_1 + \gamma_1 \times \text{private}_i + \gamma_2 \times \text{public}_i + \beta_1' X_i + \rho_{m,i} - \varepsilon_i \tag{1}$$

The vector X_i represents observed exogenous covariates that determine mortality propensity, such as age, gender, and education. Mortality is also affected by insurance status, where *private_i* represents whether the patient was covered by private insurance (only), and *public_i* represents whether the patient was covered by (any) public insurance. We model mortality as function of insurance rather than medical care since insurance is often the lever for

---

[2] We check the sensitivity of our results in an alternate specification where we exclude patients with Medicaid and private insurance, Medicare and private insurance, and Medicare only from the public insurance category. Patients in these insurance categories had higher use of HAART than others in this category.

increasing access to medical care. In addition, there are theoretical reasons (moral hazard) to believe that insurance might increase use of "non-essential" medical care only and thus have no effect on health.

Mortality is also assumed to depend on an unobservable heterogeneity component, $\rho_{m,i}$ that will also relate to insurance choices. It is useful to think of it as unobserved health status or attitudes towards risk, and it is assumed to be orthogonal to the covariates X_i. There is also a random error $\varepsilon_{m,i}$ that is uncorrelated with X_i and insurance choice. We want to consistently estimate the parameters c_1, β_1, γ_1 and γ_2, after accounting for the endogeneity of insurance status. We define m_i as an indicator variable that represents whether patient i actually died 1 year after interview:

$$m_i = \begin{cases} 1 & \text{if } m_i^* > 0 \\ 0 & \text{if } m_i^* \leq 0 \end{cases} \tag{2}$$

We assume that $\varepsilon_{m,i}$ are i.i.d standard normal errors, with zero mean and unit variance. This distributional assumption implies a probit model for m_i, where the probability of death, conditional on observed characteristics $\{private_i, public_i, X_i\}$ and unobserved characteristics $\rho_{m,i}$, is:

$$P[m_i = 1 | private_i, public_i, X_i, \rho_{m,i}] = \Phi(c_i + \gamma_1 private_i + \gamma_2 public_i + \beta_1' X_i + \rho_{m,i}) \tag{3}$$

Here, $\Phi(\cdot)$ is the cumulative distribution function for the standard normal distribution. Patients choose among insurance types $j \in \{private, public, uninsured\}$ on the basis of a standard random indirect utility function:

$$V_{j,i}^* = c_j + \beta_j' Z_i + \rho_{j,i} + \varepsilon_{j,i} \tag{4}$$

The vector Z_i represents variables that determine insurance status including our set of instrumental variables (IV, that is, variables that belong in the insurance equation, but not in the mortality equation); and $\rho_{j,i}$ is a patient-specific random intercept that reflects the patients' propensity for insurance status j that is unobserved by the researcher. Again, it could be due to unobserved disease severity or preferences for risk. The parameters c_j and β_j are additional parameters to be estimated; and $\varepsilon_{j,i}$ represents the orthogonal error term.

Patients choose the insurance status that maximizes their indirect utility. We assume that $\varepsilon_{j,i}$ are independently and identically distributed according to the Type I extreme value distribution. This distributional assumption and normalizing $\{c_{uninsured}, \beta_{uninsured}, \rho_{uninsured}\}$ to zero yields a multinomial logit model for insurance choice.

$$P[private_i = 1 | Z_i, \rho_{private,i}, \rho_{public,i}] = \frac{\exp(c_{private} + \beta_{private}' Z_i + \rho_{private,i})}{1 + \sum_{j \neq uninsured} \exp(c_j + \beta_j' Z_i + \rho_{j,i})} \tag{5}$$

$$P[public_i = 1 | Z_i, \rho_{private,i}, \rho_{public,i}] = \frac{\exp(c_{public} + \beta_{public}' Z_i + \rho_{public,i})}{1 + \sum_{j \neq uninsured} \exp(c_j + \beta_j' Z_i + \rho_{j,i})} \tag{6}$$

To complete the model and allow for correlation between mortality and insurance choices, we need to assume a joint distribution for the unobserved heterogeneity vector $\rho =$

(ρ_m, $\rho_{private}$, ρ_{public}). Our approach is semi-parametric. We allow the unobserved heterogeneity in each equation to take one of three values—intuitively, there are three types of people that occur with probabilities p_1, p_2, and $1 - p_1 - p_2$. The effect of being a certain type has different effects on each outcome: (ρ_m^1, ρ_m^2, ρ_m^3) for mortality, ($\rho_{private}^1$, $\rho_{private}^2$, $\rho_{private}^3$) for private insurance, and (ρ_{public}^1, ρ_{public}^2, ρ_{public}^3) for public insurance. For example, there is a p_1 probability that a person will be of the first type, which would imply realizations of ρ_m^1 for the propensity to die, ρ_{public}^1 for the propensity to have public insurance, and $\rho_{private}^1$ for the propensity to privately insure.

