Managed Care, Drug Benefits and Mortality: An Analysis of the Elderly

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We evaluate the impact of the Medicare HMO program and prescription drug coverage on elderly mortality using data from 1993 to 2000. We specify a model of plan entry and benefit choice and Medicare enrollee plan choice and health outcomes. We derive an estimator that is consistent with endogenous plan selection by using the quasi-experimental variation caused by peculiarities of the Medicare reimbursement system for HMOs. We find that, relative to traditional Medicare, enrollment in an HMO without drug coverage increases mortality while enrollment in an HMO with drug coverage has no significant impact. The economic value of the reduction in mortality from drug coverage far outweighs the costs. HMOs, in particular those without drug coverage, attract healthier enrollees than average.

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1. Introduction

In the United States, the 1990s saw a dramatic rise of a relatively new organizational mechanism for financing and organizing health care: managed care. By the end of the decade the vast majority of individuals with private health insurance were enrolled in some form of managed care (Glied, 2000). By using utilization management, writing high-powered contracts with plan providers, and perhaps most importantly, exercising bargaining power over providers, managed care offers a potentially more efficient alternative to traditional indemnity health insurance.[1] However, part of the reduction in costs from managed care may be due to a lower quality of care.

The potential efficiency gains from managed care have influenced government policy. Starting in 1982, the Medicare administration offered managed care plans to its enrollees as a way to reduce expenditures in this gigantic government program. By 2000, 15% or over 6 million Medicare enrollees opted for a private health maintenance organization (HMO) instead of traditional Medicare insurance. The 2003 Medicare Prescription Drug, Improvement and Modernization Act (MMA) had two intertwined legislative intents: moving Medicare further towards managed care and increasing access to drug coverage for the elderly. Drug coverage has become increasingly important because of the development of many useful drug therapies over the last 30 years (Lichtenberg, 2002a, b). The MMA has been criticized on several levels, including by researchers who believe that it may result in lower quality care and cause plans to design benefits to exploit adverse selection (see McAdams and Schwartz, 2006). More recently, the Patient Protection and Affordable Care Act (PPACA) of 2010 increases the subsidies for drug coverage for the elderly by reducing coverage gaps and finances the subsidies for private health coverage by reducing payments to Medicare managed care plans.

The purpose of this study is to evaluate the impact of Medicare HMOs on the quality of health care, using data from 1993 to 2000. We define quality using what is generally considered the most important outcome measure, mortality, in our case mortality for the elderly.[2] Medicare HMOs may offer benefits that exceed the basic Medicare package. Generally the most valued additional benefit is drug coverage, and hence a related purpose of this study is to evaluate the impact of drug coverage, as provided by HMOs, on mortality. We specify a model of HMO entry, pricing, and benefit decisions and Medicare enrollee plan choices

[1] For instance, Cutler et al. (2000) find that the main effect of managed care is to lower prices paid to providers.

[2] There are many other measures of health outcomes including (but not limited to) morbidity, physical functioning status, psychosocial functioning and quality of life (Donabedian, 1985). We focus on mortality because it is unambiguously a measure of health outcomes, measurement error is modest, and it is readily measured with available data.

and health outcomes. We use the model to derive estimating equations and methods that are consistent with endogenous selection into HMOs and, in conjunction with the estimates, to provide evidence on the equilibrium provision of quality and the extent of adverse selection in this market and on the relative benefit to costs of the Medicare HMO program and Medicare drug coverage.

We hope that our study will inform the policy debate on the impact of Medicare HMOs and drug coverage on health outcomes and, through that, provide more general evidence on the impact of managed care on the quality of health care. The literature on the impact of Medicare HMOs on mortality has not reached any consistent conclusions,[3] nor has the overall literature on the effect of managed care penetration on health outcomes.[4] A likely reason for the inconsistent findings may be an endogeneity problem, where people are likely to select into HMOs and drug coverage on the basis of their health status, and health status is not perfectly observed. A number of other studies that assess the impact of managed care penetration on different outcomes have attempted to control for this endogeneity problem with a variety of methods.[5]

Our data provide us with a unique source of quasi-experimental variation that allows us to address the endogeneity problem in a manner that is consistent with our model. The quasi-experimental variation is based on peculiarities of the method by which the Medicare administration reimbursed HMOs for providing health care to its enrollees: until 1997, HMOs were reimbursed a fixed payment rate based on the mean realized per capita fee-for-service (FFS) expenditures in the county over a six-year period, from eight years prior to three years prior.[6] Provided that *unobserved shocks* to health status in a county are sufficiently independent that there is no correlation between the shocks in a given year and for three years prior, the payment rate can be used to construct instruments.[7] The unobserved shocks are more likely to be independent if we include sufficient observed measures of health status. Our observed measures are extensive; they

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[3] While Maciejewski et al. (2001) and Riley et al. (1989, 1991) find that Medicare HMO enrollees have a lower probability of death than other Medicare enrollees, other studies find no significant difference between the groups for breast cancer, prostate cancer, end-stage renal dialysis, and acute myocardial infarction (see Lee-Feldstein et al., 2000, Roetzheim et al., 2000, Potosky et al., 1999, Eggers et al., 2002, and Sada et al., 1998).

[4] A review by Miller and Luft (2002) reports that over the period 1997-2001, nine studies find that HMOs lead to lower mortality, twelve find that HMOs lead to higher mortality, and six find no difference.

[5] Baker and Brown (1999) use instruments formed from the size distribution of firms (without fixed effects), Cutler et al. (2000) use fixed effects, and Dranove et al. (2002) use fixed effects with long differences.

[6] As we detail below, the reimbursement scheme was different for the last three years of our sample, but the same basic idea for identification will apply.

[7] Chernew et al. (2008) used the same identification scheme to analyze the impact of Medicare managed care enrollment on Medicare FFS costs.

include a county fixed effect, the contemporaneous health status of younger people (as measured by mortality), detailed demographic data, and measures of supplemental health coverage (such as Medigap and Medicaid). We also provide evidence on the empirical validity of the independence assumption.

As an example of the forces that will identify our parameters, suppose County A in 1993 has a Medicare patient who undergoes a costly and rare procedure for a potentially fatal condition, such as a heart transplant or a heart assist implant.[8] This will substantially boost County A's reimbursement rate in 1996 relative to 1995, which will cause plans to enter and offer drug benefits and lower prices (see Town and Liu, 2003). This will, in turn, increase enrollment in HMOs that offer drug coverage, which may affect County A's mortality rate in 1996. If unobserved health shocks that cause mortality are not correlated between 1993 and 1996, this will provide an appropriate and useful source of identification of the impact of managed care and drug coverage on mortality.

The remainder of this paper is divided as follows. Section 2 provides a background on the institutional framework. Section 3 explains our model and inference. Section 4 details the data. Section 5 presents the results. Section 6 concludes.

2. Institutional background

We focus on the Medicare program for the aged, which served 35 million of the 41 million Medicare enrollees in 2003.[9] This traditional FFS Medicare program consists of two parts. Part A covers hospital stays (with a small deductible) and catastrophic care. Part A enrollment is automatic. In addition, enrollees can (and most do) enroll in Part B for a premium ($43.80 per month in 1998). Part B covers physician services with a 20% coinsurance, lab and diagnostic tests, outpatient services with a 20% copayment, and mental health care with a 50% copayment. Medicare's Part A and B programs do not cover long-term care, prescription drugs, most preventive care, dental care, or eye care.