This distributional approach has several advantages over specifying a continuous parametric density for the unobserved heterogeneity vector. First, an incorrect specification of the parametric density function will lead to inconsistent parameter estimates. The discrete factor density allows us to approximate any underlying distribution of heterogeneity. In fact, Monte Carlo studies show that discrete factor distributions with two to four points of support adequately model many distributions (Heckman, 2001; Mroz, 1999). Second, discrete factor models are computationally simpler than parametric models as they avoid multiple numerical integration in the construction of the likelihood function. Finally, these models have been successfully used in the health economics literature. Cutler (1995) used a similar model to study the effects of prospective payment schemes on mortality and other adverse medical outcomes, and Goldman (1995) used a similar model to study the effect of managed care on costs. The evidence from these and other studies suggest that discrete factor models effectively recover the parameters of the underlying structural models.

Since the mortality, public insurance, and private insurance equations have intercept terms we normalize the mean of each heterogeneity component to be zero. This implies that the third point of support in each equation is not "free".[3] Thus, we need to estimate eight additional parameters: two points of support in the mortality equation, two points of support in the private insurance equation, two points of support in the public equation, and two probabilities. The resulting variance–covariance matrix for the unobserved heterogeneity is given in Appendix A. This model not only allows non-zero covariance across mortality and insurance propensities but also allows non-zero covariance between private and public insurance propensities. Thus, our model relaxes the independence of irrelevant alternatives assumption of standard multinomial logit models and allows a completely general variance–covariance matrix.

We use maximum likelihood to estimate the parameters of our model; more details are given in Appendix A. Because it is difficult to interpret the magnitude of the parameter estimates directly, we also report the relative impact of private and public insurance on average mortality as shown in Eqs. (A.7) and (A.8) in Appendix A. They are reported as the percentage reduction in mortality associated with private and public insurance relative to the uninsured case.

---

[3] For example, the third point of support (ρ_m^3) in the mortality equation can be written as a function of the other two points of support and the probabilities of each type, as follows:

$$E(\rho_m) = 0$$
$$\Rightarrow p_1\rho_m^1 + p_2\rho_m^2 + (1 - p_1 - p_2)\rho_m^3 = 0$$
$$\Rightarrow p_1\rho_m^1 + p_2\rho_m^2 + (1 - p_1 - p_2)\rho_m^3 = 0$$

4. Identification

We use state Medicaid policies and average firm size as our instrumental variables to explain insurance status but not mortality. Medicaid is the most common form of insurance for the HIV+ population in care, covering 46% of the population. HIV+ patients can qualify for Medicaid through three distinct pathways.

First, patients who meet the state's income eligibility and family composition requirements for Aid to Families with Dependent Children (AFDC) as they existed on 16 July 1996 qualify for Medicaid coverage. Second, Supplemental Security Income (SSI) beneficiaries are automatically eligible for Medicaid in 38 states. The other states have different standards for eligibility either as a 209(b) state or a waiver state. Section 209(b) of the Social Security Amendments Act of 1972 allows States to include more restrictive definitions of "disability" and lower income and assets standards for Medicaid eligibility. Medicaid eligibility is also sometimes available through a "medically-needy" program for individuals who meet Medicaid's disability criteria but have incomes that exceed the financial eligibility limit. The program allows individuals to "spend down" to Medicaid eligibility by deducting medical-related expenses from their reported income. Only some states have a "medically-needy" program, and they vary in their income eligibility levels. Thus, we define the following four variables based on Medicaid eligibility rules for the state in which the patient resides:

- *Medically-needy threshold*: This is the state's income eligibility threshold for the medically-needy program expressed as a percentage of the federal poverty line (FPL).[4]
- *AFDC threshold*: This is the State's income eligibility threshold in 1996 for AFDC, expressed as a percentage of the federal poverty line.
- *SSI < 65% FPL*: This is an indicator variable for whether the state's income eligibility threshold for Medicaid through the "SSI" category was at least 10% points lower than the federal guideline of 75% of the federal poverty line.[5]
- *Average firm size*: This is the average firm size in the state in which the patient was sampled. Several studies have documented the strong positive association between firm size and employer provided insurance offers (Bundorf, 2002; Cantor et al., 1995; Employee Benefit Research Institute, 2001).

Distributional assumptions aside, as in all instrumental variable-based studies, the credibility of our study rests on the believability of our instruments. Our state policy and average firm size instruments could fail in at least two ways. First, the estimators perform poorly if the instruments are only weakly correlated with the treatment variable—in this case, insurance status (Nelson and Startz, 1990; Bound et al., 1995; Staiger and Stock, 1997). Thus, we report Wald statistics for the joint significance of our instruments in predicting insurance status. Second, our instruments might be correlated with unobserved determinants of mortality (like unmeasured health status). The assumption that an instrumental variable is uncorrelated with the outcome measure cannot be directly tested, and some

---

[4] States which did not institute a medically-needy program were coded as having an income threshold of 0%.

[5] We coded this as a dummy variable as there were only a four states that implemented a significantly more restrictive eligibility standard than the federal guideline.

researchers have argued that IV estimates should be viewed with caution (Bound et al., 1995).

In our application, it seems clear that patients have little direct influence at an individual level on state policies or firm size, so our state-level instruments seem valid. This argument is not enough to establish exogeneity; if there are unobserved state-level variables that determine both health and insurance status, state policies would be endogenous despite the lack of control by patients over these policies. To address this issue, we develop some indirect evidence that our instruments are not simply picking up differences in unobserved health across states. If the latter hypothesis were true, then one would expect to find that our state-level instruments predict the mortality of patients even in a non-HIV population.

To check this assumption, we estimate a logit model of 1-year mortality using data from the Medicare Current Beneficiary Survey (MCBS). On the right-hand side, this mortality model includes a sparse set of health status indicators, such as measures of activities of daily living (ADLs) and a general health index, and our state-policy instruments. Since this elderly or disabled population is by definition insured by Medicare, our instruments should not predict their mortality unless they proxy for unobserved state-level effects. Table 2 reports the regression results and shows that our instruments are not statistically significant in the model, with odds ratios near one. Of course, these results do not prove that unobserved state effects are unimportant in the HCSUS population, but they are certainly suggestive.

Table 2
Medicare beneficiary 1-year mortality—logit regression

Variables	Odds ratio	95% Confidence interval	
		Lower limit	Upper limit
State instruments			
Medically-needy threshold	1.001	0.997	1.004
AFDC threshold	1.001	0.997	1.003
SSI threshold <65% of FPL	1.140	0.816	1.592
Average firm size	0.985	0.924	1.050
Number of ADL limitations			
No limitation (Ref. Cat.)	–	–	–
1. ADL	2.375	1.803	3.129
2. ADL	2.503	1.819	3.443
3. ADL	3.296	2.307	4.723
4. ADL	3.376	2.331	4.889
5. ADL	5.857	4.347	7.893
6. ADL	7.589	5.786	9.954
Self reported health status			
Excellent (Ref. Cat.)	–	–	–
Very good	1.625	1.042	2.536
Good	1.839	1.205	2.805
Fair	2.415	1.574	3.703
Poor	4.283	2.7726	6.618

Source: Medicare Current Beneficiary Survey, 1996 ($N = 9965$). Excludes states not included in the HCSUS sample.

There is no good reason to expect that such effects should be present for HIV+ patients when they are not present for the elderly or disabled.

5. Results

Descriptive means (by insurance type) for all model variables are given in Table 3. Most of the variables are self-explanatory. In some models, we include measures of the lowest ever CD4+ T-lymphocyte cell count, a critical measure of the function of a patient's immune system. A depletion in these cells correlates strongly with the worsening of HIV disease and the risk of death (Fauci et al., 1998). We categorize CD4+ counts into four categories. Patients with CD4+ lymphocyte counts below 50 have a very poor prognosis in general; while those with counts above 500 are considered much healthier. Table 3 shows that patients with insurance are more likely to be in poor health (lower CD4+ cell counts) and to have

Table 3
Descriptive statistics by insurance status

Variable	Private ($N = 718$)	Public ($N = 1295$)	Uninsured ($N = 453$)
Demographics			
Age (years)	39	39	37
Non-white (%)	29	61	58
Female (%)	10	30	23
Education			
Less than HS degree (%)	5	35	27
High school degree (%)	20	31	31
Some college or more (%)	75	34	42
Years since diagnosis			
Less than 1 year (%)	6	8	18
1–2 years (%)	12	11	15
2–3 years (%)	22	23	25
3–4 years (%)	26	30	24
4–5 years (%)	21	19	11
>5 years (%)	13	9	7
State instruments			
Medically-needy threshold	54% of FPL	50% of FPL	42% of FPL
AFDC threshold	182% of FPL	180% of FPL	181% of FPL
SSI threshold <65% of FPL	9%	7%	6%
Average firm size	149 workers	146 workers	146 workers
Lowest ever CD4 count (cells/μl)			
>500 (%)	9	6	11
200–499 (%)	40	31	52
50–200 (%)	28	34	27
0–50 (%)	23	29	10
HAART by first follow-up (%)	50	34	32
Deaths per 1000 patient-years	21	59	21

Note: FPL, federal poverty line; *source*: HCSUS first follow-up survey ($N = 2466$).