In 1982, Congress passed the Tax Equity and Fiscal Responsibility Act which directed the Health Care Financing Administration, now called the Centers for Medicare and Medicaid Services (CMS), to contract with HMOs to provide a managed care option to Medicare enrollees. The Medicare HMO program has been given several names throughout its history and is currently called Medicare Advantage. During the 1997 to 2003 period, which much of our data span, the program was called Medicare+Choice (M+C), and hence we refer to the program

[8] In 2002 Medicare paid for 484 heart transplants and 562 heart assist implants. The cost of these procedures to Medicare often exceeds $370,000 and $240,000, respectively. A good portion of health care costs consist of such costly and rare procedures.

[9] The other 6 million Medicare beneficiaries are either disabled or End Stage Renal Dialysis eligible.

by this name. In counties with available M+C plans, Medicare beneficiaries can enroll in or withdraw from an M+C plan on a monthly basis. Beneficiaries who withdraw from an M+C plan are automatically enrolled in Medicare FFS Part A and have the option of enrolling in Medicare Part B.

Each contracting HMO agrees to accept all Medicare enrollees residing in the county who enroll with it, and to provide and assume the risk for all Part A and B covered services to the individual within a set provider network.[10] The HMO is also allowed to provide extra benefits, most notably drug coverage, reduced copayments for physician visits, dental benefits, and coverage for eyeglasses and durable medical equipment. In exchange for providing health care, the HMO receives a per-enrollee payment from CMS. In addition, the plan could charge a monthly premium to the enrollee.[11] The offered premiums and benefits are subject to CMS approval. For instance, before the enactment of the Balanced Budget Act of 1997 (BBA) each Medicare HMO had to be an existing commercial HMO in the metropolitan area with a Medicare HMO enrollment capped at 50% of its total enrollment in the area.

Each year from 1982 until 1997, CMS set the per-enrollee HMO payment at 95% of its projected Part A and B cost to treat a similar enrollee in the FFS program. The per-enrollee payment is the sum of a county/year-specific base payment and an increment based on age and gender and on institutional, Medicaid, and end-stage renal disease status. Until 1997, the projected base cost was the mean Medicare FFS claims for that county, for the period from eight to three years prior.

The BBA and its subsequent modifications altered Medicare's payment methodology. From 1998 onwards, the monthly base payment rate was set to the maximum of three figures: a blended input price (which mixes an adjusted national rate and an area-specific rate); a floor rate of $367; and a minimum rate increase of 2% per year. Subsequent legislation in the Balanced Budget Refinement Act of 1999 increased the floor rate to $475 (and $525 for large urban counties) and added a 5% increase in the payments to previously underserved markets starting in 2000. Importantly, updates to the county payments remained unaffected by new Medicare FFS claims in the county, implying that the base payment rate continued *not* to be based on claims data in the county within the previous three years. Following these legislative changes, the payment formula led to a substantial relative decrease in the payment rate for most counties. Most Medicare FFS enrollees (92%) also have supplemental insurance that offers benefits beyond Parts A and B. This supplemental insurance is either individually

[10] More precisely, for a given M+C contract, HMOs submit proposed service areas, which are clusters of counties in a given locale, to CMS for approval.

[11] Premiums were constrained to be non-negative during our sample period, although CMS started permitting negative premiums after our sample period.

purchased, provided by the government (through Medicaid, Veterans Affairs, or State Pharmaceutical Assistance) or by an employer. Often this coverage provides prescription drug benefits. Davis et al. (1999) report that of the non-M+C Medicare beneficiaries in 1995, 13% also had Medicaid (which provided drug coverage to 90% of this group), 35% had employer-sponsored Medigap insurance (86% with drug coverage), 31% purchased Medigap in the individual market (46% with drug coverage), 3% had other government-sponsored coverage (80% with drug coverage), and 9% had a mixture of coverage (80% with drug coverage).

3. Model and inference

We develop a simple model of health plan entry, benefit design, consumer choice, and mortality for the M+C market. We do not structurally estimate the model but rather use it to guide the development of estimating equations and the identification strategy.

We consider a metropolitan area that is composed of counties $c = 1,\ldots,C$ and existing commercial HMOs $j = 1,\ldots,J$. Each year, each commercial HMO simultaneously decides in which counties, if any, to offer M+C plans. An HMO that offers an M+C plan would provide and be at-risk for the health care of any Medicare enrollee who chooses to enroll with it. Following the entry decision, each HMO must simultaneously decide on price and benefit structures for each county in which it operates in the M+C market. Let p_{jtc} denote plan j's price and b_{jtc} its benefit design for time t and county c.[12] Prices are constrained to be nonnegative. We focus on the binary benefit choice of whether or not drug coverage is provided. Let $\tilde{d}_{jtc}$ denote managed care plans with drug coverage and $\tilde{n}_{jtc}$ denote those without.

There exists a set of Medicare enrollees in each county, $i = 1\ldots,I$. Let θ_{itc} denote health status, where a higher value of θ_{itc} indicates a higher probability of dying during the year. Each month, each enrollee observes the prices and benefits for each offered plan and then must make a discrete choice between one of the M+C plans offered in her county and the outside option, which is traditional FFS Medicare. We can write utility for any offered plan as:

$$(1) \qquad u_{ijtc} = f\left(p_{jtc}, \theta_{itc}, b_{jtc}\right) + \varepsilon_{ijtc},$$

[12] Plans may offer multiple products with different service offerings, but we do not model this.

where $f(\cdot)$ is some function and ε_{ijtc} is an idiosyncratic unobservable. We normalize the outside option, FFS Medicare, to have utility $u_{i0tc} = \varepsilon_{i0tc}$. Let $h_{itc} \in \{0,\ldots,J\}$ denote the chosen health plan, which maximizes equation (1).

Our choice model may result in adverse selection by enrollees. For instance, an enrollee in poor health (i.e., with a high θ_{itc}) may be likely to choose an HMO because she highly values the service offerings. Alternately, she may be unlikely to choose an HMO because she values access to a broad set of health care providers. One would expect enrollees in poor health to care more about drug coverage and hence be more likely to choose a plan with $\tilde{d}_{jtc} = 1$ than with $\tilde{n}_{jtc} = 1$, all else being equal.

The marginal cost to a plan for treating an enrollee depends on the enrollee's health status and the plan's benefits:

$$(2) \qquad mc_{ijtc} = g(\theta_{itc}, b_{jtc}),$$

for some function $g(\cdot)$. HMOs will likely compete by offering benefits which are both beneficial to enrollees and costly.[13] Firms may endogenously choose benefits, price, and entry or exit to attract patients with a favorable cost profile, generating a second source of adverse selection.

In addition to the marginal cost, HMOs face fixed costs for being in the M+C market in any year. We also allow for dynamic and spatial fixed cost variation: HMOs that were not in the M+C market in the previous year may incur a sunk cost of entry, HMOs that were in the market in the previous year and then exit may incur a sunk cost of exit (potentially negative), and HMOs that operate in more than one county may incur geographic spillover costs (generally negative).

Each year, the Medicare administration sets a county/year reimbursement rate r_{tc}. The gross benefit to the HMO is the reimbursement rate r_{tc} times its number of enrollees. The reimbursement formula changed over time due to the BBA (and subsequent legislation), but never depended on FFS costs within the immediate three previous years or directly on the M+C plan's cost history. Thus, we can express

[13] Town and Liu (2003) find that the provision of drug coverage by M+C plans generates significant consumer welfare.