Table 4
Results for the naive model

Coefficient	No severity controls	With severity controls
Insurance		
Private	−0.0442	−0.3071
Public	0.4521[a]	0.1389
No insurance (Ref. Cat.)	–	–
Demographics		
Age	0.0139	−0.0098
Age^2	−0.0001	0.0003
Non-white	−0.1327	−0.1520
Female	0.0516[b]	0.0154
Education		
High school degree	−0.0134	−0.0075
Some college or more	0.0326	−0.0568
Less than HS degree (Ref. Cat.)	–	–
Years since diagnosis		
Less than 1 year	0.1836	0.0633
1–2 years	0.2441	0.3820
2–3 years	−0.0200	−0.0152
3–4 years	0.3746[a]	0.4657[a]
4–5 years	0.0761	0.1171
>5 years (Ref. Cat.)	–	–
Lowest ever CD4 count (cells/μl)		
>500	–	0.9305[a]
200–499	–	1.3007[a]
50–200	–	0.7420[a]
0–50 (Ref. Cat.)	–	–
Constant	2.5688[a]	−1.3810

Note: Estimates are from a single equation (weighted) probit of 1-year mortality with and without severity controls ($N = 2466$).

[a] Statistically significant at 95% confidence level.
[b] Statistically significant at 90% confidence level.

received HAART as compared to uninsured patients. There are also important differences between those with private and public insurance—patients with private insurance are more educated, healthier, more likely to receive HAART and are predominantly white males.

To illustrate the consequences of selection bias, we estimate a "naïve" probit model where insurance status is treated as an exogenous variable. To demonstrate the importance of including information on health status, we present in Table 4 two sets of estimates; one set with controls for disease progression, particularly CD4 cell count, and one without.

In the regression without severity controls, public insurance is associated with an increased probability of 1-year mortality, and the effect is statistically and substantively significant. This finding persists even after including severity controls, although the effect is no longer significant. This result is analogous to that of Lancaster and Intrator (1998) who also find the perverse result that health insurance increases the risk of death for HIV+ patients.

Table 5
Relative impact of insurance on mortality

	Insurance status	
	Private	Public
Unadjusted raw means (%)	0	+65
Naïve model (%)		
No severity controls	−9	+168
With severity controls	−46	+28
Structural model (%)		
No treatment controls	−79	−66
With treatment controls	−50	+8

Note: Numbers reported are percentage change in mortality (deaths per person-year) relative to no insurance case (RI) for each empirical model (Eqs. (A.7) and (A.8)).

In both the regression models, private insurance is associated with a decreased probability of death, although the effect size is statistically insignificant in both models.

Table 5 summarizes the results from the "naive" model in terms of relative mortality.[6] In general, the use of severity controls reduces the mortality effect of both the public and private insurance substantially. For instance, the results without severity controls indicate that public insurance increases relative mortality by 168%; including health status measures like CD4 cell count reduces this effect to 28%, but it does not disappear. Similar results obtain for the private insurance effect—private insurance decreases relative mortality by 9% without severity controls and by 46% with severity controls. We attribute these findings to a spurious correlation between severity of illness and insurance for HIV patients. The correlation between public insurance and severity of illness seems to be especially strong. One possible explanation is that the eligibility rules for Medicaid or Medicare require HIV+ patients to demonstrate a "disability"—almost always associated with advanced disease.[7] These findings help motivate our structural approach.

The parameter estimates for the structural model are shown in Table 6, along with the correlation between insurance status and mortality, and the correlation between public and private insurance. We also report the joint significance of the excluded instruments in Eq. (4) using a Wald statistic, since these are considered a test of the small sample bias associated with the IV estimator (Staiger and Stock, 1997).

The parameter estimates from the insurance equations show that the state-level instruments are highly correlated with both private and public insurance choice and are jointly significant ($P < 0.01$). As expected, we find that higher average firm size is associated with higher likelihood of having private insurance. We also find that less generous Medicaid

---

[6] We also report results from the structural model; these are discussed later.

[7] We also estimated an instrumental variables (IV) model that linearizes the mortality and insurance equations. However, the IV model yielded completely implausible estimates for virtually all insurance classes. First, the estimated 1-year probabilities were negative (−0.041) for those with public insurance . Also, the probability of death conditional on private insurance increased implausibly (from an unadjusted mean of 0.021–0.101); if anything, one would expect private insurance to provide protection at least equivalent to public insurance because of its generous drug coverage. It was this instability that motivated our discrete factor approach.

Table 6
Estimates from structural model

Parameters	Mortality	Private insurance	Public insurance
Private	6.7153[a,b]	–	–
Public	5.2764[a,b]	–	–
No insurance (Ref. Cat.)	–	–	–
Demographics			
Age	−0.2059	0.0932[c]	−0.0143
Age^2	0.0032	−0.0007	0.0007
Non-white	−0.5536	0.9247[a]	0.0508
Female	0.3652	−0.0141	0.7064[a]
Education			
High school degree	0.4813	0.8768[a]	−0.2089
Some college or more	−0.5750	1.5504[a]	0.6675[a]
Less than high school (Ref. Cat.)	–		
Years since diagnosis			
Less than 1 year	−1.9967[c]	1.0090[a]	1.4389[a]
1–2 years	−0.1727	−0.0787	0.5448[a]
2–3 years	−0.9995	−0.1152	−0.4162
3–4 years	−0.1966	0.0215	−0.0699
4–5 years	−0.2927	0.2848	0.2300
>5 years (Ref. Cat.)	–	–	–
Lowest ever CD4 count			
>500	4.5941[a]	0.8453[a]	1.8698[a]
200–499	4.8722[a]	1.1291[a]	1.8803[a]
50–200	3.5580[a]	1.0054[a]	1.1391[a]
0–50 (Ref. Cat.)	–	–	–
State-level instruments			
SSI threshold < 65		−0.1179	−0.1526
AFDC threshold		0.0107[c]	0.0142[a]
Medically-needy threshold	–	0.0167[a]	0.0166[a]
Average firm size		0.1494[a]	0.0461
Constant	−0.6411	−2.7945	3.6412
Wald test (of instruments)	–	41.83[a]	38.08[a]
Correlations			
Corr (ρ_m, $\rho_{private}$)	0.325		
Corr (ρ_m, ρ_{public})	0.892		
Corr (ρ_{public}, $\rho_{private}$)	0.717		

Note: Estimates are maximum likelihood estimates of Eq. (A.6) ($N = 2466$). Correlations are computed using Eqs. (A.2)–(A.4).

   [a] Statistically significant at 95% confidence level.

   [b] Coefficient on private insurance is not equal to coefficient on public insurance at 95% confidence level.

   [c] Statistically significant at 90% confidence level.

eligibility rules are associated with lower likelihood of both public and private insurance coverage (relative to no insurance). Therefore, these results suggest that as eligibility for public insurance becomes stricter, HIV+ patients are more likely to transition to being uninsured rather than privately insured, thus lowering the likelihood of private insurance

relative to no insurance. This seems reasonable as HIV+ patients are often too frail to work and therefore might have problems finding employment and hence private insurance. Given that these patients suffer from a high cost disease, they are virtually excluded from the individual health insurance market. In other words, our results suggest that the crowd-out effect of public insurance (Cutler and Gruber (1996)) is mitigated for the HIV+ population due to their poor health and lack of employment opportunities. Finally, and contrary to our expectation, the results indicate that a higher AFDC threshold is associated with a lower likelihood of public insurance. Multi-collinearity between the three Medicaid policy variables, which reduces the power of our estimates, seems to be the most likely explanation for this result (the correlation between the AFDC and the medically-needy threshold is 0.62). In an alternate specification in which we excluded other Medicaid policy variables, we find that AFDC threshold has the expected sign and a higher AFDC threshold is associated with greater likelihood of public insurance.

The second column of Table 6 shows the parameter estimates for the mortality equation. In contrast to the naive model, we see that both private and public insurance decrease the likelihood of death. This reversal in the effects of private and public insurance on mortality can be explained by the positive correlation between unobserved health status and insurance choice. For instance, our results show that unobserved mortality propensity and public insurance propensity are positively correlated (correlation coefficient 0.89). Similarly, we also find positive correlation between unobserved mortality propensity and private insurance propensity, although the degree of correlation is much weaker than that for public insurance. It is also interesting to note that private and public insurance propensities are positively correlated. This might be explained by unobserved characteristics such as risk aversion and poor health that increase the propensity for insurance, in general.

These parameter estimates yield relative reductions in mortality as shown in Table 5. Overall, we find large and significant mortality benefits from private and public insurance. The effect of private insurance (79%) is larger than for public insurance (66%), and this difference is statistically significant.

Our model looks at the causal effects of insurance on mortality both because the empirical literature has been unable to identify instances where insurance matters for health and because insurance expansions for HIV+ patients are of keen interest to policy makers. Our underlying hypothesis, however, is that much of the explanation for the structural benefit of therapy is due to better access to highly active anti-retroviral therapy. The descriptive statistics in Table 3 suggest such a link; those in private and public insurance are more likely to receive HAART and, as our model demonstrates, much less likely to die.

To further investigate this issue, we estimated an alternate model in which we excluded patients with Medicare and private insurance; Medicaid and private insurance; and Medicare only from the public insurance categories. Since these patients had higher HAART use than others on public insurance, we would expect that excluding them would mitigate the benefits of public insurance. Our results confirm this hypothesis—the relative impact of private insurance on mortality increases marginally to 80% and the relative impact of public insurance on mortality decreases to 63%.[8]

---

[8] The full results from this model are available from the authors upon request.