(3) $$r_{tc} = R_t\left(h_{i\tilde{t}\tilde{c}}, mc_{i0\tilde{t}\tilde{c}} \middle| 1 \le i \le I, \tilde{t} \le t-3, 1 \le \tilde{c} \le C\right),$$

for some formula $R_t(\cdot)$. Firms will have some information, not necessarily perfect, about future reimbursement rates.

Following the choice of plans, individuals will realize their illness state and obtain medical treatment that will either result in a cure for their illness or in death.[14] Let m^*_{itc} denote a latent index for the likelihood of death (we explain below how m^*_{itc} translates into actual mortality). We let m^*_{itc} be a function of the individual's health status and health coverage:

(4) $$m^*_{itc} = \theta_{itc} + \gamma_d \tilde{d}_{h_{itc}tc} + \gamma_n \tilde{n}_{h_{itc}tc},$$

where γ_d and γ_n, the impacts of managed care with and without drug coverage relative to remaining in the Medicare FFS sector, are the key parameters of interest.[15]

To estimate our model, we express an individual's health status as a deviation from the county/year mean, by writing

(5) $$\theta_{itc} = \beta x_{tc} + \xi_{tc} + \tilde{\theta}_{itc},$$

where x_{tc} are county/year mean observed health status measures, β is a coefficient vector, ξ_{tc} is a mean zero unobserved component of aggregate mean health status, and $\tilde{\theta}_{itc}$ is a mean zero individual deviation from the aggregate health status. Rather than actual enrollment, we observe only county/time population mean enrollments by plan types, which we denote d_{tc} and n_{tc} respectively. Let $e_{itc} \equiv \tilde{\theta}_{itc} + \gamma_d\left(\tilde{d}_{h_{itc}tc} - d_{tc}\right) + \gamma_n\left(\tilde{n}_{h_{itc}tc} - n_{tc}\right)$. Then, we can rewrite equation (4) as

(6) $$m^*_{itc} = \beta x_{tc} + \gamma_d d_{tc} + \gamma_n n_{tc} + \xi_{tc} + e_{itc}.$$

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[14] Our model does not allow for dynamic treatment effects where an individual might survive but be more likely to die in a subsequent year based on the received treatment. We explored such specifications but the coefficients on lagged treatment were small and insignificant.

[15] We do not allow service offerings besides drug coverage and managed care to affect mortality.

We estimate equation (6) by aggregating from the individual level to the county/year level and using linear instrumental variables (IVs) weighted by the mean number of Medicare enrollees in the county. Note that the unobserved component of mortality in equation (6) includes both $\tilde{\theta}_{itc}$ and the difference between the actual plan type and the population mean plan type.

We estimate results from two different aggregation functional forms to examine the robustness of our results. For our first functional form, we let m^*_{itc} be a latent illness index, with death occurring if and only if $m^*_{itc} > 0$, and we let e_{itc} be distributed as a logit (i.e., as the difference between two Type 1 extreme value error terms). We further make the assumption that there are enough people within a county/year so that the sampling error in the Medicare mortality rate will be roughly zero. For comparison with the other functional form, let $\nu_{tc} \equiv \xi_{tc}$ for this specification. Following Berry (1994), a transformation of the aggregate mortality rate can then be expressed as a linear function of the regressors:

$$(7) \qquad log\left(\frac{m_{tc}}{1-m_{tc}}\right) = \beta x_{tc} + \gamma_d d_{tc} + \gamma_n n_{tc} + \nu_{tc}.$$

For our second functional form, we let m^*_{itc} be the probability of death for person *i* and take the mean of equation (6) across individuals in the county. Let m_{tc} denote the Medicare enrollee mortality rate, and let $\nu_{tc} \equiv \xi_{tc} + \sum_{i=1}^{I} e_{itc} / I$. We then obtain:

$$(8) \qquad m_{tc} = \beta x_{tc} + \gamma_d d_{tc} + \gamma_n n_{tc} + \nu_{tc}.$$

Regardless of the functional form, managed care enrollment rates may be endogenous. From equation (1), enrollees may choose plans with different service offerings on the basis of θ_{itc} and, in particular, ξ_{tc}. This implies that ν_{tc} will be correlated with enrollees' choices of plan, h_{itc}, and through that with d_{tc} and n_{tc}.[16] We control for the endogeneity using an instrumental variables approach, where functions of the payment rate are used as instruments for d_{tc} and n_{tc}. Our main identifying assumption is that the residual component of health status, ν_{tc}, is

[16] In contrast, because our data are all at the county/time level, using the mean enrollment patterns does not introduce any further endogeneity as the deviation from the mean will be uncorrelated with county/time mean information.

independent across time, specifically that ν_{tc} is independent from any three-year-old or older health shocks. The validity of this assumption depends on the quality of the aggregate health status variables x_{tc}, which need to account for other time-varying attributes that might affect elderly mortality. We include county fixed effects, detailed demographic information, mortality rates among younger people, and supplemental health coverage for the elderly.

The model and identifying assumption imply that functions of r_{tc} are valid instruments for both d_{tc} and n_{tc}: functions of r_{tc} will affect the set of available M+C plans, the benefit structure and premiums of the plans, and through that the share of consumers who choose drug and non-drug plans. In addition, r_{tc}, and hence any function of r_{tc}, is based only on variables three years old or older and will not be correlated with ν_{tc}. Moreover, the model implies that different functions of r_{tc} are *jointly* valid instruments for d_{tc} and n_{tc}. The economic reason for this is that the entry and benefit decisions are nonlinear functions of r_{tc}. Hence, d_{tc} and n_{tc} can each be expressed as a linear combination of multiple non-collinear functions of r_{tc} plus a residual, where the functions can be indicators for r_{tc} being in different quantiles, for instance. As these multiple functions can all be excluded from the treatment equation (7) or (8) but all enter separately into the underlying selection equations determining d_{tc} and n_{tc}, they allow us to jointly identify the impacts of d_{tc} and n_{tc} on mortality.

We illustrate the joint identification with a numerical example. Consider a simple data generating process with many similar counties which differ only in two exogenous dimensions: ν_{tc} and r_{tc}. We let $\theta_{itc} = \nu_{tc}$, let $\nu_{tc} = -.1$ half of the time and $\nu_{tc} = .1$ the other half, and let r_{tc} vary continuously with a distribution that is independent from ν_{tc}. We assume that each county contains one (monopolistic) HMO, plan 1, with no fixed or sunk costs or geographic spillovers. Each HMO knows ν_{tc} and r_{tc} before deciding on entry. We also assume that there is no consumer selection based on θ_{itc}, that the price elasticity of demand is high enough that price is always zero, and that the idiosyncratic enrollee choice unobservables ε_{ijtc} are distributed Type 1 extreme value. The $f(\cdot)$ utility function from equation (1) will then be only a function of benefits b_{1tc}. We let $f(\tilde{d}_{1tc}) = 1$, $f(\tilde{n}_{1tc}) = 0$, $mc_{i1tc}(\nu_{tc}, \tilde{d}_{1tc}) = 1.1(\nu_{tc} + 1)$, and $mc_{i1tc}(\nu_{tc}, \tilde{n}_{1tc}) = \nu_{tc} + 1$ so that drug

coverage increases both costs and utility, with a cost complementarity between drug coverage and illness severity. It is not necessary for us to specify the mortality process.