If access to treatment is what is explaining mortality benefits, then controlling for treatment should compress the differences between insurance groups. Thus, we also estimated a version of the structural model that included proxies for treatment on the right-hand side. The model specification was the same as outlined in Section 3, with the exception that Eq. (1) (the mortality equation) also included: (1) binary indicators for use of a protease inhibitor and an anti-retroviral such as AZT at the time of the first follow-up interview; and (2) binary indicators for whether the person had ever been on monotherapy, polytherapy, or HAART as of December 1996. The latter variables were constructed using the definitions from Andersen et al. (1999).[9] After controlling for these factors, the mortality gradient compresses substantially between insurance categories as shown in Table 5. There is no significant difference between public and private mortality rates. Private coverage still has a significant benefit, but improvement in mortality is less (50% versus 79%). Thus, it appears that HAART may explain about 40% of the benefit for private insurance and all of the benefit for public insurance. It also suggests that private insurance confers benefits other than through receipt of medication—perhaps through more effective or more sustained HAART regimens, better care for opportunistic infections, or better disease management. Of course, there is concern that HAART itself may be endogenous, since receipt of therapy almost certainly reflects disease severity and ability to adhere to the complicated regimen (Goldman and Smith, 2002). As such, a fuller investigation of this issue is left to further research.

6. Conclusion

For HIV+ patients, we show that insurance reduces mortality rates significantly, but only after one adjusts for observed and unobserved heterogeneity. The discovery of effective highly active anti-retroviral therapy supports the conclusion that this a causal relationship, beyond the fact that we have valid instruments for insurance status. By July 1997, approximately 75% of the HCSUS sample had used a protease inhibitor or non-nucleoside reverse transcriptase inhibitor (Shapiro et al., 1999). This rapid proliferation in clinical practice is remarkable given the approval of the first protease inhibitor was only 2 years earlier, and the expense associated with these new drugs.

Since HAART is a key mediator explaining our casual relationship between insurance and mortality (especially for public insurance), it is reasonable to ask why not look at HAART directly? We look at insurance because it an obvious policy lever available for affecting HIV care, as there have been many suggestions to expand Medicaid eligibility for patients with HIV who do not meet traditional eligibility rules. In addition, our research contributes to the broader question about whether insurance has any impact on health outcomes. While the association is clear, studies with clear causation are limited. We show, at least in a context where effective therapies are available but very expensive, that insurance can save lives.

---

[9] HAART was defined as using a protease inhibitor, a non-nucleoside reverse transcriptase inhibitor, or a nucleoside reverse transcriptase inhibitor in various combinations. For example, HAART includes two or more NRTIs in combination with at least one PI or one NNRTI; and one NRTI in combination with at least one PI and at least one NNRTI. Combinations of older drugs such as zidovudine (AZT), which is an NRTI, with either a PI or NNRTI were considered polytherapy.

We also find that private insurance is more effective than public insurance in preventing HIV-related death. In the HCSUS, more than 95% of those with private insurance have drug coverage, and have higher rates of HAART use even though they are less sick. But while all Medicaid programs provide prescription drug coverage to those who are categorically eligible, only 39 states provide drug coverage to the medically-needy.[10] Thus, many of the HIV+ population with Medicaid do not have coverage of HAART.

In addition, differences in when therapy is initiated may play a role. A few private insurers may place limits on when it will cover HAART, but Medicaid limits can be quite severe. Many states place limits on how many prescriptions can be filled per month, and since HAART therapy alone averages 4.8 prescriptions, these can limit coverage for not only HAART but also drugs to treat opportunistic infections associated with advanced disease. Many anti-retroviral drugs also required prior authorization from Medicaid that restricted use to advanced illness. The result is that privately insured patients are able to start treatment earlier in the disease than the publicly insured.

Our results suggest that insurance expansions for HIV+ patients can save lives. Examples include allowing HIV+ patients to keep private coverage as their disease progresses, perhaps through buy-in provisions beyond the current period limitations or extending Medicaid coverage to HIV+ populations. Public insurance expansions will save the most lives if they include alleviating restrictions on drug coverage that limit access to HAART. Of course, all expansions need to consider the costs in terms of crowd-out and effects on employment and total costs. Given the efficacy of treatment, however, it is likely that these expansions will be worth the costs.

Acknowledgements

Appendix A

The variance–covariance matrix for the unobserved heterogeneity is:

$$
\begin{aligned}
&\text{Var}(\rho_{\text{m}}, \rho_{\text{private}}, \rho_{\text{public}}) \\
&= \begin{bmatrix}
\sum_k p_k (\rho_{\text{m}}^k)^2 & \sum_k p_k \rho_{\text{m}}^k \rho_{\text{private}}^k & \sum_k p_k \rho_{\text{m}}^k \rho_{\text{public}}^k \\
 & \sum_k p_k (\rho_{\text{private}}^k)^2 & \sum_k p_k \rho_{\text{private}}^k \rho_{\text{public}}^k \\
 & & \sum_k p_k (\rho_{\text{public}}^k)^2
\end{bmatrix}
\end{aligned}
\tag{A.1}
$$

---

[10] Using program participation and income data in the HCSUS, we estimated elsewhere that 30% of those with HW qualify for Medicaid as medically-needy.

This model not only allows non-zero covariance across mortality and insurance propensities but also allows non-zero covariance between private and public insurance propensities. Thus, our model relaxes the IIA assumption of standard multinomial logit models and allows a completely general variance–covariance matrix. The key correlations in our model may thus be written as

$$\text{Corr}(\rho_\text{m}, \rho_\text{private}) = \frac{\sum_k p_k \rho_\text{m}^k \rho_\text{private}^k}{\sqrt{\sum_k p_k (\rho_\text{m}^k)^2 \sum_k p_k (\rho_\text{private}^k)^2}} \tag{A.2}$$

$$\text{Corr}(\rho_\text{m}, \rho_\text{public}) = \frac{\sum_k p_k \rho_\text{m}^k \rho_\text{public}^k}{\sqrt{\sum_k p_k (\rho_\text{m}^k)^2 \sum_k p_k (\rho_\text{public}^k)^2}} \tag{A.3}$$

$$\text{Corr}(\rho_\text{private}, \rho_\text{public}) = \frac{\sum_k p_k \rho_\text{private}^k \rho_\text{public}^k}{\sqrt{\sum_k p_k (\rho_\text{private}^k)^2 \sum_k p_k (\rho_\text{public}^k)^2}} \tag{A.3}$$

We use maximum likelihood to estimate the parameters of our model. The problem is that we do not observe any patient's type and so we must integrate over all possible types (three in our model). However, conditional on a given type, the heterogeneity is known. The contribution of patient i to the likelihood function is thus given by

$$\begin{aligned}
l_i = \sum_{k=1}^{3} p_k &(P[m_i = 1|\rho_\text{m}^k]^{m_i}) \times (1 - P[m_i = 1|\rho_\text{m}^k]^{1-m_i}) \\
&\times (P[\text{private}_\text{i} = 1|\rho_\text{private}^k]^{\text{private}_\text{i}}) \times (P[\text{public}_\text{i} = 1|\rho_\text{private}^k]^{\text{public}_\text{i}}) \\
&\times (1 - P[\text{private}_\text{i} = 1|\rho_\text{private}^k] - P[\text{public}_\text{i} = 1|\rho_\text{private}^k])^{(1-\text{private}_\text{i})(1-\text{public}_\text{i})}
\end{aligned} \tag{A.5}$$

We have six possible outcomes for the dependent variables in our sample: (dead/alive) × (uninsured/private/public). To construct the contribution to the likelihood function for each set of outcomes, we first obtain the likelihood of observing that value of the dependent variables conditional on a realization k of the unobserved heterogeneity $\rho^k = (\rho_\text{m}^k, \rho_\text{private}^k, \rho_\text{public}^k)$. We then integrate over all possible realizations to get Eq. (A.5). Finally, we obtain the weighted log-likelihood function by summing the log-likelihood across individuals:

$$\ln L(\Gamma) = \sum_{i=1}^{N} w_i \ln(l_i) \tag{A.6}$$

Γ is the vector of model parameters; w_i are the analytic weights and N is the sample size. Because it is difficult to interpret the magnitude of the parameter estimates directly, we also report the relative impact of private and public insurance on average mortality. More

precisely, we compute:

$$\text{RI (private)} \equiv \frac{\text{average mortality private}}{\text{average mortality not insured}} - 1$$

$$= \frac{\sum_i \sum_k w_i p_k \Phi(c_1 + \gamma_1 + \beta_1' X_i + \rho_m^k)}{\sum_i \sum_k w_i p_k \Phi(c_1 + \beta_1' X_i + \rho_m^k)} - 1 \tag{A.7}$$

$$\text{RI (public)} \equiv \frac{\text{average mortality public}}{\text{average mortality not insured}} - 1$$

$$= \frac{\sum_i \sum_k w_i p_k \Phi(c_1 + \gamma_2 + \beta_1' X_i + \rho_m^k)}{\sum_i \sum_k w_i p_k \Phi(c_1 + \beta_1' X_i + \rho_m^k)} - 1 \tag{A.8}$$

References

Andersen, R., Bozzette, S., Shapiro, M., St. Clair, P., Goldman, D., Wenger, N., 1999. Access of vulnerable groups to antiretroviral therapy among persons in care for HIV in the US. Health Services Research 35 (3), 389–416.

Bound, J., Jaeger, D., Baker, R., 1995. Problems with instrumental variables estimation when the correlation between the instruments and the endogenous explanatory variable is weak. Journal of the American Statistical Association 90 (430), 443–450.