By standard logit formulas, the HMO would capture 50% of the market share with a non-drug M+C plan, but 73.1% if it had a drug M+C plan. Per-capita profits will be $\pi\left(\tilde{n}_{1tc}\right) = .5\left(r_{jtc} - \left(\nu_{tc} + 1\right)\right)$ and $\pi\left(\tilde{d}_{1tc}\right) = .731\left(r_{jtc} - 1.1\left(\nu_{tc} + 1\right)\right)$. Simple algebra then shows that when $\nu_{tc} = -.1$, the firm will enter with no drug coverage for $.9 \le r_{jtc} \le 1.18$, with no entry below this range and drug entry above this range. When $\nu_{tc} = .1$, the entry pattern is similar, but the comparable range for entry with no drug coverage is $1.1 \le r_{jtc} \le 1.45$. Thus, non-drug entry will occur with a mid-range r_{tc} and drug entry with a high r_{tc}, with the exact range depending on ν_{tc}.

Because of the correlation between ν_{tc} and both d_{tc} and n_{tc}, OLS estimates of equation (7) with this data generating process would be inconsistent. However, we could consistently estimate (7) with instruments $\hat{d}_{tc} = \left\{r_{jtc} > 1.3\right\}$ and $\hat{n}_{tc} = \left\{1 \le r_{jtc} \le 1.3\right\}$, where $\left\{\cdot\right\}$ denotes an indicator function, as these instruments will be non-collinear predictors of both d_{tc} and n_{tc}. Adding in other features of the model (e.g., multiple firms, consumer adverse selection) will make the relation between the reimbursement rate and drug or non-drug entry more complicated, but the same fundamental identification will apply. While the example uses two instruments for simplicity of exposition, one could add to the predictive power of the instruments by generating four instruments of indicators based on the cutoffs .9, 1.1, 1.18, and 1.45. Moreover, the dynamic and geographic cost complementarities in our model imply that we can add further predictive power by using future and past payment rates and payment rates from nearby counties as instruments.

Our actual choice of instruments is complicated by the fact that costs vary significantly across counties, implying that a payment rate that is generous in one county is not generous in another. As county population is highly correlated with costs, we normalize the payment rate based on population by regressing the payment rate on four measures of population (county population, health services area population, metropolitan statistical area (MSA) population, and county elderly population). We define the residual from this regression to be the "normalized payment rate," $\hat{r}_{tc}$. We then create ten instruments based on $\hat{r}_{tc}$: the rate, its second, third, and fourth powers, its log and the square of its log, and four

dummies indicating its quintile (with one excluded). We also create three instruments that indicate the mean, minimum, and maximum normalized payment rates in the MSA, to capture the geographic cost complementarities noted above. To exploit the variation from sunk costs, we then include these same 13 instruments for the previous and subsequent year,[17] for a total of 39 instruments. Because there is a potential tradeoff between asymptotic efficiency (which dictates more instruments) and small sample bias (which dictates less),[18] we also examined results with smaller sets of instruments.

A potential statistical issue with using these instruments is that they are not strictly exogenous: though our model and assumptions imply that $\hat{r}_{tc}$ is uncorrelated with ν_{tc} they also imply that $\hat{r}_{tc}$ is correlated with $\nu_{t-3,c}$ and further lagged residuals. Since fixed-effects IV estimates are equivalent to estimates from a mean-differenced instrumental variables specification, the residual can be expressed as $\nu_{tc} - \frac{1}{T}\sum_{\hat{t}=1}^{T}\nu_{\hat{t}c}$. In a short panel, there may be a correlation between $\hat{r}_{tc}$ and this residual due to the $\frac{1}{T}\sum_{\hat{t}=1}^{T}\nu_{\hat{t}c}$ component of the residual. With a sufficiently long panel, shocks to $\nu_{\hat{t}-3,c}$ (for instance) will have little impact on $\frac{1}{T}\sum_{\hat{t}=1}^{T}\nu_{\hat{t}c}$. Thus, the consistency of IV estimates with this type of instrument depends on asymptotics in time.

With eight years of data, the asymptotic approximation is likely to be close to valid. Nonetheless, we develop a forward mean-differenced specification which provides consistent results without requiring a long panel. This specification transforms the estimating equation by subtracting the *forward mean* (from $t-1$ to T) for each variable instead of the overall mean, keeping the same untransformed instruments as in the base specification. Thus, the transformed residual is $\nu_{tc} - \frac{1}{T-t}\sum_{\hat{t}=t-1}^{T}\nu_{\hat{t}c}$.[19] The instrument $\hat{r}_{tc}$ will then not be correlated with the transformed residual since the residual does not include long-lagged terms such as $\nu_{t-3,c}$. The transformation does result in a serial correlation in the residuals and thus we cluster our reported standard errors at the county level.

4. Data

Our study period is 1993 to 2000. We choose 1993 as the start of the sample

[17] We let the instrument be zero for years that are not in the sample.

[18] Different authors suggest different points along this tradeoff. For instance, for panel data with serial correlation, Arellano and Bond (1991) suggest using every available instrument while Keane and Runkle (1992) suggest a more limited set.

[19] For observations in year one, the difference goes back only to year one.

because prior to this year enrollment in Medicare HMOs was very small. We create a county-level panel data set of mortality rates and other county-specific information. The data come from six different sources, listed in Table 1, which we merge together.

Table 1
Data sources

Data Set	Source	Variables
Compressed Mortality File	National Center for Health Statistics	Mortality rates by age and cause of death
State-County-Plan Penetration file and M+C/AAPCC Standardized Per Capita Rates of Payment	Center for Medicare and Medicaid Services	M+C enrollments by HMO benefit structure and CMS payment data
Medicaid Program Statistics	Center for Medicare and Medicaid Services	Medicaid enrollments by age classification
Area Resource File	Area Resource File	Population by race, per-capita income, number of MDs and hospitals.
Population Estimates Program	Bureau of the Census	Predicted population by age and sex categories
Medigap Premium	AARP	Medigap Premiums for Plan F

First, the mortality data are constructed using the 1989–2004 Compressed Mortality Files (CMFs) from the National Center for Health Statistics (NCHS). The CMFs contain death certificate information including the county of residence, year of death, age, gender, and diagnosed cause of death for all deaths in the United States.[20] We use mortality from two diagnosed causes, heart disease and neoplasms (cancer), as well as all-cause mortality. In 1999, there was a change in the cause-of-death coding. To compare the cardiac and cancer mortality rates across coding systems, we multiplied the pre-1999 mortality rates by comparability ratios provided by the NCHS in the 1999–2004 CMF documentation. The data also include population estimates by county, gender, and five-year age groups. We calculate mortality rates by age group and cause by dividing the number of deaths in each group by the population measures by age

[20] We used these data following a data use agreement with the NCHS.

group. In a small number of cases (less than 0.05%), changes in county definitions resulted in missing mortality rates.

Second, we merge the mortality data with county-level data from CMS on M+C plan enrollments, plan prescription drug benefits, total Medicare enrollment, and the M+C constant dollar payment rate. We define the drug benefit using the base plan as reported by CMS.[21] Our data do not provide the specific limitations of the drug coverage and other available benefit information is prone to substantial reporting errors, and thus our measure of plan benefits is binary. This is a limitation, as it implies that we must lump different levels of drug coverage together.

Third, we use information from CMS on the number of Medicaid enrollees by state and age category (65 to 74, 75 to 84, and 85 and older). We proxy for the county-level Medicaid penetration rate with the state-level rate.

We gather demographic information from two sources. We use data on county per-capita income, population by age and race, number of practicing physicians, and number of hospitals from the Area Resource File. We use detailed demographic data from the Census Bureau's Population Estimates Program to provide a more complete account of the entire age distribution of the elderly by county. These data provide annual county-level projections of the population in each year in each county by age and sex category. The age categories that we use are 64, 65, …, 84, and 85 and older.