Bundorf, K., 2002. Employee demand for health insurance and employer health plan choices. Journal of Health Economics 21 (1), 65–88.

Cantor, C., Long, S., Marquis, S., 1995. Private employment-based health insurance in ten states. Health Affairs 14 (2), 199–211.

Center for Disease Control, 1998. Diagnosis and reporting of HIV and AIDS in states with integrated HIV and AIDS surveillance—United States, January 1994–June 1997. Morbidity and Mortality Weekly Report 47 (15), 309–314.

Cutler, D., 1995. The incidence of adverse medical outcomes under prospective payment. Econometrica 63 (1), 29–50.

Cutler, D., Gruber, J., 1996. Does public insurance crowd out private insurance? Quarterly Journal of Economics 111 (2), 391–430.

Duan, N., McCaffrey, D.F., Frankel, M.R., St. Clair, P.A., Beckman, R., Keesey, J.W., Chien, C., Goldman, D.P., Smith, S., Morton, S.C., 1999. Hcsus baseline methods technical report: weighting, imputation and variance estimation. RAND Report MR-1060-AHCPR, RAND, Santa Monica, CA.

Employee Benefit Research Institute, 2001. Sources of health insurance and characteristics of the uninsured: Analysis of the march 2001 current population survey. EBRI Issue Brief #240.

Fauci, A., Braunwald, E., Isselbacher, K., Martin, J. (Eds.), 1998. Harrison's Principles of Internal Medicine, 11th ed. Mc-Graw Hill, New York.

Fleishman, J., Hsia, D., Hellinger, F., 1994. Correlates of medical service utilization among people with HIV infection. Health Services Research 29 (5), 527–538.

Goldman, D., 1995. Managed care as a public cost containment mechanism. RAND Journal of Economics 26 (2), 277–295.

Goldman, D., Bhattacharya, J., McCaffrey, D., Duan, N., Leibowitz, A., Joyce, G., Morton, S., 2001. The effect of insurance on mortality in an HIV+ population in care. Journal of the American Statistical Association 96 (405), 883–894.

Goldman, D., Smith, J., 2002. Can patient self-management help explain the SES health gradient? Proceedings of the National Academy of Sciences 99 (16), 10929–10934.

Heckman, J., 2001. Micro data, heterogeneity, and the evaluation of public policy: nobel lecture. Journal of Political Economy 109, 673–748.

Horner, R., Bennett, C., Achenbach, C., Rodriguez, D., Adams, J., Gilmann, S., Cohn, S., Dickinson, G., DeHovitz, J., Weinstein, R., 1996. Predictors of resource utilization for hospitalized patients with *Pneumocystis carinii* pneumonia (pcp): a summary of effects from the multi-city study of quality of pcp care. Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology 12, 379–385.

Institute of Medicine, 2002. Care Without Coverage: Too Little, Too Late. Institute of Medicine.

Joyce, G., Goldman, D., Leibowitz, A., Duan, N., Carlisle, D., Shapiro, M., Bozzette, S., 1998. Variation in inpatient resource use in the treatment of HIV: do the privately insured receive more care? Medical Care 37 (3), 220–227.

Lancaster, T., Intrator, O., 1998. Panel data with survival: hospitalization of HIV-positive patients. Journal of the American Statistical Association 93 (441), 46–53.

Levy, H., Meltzer, D., 2001. What do we really know about whether health insurance affects health? Working Paper #274, Joint Center for Poverty Research.

Mroz, T., 1999. Discrete factor approximations in simultaneous equation models: Estimating the impact of a dummy endogenous variable on continuous outcomes. Journal of Econometrics 92 (2), 233–274.

Nelson, C.R., Startz, R., 1990. Some further results on the exact small sample properties of the instrumental variable estimator. Econometrica 58 (4), 967–976.

Nyman, J., 1999. The value of health insurance: the access motive. Journal of Health Economics 18 (2), 141–152.

Palella, F., Delaney, K., Moorman, A., Loveless, M., Fuhrer, J., Satten, G., Aschman, D., Holmberg, S., 1998. The HIV outpatient study investigators: declining morbidity and mortality among patients with advanced human immunodeficiency virus infection. New England Journal of Medicine 338 (13), 853–860.

Shapiro, M.F., Morton, S.C., McCaffrey, D.F., Andersen, R.M., Berk, M.L., Bing, E.G., Cleary, P.D., Cunningham, W.E., Fleishman, J.A., Kanouse, D.E., Senterfitt, J.W., Bozzette, S.A., 1999. Variations in the care of HIV-infected adults in the United States: results from the HIV cost and services utilization study. Journal of the American Medical Association 281, 2305–2315.

Staiger, D., Stock, J.H., 1997. Instrumental variables regression with weak instruments. Econometrica 65 (3), 557–586.