Finally, in some specifications we use Medigap premium data from the American Association of Retired Persons (AARP), one of the largest sellers of Medigap policies. By regulation, Medigap plan benefits fall into 10 different categories, labeled A through J. We use the Plan F data because they included the most widely available data on Medicare FFS prices. We were able to obtain price data for 1993–99, but not for 2000.

It is useful to characterize the M+C drug coverage since it is central to this paper. The structure of the benefit varies across three dimensions: generic drug copayments, branded drug copayments, and the total maximum drug expenditure covered by the plan. We have detailed information on the plan drug benefit structure only for 2000.

In 2000, approximately 80% of the M+C plans offered drug coverage with a mean monthly premium of $34.85. Of the plans offering drug coverage, the mean copayment for generic prescription drugs is $7.80 (std. dev. = $2.93; median = $7), and the mean copayment for branded prescription drugs is $16.16

[21] This may lead to measurement error to the extent that HMOs offer multiple M+C plans in a county, some with drug coverage and some without. However, the 2000 Medicare Current Beneficiaries Survey reports that 17.8% of all M+C enrollees are enrolled in a plan without drug benefits. The corresponding figure in our data is 17.3%, suggesting that any measurement error is small.

(std. dev. = $6.12; median = $15). Of these plans, 89% cap the total annual enrollee expenditures on drugs, with 37% setting the cap at less than $1,000 per year, and another 37% setting caps of over $3,000 per year.

Table 2
Summary statistics

Variable	Entire sample	Number of Observations	1993	2000
Mortality rate, age 65+ (%)	5.06 (0.57)	25,014	5.01 (0.54)	5.14 (0.60)
Mortality rate, age 65-74 (%)	2.51 (0.43)	25,014	2.60 (0.41)	2.40 (0.44)
Mortality rate, age 75-84 (%)	5.76 (0.72)	25,014	5.93 (0.68)	5.65 (0.75)
Mortality rate, age 55-64 (%)	1.07 (0.25)	25,014	1.15 (0.24)	0.99 (0.24)
Heart disease mortality rate, age 65+ (%)	2.33 (0.36)	25,014	2.42 (0.36)	2.21 (0.34)
Cancer mortality rate, age 65+ (%)	1.00 (0.16)	25,014	1.06 (0.14)	0.87 (0.13)
MDs per capita (thousands)	2.40 (1.83)	25,112	2.20 (1.70)	2.51 (1.79)
Hosp. beds per capita (thousands)	3.46 (2.40)	25,112	3.84 (2.54)	3.12 (2.22)
Income (thousands of $)	24.9 (7.91)	25,112	20.7 (5.53)	29.5 (9.48)
Unemployment rate (%)	5.41 (2.53)	25,112	7.02 (2.46)	4.20 (2.08)
Total population (thousands)	900 (1,674)	25,115	877 (1,649)	944 (1,742)
M+C drug penetration rate (%)	8.92 (13.7)	25,000	2.63 (6.87)	14.1 (16.4)
M+C drug penetration = 0 (%)	45.1 (49.8)	25,000	68.4 (46.5)	33.5 (47.2)
M+C non-drug penetration rate (%)	3.67 (7.74)	25,000	2.72 (5.94)	3.22 (7.78)
M+C non-drug penetration = 0 (%)	59.1 (49.2)	25,000	57.8 (49.4)	71.6 (45.1)
M+C monthly payment rate (2000 $)	454 (97.5)	24,998	409 (88.5)	487 (83.7)
Medigap F premium (2000 $)	106 (23.0)	19,870	93.6 (15.1)	—*

Note: Each cell provides the mean value, weighted by the number of Medicare enrollees, with the standard deviation in parentheses.
*Our Medigap data are from 1993–99 only.

It is also useful to compare the prescription drug benefits to those offered through Medigap. Plans H through J offer drug coverage. All require a 50% coinsurance on prescription drugs with Plans H and I capping the annual prescription drug expenditure at $1,250 and Plan J capping it at $3,000.[22] The cost

[22] Plans H and I differ in other small respects. Plan I covers Medicare Part B excess charges and at-home recovery expenses while Plan H does not. Plan J offers the same benefits as Plan I with the addition of covering Medicare Part B deductible and preventive care.

of enrolling in a Medigap policy varies across geography and insurers. For AARP, the mean (unweighted) monthly premium across states for ages 65 to 69 is $153.90 for Plan H, $156.57 for Plan I, and $192.57 for Plan J. Thus, M+C plans with drug benefits are significantly less expensive to Medicare enrollees than Medigap plans and, in general, they offer more generous coverage.

Table 2 summarizes the major variables used in the study, weighted by the number of Medicare enrollees. The average elderly mortality rate was 5.06% during the sample period. Cancer and heart disease make up the biggest components of mortality, together accounting for about 60% of the average mortality rate. Mortality rates are rapidly increasing in age, ranging from 1.07% for ages 55 to 64 to 5.76% for ages 75 to 84.

Table 3
Changes in mortality and changes in M+C enrollment from 1993 to 2000 (by 2000 payment)

Quintile of 2000 payment rate	Percentage point change in total M+C enrollment	Percentage point change in M+C non-drug enrollment	Percentage point change in M+C drug enrollment	Percentage point change in elderly mortality
1 [$218 – $381]	1.28%	0.81%	4.78%	0.07%
2 [$381 – $397]	2.74%	1.00%	1.74%	0.12%
3 [$397 – $426]	5.81%	2.24%	3.57%	0.17%
4 [$426 – $464]	8.11%	2.93%	5.19%	0.17%
5 [$464 – $772]	17.6%	–0.83%	18.4%	0.12%

Note: Each cell provides the mean percentage point change from 1993 to 2000 of the given variable when the 2000 M+C payment rate is in the specified quintile. All figures are weighted by the mean number of Medicare enrollees in the county.

In 1993, the average M+C penetration rate across counties was 5.35%, a figure that increased to 17.3% by 2000. Medicare M+C payments were about $454 per month (in constant 2000 dollars), also rising over time. Drug penetration rates were always higher than non-drug penetration rates. There is substantial variation in the payment rate across counties—the standard deviation is 20% of the mean in 1993. Almost half of counties (45.1%) have no M+C plans with drug coverage, although by the end of the sample period this figure had dropped to 33.5%.

Table 3 provides some evidence on the relation between the 2000 M+C payment rate and the changes in M+C enrollment and elderly mortality rates over

the period 1993 to 2000. This table is meant to give an indication of the forces that will identify the estimates, as the fixed-effects IV estimator with one (endogenous) regressor would be the ratio of the coefficients of the differenced regressions of mortality on the instruments to the endogenous regressor on the instruments. The payment rate (the instrument) is broken into five quintiles. As we might expect, a higher payment rate is correlated with higher increases in the total M+C enrollment rate. The trend is most pronounced between the third and fifth quintiles of the rate. Breaking down the enrollment change into drug and non-drug enrollment, a movement from the fourth to the fifth quintile is associated with an increase in drug enrollment, but a decrease in non-drug enrollment. In other words, a moderately high payment rate in 1993 and 2000 was linked with a general increase in managed care enrollment, but a large increase in the payment rate was linked specifically with an increase in drug coverage and not with non-drug coverage.

Turning to the elderly mortality rate, the third and fourth payment quintiles (those associated with the largest increases in non-drug HMO enrollment) show the largest increases in elderly mortality rates. In contrast, the fifth payment quintile (associated with the largest increase in drug HMO enrollment) shows no increase in elderly mortality rates. These results foreshadow our regression analysis findings and suggest that non-drug M+C plans, but not drug M+C plans, may increase elderly mortality.

5. Results

Parameter estimates and implied magnitudes

Table 4 presents the main results of the paper, estimates of equations (7) and (8). We present eight specifications, which differ in the estimation methods, dependent variables, controls, and weighting. Specification 1 provides an OLS regression of the log model (7) of the impacts of M+C drug and non-drug coverage with only year dummies as controls, as a baseline comparison. In this and all other specifications, the omitted category is Medicare FFS. This specification shows that a higher M+C non-drug coverage is associated with a lower elderly mortality rate than Medicare FFS within a year, although without a statistically significant difference, and that M+C drug coverage is associated with a still lower elderly mortality rate, with a statistically significant difference from Medicare FFS. This regression is consistent with the idea that managed care and drug coverage both lower mortality, but is also consistent with a number of other hypotheses based on selection.

Specification 2 adds in county and year fixed effects and the detailed demographic controls noted in the table. Unlike Specification 1, this specification shows that counties with an increase in non-drug managed care penetration had an increase in mortality. While this specification does control for fixed effects, it

does not directly use any credible source of exogenous variation in the managed care penetration rate or in drug coverage.

Table 4
Estimates of the impact of M+C enrollment on mortality

		Dependent variable using 65+ mortality	M+C drug coverage (γ_d)	M+C non-drug coverage (γ_n)	log (55-64 mortality rate)	Estimation method	N
No IV	(1)	$log\left(\frac{m_{tc}}{1-m_{tc}}\right)$	–.26** (.034)	–.055 (.043)	---	Weighted OLS with only year dummies	24,936
	(2)	$log\left(\frac{m_{tc}}{1-m_{tc}}\right)$	.017 (.010)	.040** (.012)	.016** (.003)	Weighted fixed effects	24,433
Base	(3)	$log\left(\frac{m_{tc}}{1-m_{tc}}\right)$	.038 (.043)	.299** (.068)	.012** (.003)	Weighted forward mean-differenced fixed effects IV	24,401
Robustness checks	(4)	$log\left(\frac{m_{tc}}{1-m_{tc}}\right)$	.021 (.040)	.248** (.072)	---	Weighted forward mean-differenced fixed effects IV	24,678
	(5)	m_{tc}	.001 (.0019)	.012** (.0037)	.059** (.014)	Weighted forward mean-differenced fixed effects IV	24,683
	(6)	$log\left(\frac{m_{tc}}{1-m_{tc}}\right)$	–.017 (.035)	.255** (.089)	.015** (.004)	Weighted fixed effects IV	24,401
	(7)	$log\left(\frac{m_{tc}}{1-m_{tc}}\right)$	–.009 (.034)	.214** (.066)	.014** (.003)	Weighted forward mean-differenced fixed effects IV, Medigap	19,426
	(8)	$log\left(\frac{m_{tc}}{1-m_{tc}}\right)$	–.065 (0.091)	.390** (.130)	0.012 (.003)	Weighted forward mean-differenced fixed effects IV, fewer instruments	24,432

Note: All specifications include year fixed effects. Specifications 2 through 6 include as controls: percent of elderly in Medicaid; percent of population age 65 and over; log per capita income; unemployment rate; MDs and hospital beds per capita; percent of white, black and Hispanic; five regional time trends; and the percent of elderly at each age/sex cell and county fixed effects. Specification 7 adds the log of the Medigap Plan F premium. All standard error calculations are clustered at the county level. The instrument set is specified in Section 3. Specification 8 uses only contemporaneous values of the instrument set – 5 total instruments.
** Significant at the 1% level.
* Significant at the 5% level.

Specification 3 provides our base specification, and uses the fixed effects IV model with the instruments and forward-mean-differencing procedure detailed in Section 3. We estimate the impact of M+C drug coverage on elderly mortality to be very similar to Specification 2. However, we estimate the impact of M+C non-drug coverage to be much worse, with a coefficient that is about 7 times as large as in Specification 2. Thus, our main finding is that enrollment in M+C plans without drug coverage causes an increase in mortality but that M+C plans with drug coverage have no significant mortality impact, both relative to Medicare FFS.

Using the coefficients from this specification, we find that moving a Medicare enrollee with the mean mortality rate of 5.06% from an M+C non-drug plan to an M+C drug plan would reduce the mortality rate by 1.41 percentage points; the weighted sample average change in mortality rate from this move is 1.46 percentage points. Combining this information with the 34.3 million elderly Medicare enrollees in 2000 implies that each 1% move from an M+C non-drug plan to a drug plan would save about 5,000 lives. There are other plan attributes besides drug coverage that may be (positively or negatively) correlated with the drug benefit. These include the benefit offerings noted in Section 2 as well as the possibility of wider physician networks. We believe that these attributes are unlikely to have a mortality impact and hence that our estimated effect is due to drug coverage.[23]

Evaluating the average economic value of M+C drug coverage is somewhat difficult, because it depends on the expected number of years and quality of the remaining life for each person under consideration for receiving drug treatment. However, we can give some plausible bounds to this value. Conservatively, we assume that the mortality gains from drug coverage are limited to only one extra year of life, we value only mortality gains and we use $75,000 as the net value of a life year (following Cutler and McClellan, 2001). This yields an average per-capita value of drug coverage of approximately $1,050. An upper bound calculation can be made by assuming that a death avoided returns a person to the mean health status and life expectancy of her age cohort. Using data on mortality probabilities and life expectancy from life tables for 2000 from the National Center for Health Statistics (Arias, 2002) and the same net value of a life year yields an average per-capita value of approximately $10,539.[24] It is instructive to compare these values to the cost of drug coverage

[23] Importantly, very few M+C non-drug enrollees had drug coverage because alternative drug coverage generally duplicates M+C benefits. For instance, only 9% of M+C non-drug enrollees had drug coverage in 2000, according to the Medicare Current Beneficiary Survey.

[24] The implied benefit for the values of the coefficient is $5,477 at the 5th percentile and $16,078 at the 95th percentile.

provision. The typical difference in the annual premiums between Medigap G and J plans (which are similar in benefits except that only J offers drug coverage) in 2000 is about $760.[25] Thus, even our conservative estimates suggest that drug coverage adds a substantial benefit over its cost.

Understanding the relative mortality impact of Medicare FFS and M+C is more subtle. Due to data limitations, Medicare FFS enrollees with and without drug coverage are combined into one category. Davis et al. (1999) report that 63% of Medicare FFS enrollees had drug coverage in 1995. It is likely that Medicare FFS enrollees for whom drug coverage would lower the probability of mortality are more likely to obtain drug coverage than others, implying that 63% is a likely lower bound on FFS drug coverage for enrollees for whom drug coverage would lower mortality. The M+C drug coefficient can be interpreted as capturing both the effect of moving a group to HMOs and of moving some fraction of the group, likely less than 37%, to drug coverage. Thus, our coefficient estimates are consistent with the notion that HMOs have no overall mortality impact.

Specifications 4 to 8 provide different robustness checks of the findings in Specification 3. Specification 4 drops the log mortality rate of middle-aged individuals, ages 55 to 64, as a regressor. This statistically significant control is potentially important because it will proxy for a variety of time-varying local attributes that can affect mortality and are potentially correlated with the instruments, such as shifting unobserved local demographic patterns or time-varying local changes to practice style. We find that dropping this younger mortality rate results in estimated effects of M+C drug and non-drug coverage that are virtually unchanged from Specification 3. The implication is that while the middle-aged mortality rate in a county is an important predictor for the elderly mortality rate, the above effects for which it proxies are not correlated with the instruments, both conditional on all the other regressors. We believe that the stability of the results across these specifications also provides further evidence against other potential non-included regressors influencing our main estimated coefficients.

Specification 5 uses the same controls and instruments as the base but with the linear model in equation (8) instead of the log model in equation (7). Although the coefficients are not directly comparable to those of the base because of the different dependent variable, we find the same relative impacts as in the base. In addition, the magnitudes are very similar, with a move from M+C non-drug to drug plans reducing the elderly mortality rate by 1.2 percentage points. We prefer using the log specification because it allows for the proportional scaling of factors such as the middle-aged mortality rate.

Specification 6 reports results from the same specification as the base but

[25] Authors' calculation using AARP data from California.

using standard fixed effects IV instead of the forward mean-differenced procedure. This procedure generates coefficients that imply a similar mortality impact from both the M+C drug and non-drug plans than in the base specification. This suggests that the bias from the short panel and instruments that are not strictly exogenous is small.

Specification 7 is similar to the base but adds in a state-level proxy for the log Plan F Medigap premium. We obtain results that are very similar to the base. The Medigap coefficient, which we do not report in the table, is small and insignificant, with a t statistic of 1.60. This variable is likely not significant in part because it is a rough proxy of the availability of supplemental coverage and/or because there may be little demand elasticity to Medigap premiums among enrollees for whom drug coverage might affect mortality. We do not include the premium for our other specifications because of its lack of significance and the fact that the premium data are not available in every year and hence would lower the sample size.

Table 5
Estimates of the impact of M+C enrollment on mortality by age and disease

		M+C drug coverage (γ_d)	M+C non-drug coverage (γ_n)	log (55-64 mortality rate)	N
(1)	Mortality rate age 75+	.053 (.050)	.275** (.071)	.010* (.005)	24,379
(2)	Mortality rate age 65–74	.020 (.046)	.262** (.093)	.021** (.005)	24,356
(3)	Heart disease mortality rate age 65+	.166** (.048)	.249** (0.068)	.007** (0.003)	23,409
(4)	Cancer mortality rate age 65+	–.094* (.047)	.109 (.063)	.004 (.004)	23,341

Note: All estimation is performed using the forward mean-differenced fixed-effects IV estimator weighted by the mean number of Medicare enrollees in the county. All specifications include the following controls: percent of population in Medicaid for age range; percent of population age 65 and over; log per capita income; unemployment rate; MDs and hospital beds per capita; percent white, black and Hispanic; five regional time trends; the percent of elderly at each age/sex cell; and year and county fixed effects. Specifications 3 and 4 include the heart disease and cancer mortality rates for age 55-64 as controls, respectively. All standard error calculations are clustered at the county level. Instrument set specified in Section 3.
** Significant at the 1% level.
* Significant at the 5% level.

Specification 8 uses five instruments instead of the 39 in the base specification. Specifically, we use as instruments the contemporaneous county

payment rate and indicators for the relative payment quintile of the county (with one excluded). This specification also gives similar results to the base specification.

Table 5 provides evidence on the impact of M+C drug and non-drug coverage on a number of different groups. All specifications use the fixed-effects IV forward mean-differenced method to control for endogenous selection into plan type. The specifications follow the base with some small deviations in the controls, noted in the table.

Specifications 1 and 2 report the impact of M+C drug and non-drug coverage on mortality for the 65 to 74 and over-75 age groups, respectively. We find results that are similar to the base specification. For both age groups, the non-drug M+C coefficients remain significant and large and the drug M+C coefficients remain small and insignificant.

Specifications 3 and 4 estimate the impact of M+C drug and non-drug coverage on disease-specific elderly mortality rates. We choose the two diseases with the largest mortality for the elderly, cancer and heart disease. We find that M+C drug enrollment causes a significantly positive increase in the mortality rate for heart disease, with M+C non-drug enrollment causing an even larger impact. These results suggest that managed care causes worse outcomes for cardiac care, consistent with evidence that managed care provides less intensive cardiac care (see Chernew et al., 2002) and that intensive cardiac treatments have a beneficial impact on mortality (see Cutler, 2004). We also find that enrolling in a managed care plan with drug benefits reduces mortality rates for cancer. During our sample period, it is unlikely that intensive cancer treatments had substantial mortality impacts. However, anti-nausea drugs, which may contribute to survivorship by increasing the tolerance for chemotherapy, are mostly not covered by Medicare FFS.

Specification tests

The causal interpretation of our results above depends crucially on our assumption of exogenous instruments. Accordingly, we have performed a number of statistical tests of the validity and usefulness of our instruments, all using our base specification, as given in Table 4, Specification 3.

First, we test the power of our instruments by performing first-stage regressions of the endogenous regressors d_{tc} and n_{tc} on the instruments and exogenous regressors, as suggested by Bound et al. (1995). As in the base specification, we use forward mean difference variables to allow for fixed effects. These tests show that the instruments are strong predictors. We can strongly reject the null hypothesis that the instruments do not enter in these regressions, with $F(36, 3081) = 23.81$, $p=.00$ for d_{tc} and $F(36, 3081) = 13.78$, $p =.00$ for n_{tc}, using

clustered residuals. The reason for this result is that the substantial within-county variation in the payment rates is driving M+C enrollment and drug benefits. We also jointly estimate the same two equations with a multivariate regression to test whether the coefficients on the instruments are the same across the two equations. The test strongly rejects the coefficients being the same with $\chi^2(36) = 2{,}926$, p = .00. This implies that the first-stage projections of d_{tc} and n_{tc} are significantly different from each other, which is necessary to *jointly* identify the effects of drug and non-drug managed care coverage.[26]

Second, we provide evidence on the serial correlation of the residual health shocks v_{tc}, since our main identifying assumption is a lack of correlation between $v_{t-3,c}$ and . Although this assumption is not directly testable, we can obtain some evidence by examining the correlation of the residual from a reduced-form regression of the dependent variable $log\left(\frac{m_{tc}}{1-m_{tc}}\right)$ on all exogenous variables, including the instruments and fixed effects. We estimate this reduced-form regression specifying a within-county AR(1) process for the residual, and find an estimated correlation of $\rho = .082$, which implies that the estimated correlation after three years is $\rho^3 = .00055$. This does not seem consistent with a sizeable, positive serial correlation between $v_{t-3,c}$ and v_{tc}.

Third, we test for the endogeneity of M+C drug and non-drug coverage, by performing a Wu (1973) – Hausman (1978) test of our base specification against a specification without instruments, as given in Table 4, Specification 2. We can reject the exogeneity of the M+C coverage rates, with F(2,3087) =18.24, p=.00.[27]

Last, we perform the LM test of overidentifying restrictions created by the fact that we have 39 instruments but only two endogenous regressors (see Hansen, 1982). We fail to reject the assumption that the instruments are exogenous, with $\chi^2(37) = 44.4$, p = .19.

Explanation of our findings

A likely explanation for our finding is that M+C drug coverage encourages the elderly to take life-extending prescription drugs by lowering the marginal cost of drugs. There is substantial support for this in the literature. Anecdotally, physicians report that financially constrained patients "extend" their prescriptions

[26] The test of the hypothesis that all of the coefficients are equal across the two equations is also soundly rejected.

[27] We use the specification of the Hausman-Wu-Durbin test outlined in Davidson and MacKinnon (2004)

by taking their drugs less frequently than prescribed (Lagnado, 1999).

The literature provides data-driven supporting evidence for this explanation. In 1995, 86.6% of Medicare beneficiaries had a prescription filled (Adams et al., 2001). Several studies (Lillard et al., 1999, Davis et al., 1999, and Stuart and Grana, 1998) find a positive correlation between prescription insurance coverage and prescription drug usage in the elderly population. Poisal and Murray (2001) estimate that Medicare enrollees without drug coverage fill 2.4 fewer prescriptions than enrollees with drug coverage. Poisal and Chulis (2000) find that, among Medicare enrollees with three or more limitations to the activities of daily living, those without drug coverage use 43% less prescription drugs in dollar terms than the same population with prescription drug coverage. Using data from employer-based health coverage, Goldman et al. (2004) find that increased out-of-pocket expenditures reduced the use of drugs for a variety of conditions.

None of these studies attempts to control for unobserved selection into drug coverage or managed care. While these studies generally examine relatively small samples, without enough power to identify mortality differences, some recent studies (Lucarelli, 2006, and Yang et al., 2004) find that increased drug coverage reduces mortality among the elderly.

It is also useful to compare our estimated magnitude of 5,000 lives saved from the 1% movement to drug coverage to the literature. We perform back-of-the-envelope calculations for three common conditions for which data were available—high cholesterol, hypertension and diabetes—using the following formula: Δ mortality = base mortality rate $\times$ (% increase in mortality from condition) $\times$ prevalence of condition in Medicare population $\times$ % reduction in mortality from prescription drug use $\times$% reduction in prescription drug use from lack of insurance $\times$ 1% $\times$ size of Medicare population. We use a conservative base mortality rate of 3.5%. Adams et al. (2001) report that the absence of drug coverage reduces the use of hypertension drugs for individuals with high blood pressure by 23%. We use this estimate for all three conditions, as it appears to be roughly the median estimate from the sparse literature on compliance.

Our estimates indicate a large impact of drug coverage among patients with high cholesterol. This condition is prevalent—50% of the elderly population has blood serum cholesterol levels in excess of 240 mg/dL (National Health and Nutrition Examination Survey III). Pekkanen et al. (1990) report that serum blood cholesterol in excess of 240 mg/dL increases mortality risk by 350% for the elderly while Shepherd et al. (1995) find that pharmaceutical treatment for high blood cholesterol reduces all-cause mortality by 22%. Combining the estimates yields a saving of 700 lives from drug coverage for high cholesterol enrollees. Performing similar calculations for hypertension and diabetes yield mortality

decreases of 240 and 330 lives, respectively.[28] Since our back-of-the-envelope calculations of three common conditions result in an expected increase of approximately 1,260 lives, this suggests that our estimated value of 5,000 lives is plausible.

Adverse selection and equilibrium provision of quality

Our model implies the possibility of consumer selection into managed care plans based on their attributes. Our results from Table 4 suggest that there is in fact substantial adverse selection. In particular, a comparison of Specifications 1 and 3 shows that even though mortality rates for M+C plans are substantially lower than for Medicare FFS, the causal effect of these plans is not to lower mortality rates. This shows that managed care plans are attracting a selection of patients who are less likely to die than the average Medicare FFS enrollee.[29]

Comparing Specifications 2 and 3, we see that a substantial portion of the relative selection into non-drug plans is based on the unobserved severity of illness, ν_{tc}. In particular, an examination of the coefficients suggests that non-drug M+C enrollees are in significantly better health than drug M+C enrollees, and that this adverse selection is based on different levels of the unobserved severity of illness, ν_{tc}. We verify this pattern by performing simple regressions using fitted values of ν_{tc} from the base specification. An over-65 population weighted regression of d_{tc} on ν_{tc} and year dummies yielded a positive coefficient on ν_{tc} with a t-statistic of 2.50, while a similar regression of n_{tc} on ν_{tc} yielded a negative coefficient with a t-statistic of –4.29.[30]

Recall that our model has two potential sources of selection, enrollees and HMOs. The signs of the correlations noted above suggest that enrollees with high unobserved illness severity disproportionately select into plans offering drug coverage and those with low unobserved illness severity disproportionately select into non-drug plans, which is the demand response that we might expect. Any HMO response to unobserved illness severity is likely to be the opposite, as there is likely a complementarity between illness severity and the cost of prescription drug treatment. Thus, the results imply that any HMO response to unobserved illness severity is dominated by the consumer response. This is consistent with the fact that information about enrollee health shocks may take longer to filter to

[28] The inputs into the mortality formula for hypertension and diabetes are available from the authors upon request.

[29] Consistent with this, Town and Liu (2003) show that M+C enrollees have lower medical costs than Medicare FFS enrollees.

[30] Lustig (2009) estimates a structural model of adverse selection in the Medicare Advantage market. He also finds significant adverse selection into Medicare HMOs.

firms than to the enrollees themselves and the fact that HMOs must make decisions annually, while enrollees can make decisions monthly.

Although adverse selection was present in the market for M+C drug plans, it did not cause this market to collapse. For instance, 77% of M+C enrollees in our sample were enrolled in drug plans, a figure that was increasing over time. Moreover, in 2000, 78% of M+C enrollees in plans without drug coverage had at least one plan with drug coverage in their county. This contrasts with other situations where plans with generous benefits have gone into a death spiral and collapsed after the payer implemented a payment scheme where the generous plans were reimbursed at the same rate as the less generous plans, similar to M+C (see Cutler and Reber, 1998). The likely reasons why the M+C drug market did not collapse include the facts that the majority of plans capped the drug benefit to limit the adverse selection, that M+C plans attracted healthy elderly people on average (which compensated for the fact that drug plans attracted less healthy people than non-drug plans), and that in many counties the government reimbursement during our sample period was generous thereby overcoming incentives induced by adverse selection.

In spite of the fact that the M+C market did not collapse, our results suggest that its equilibrium outcomes may not be optimal. Specifically, there is likely to be an under provision of HMOs offering drug coverage, since the value of these plans is high. There is some evidence that this is occurring in the Medicare HMO market. Among counties/years in our sample with an HMO present, 23.6% had no HMO that offered drug coverage. In addition, the literature has found that non-drug M+C plans offer more benefits than drug plans, and that these non-drug benefits are valued by enrollees.[31] Given that these non-drug benefits and drug benefits are very unlikely to be substitutes, this suggests that the market may sometimes under provide attributes besides drug coverage. Last, though we do not model this, information problems may also cause further market failures. For instance, it is possible that some enrollees without drug coverage did not know that their drug coverage had been dropped.

6. Conclusions

This study examines the impact of the Medicare HMO program and drug coverage on the elderly mortality rate using a fixed-effects IV estimator that is consistent with endogenous selection into managed care plans and drug coverage. We find that enrollment in an HMO that does not provide drug benefits significantly increases mortality, while enrollment in an HMO offering drug

[31] Consistent with this explanation, McBride (1998) finds an inverted U-shape relationship between the payment rate and the provision of M+C non-drug benefits while Town and Liu (2003) find an inverse correlation between the value of M+C non-drug benefits offered by plans and the likelihood they offer drug benefits.

coverage has no significant impact relative to Medicare FFS coverage. This finding is robust across a variety of IV specifications and holds for different age groups of the elderly. The magnitudes of our findings are large but also consistent with the literature. Our findings suggest that the benefits of drug coverage are much greater than the cost.

The likely explanation for our results is that drug benefits cause Medicare enrollees to use more drugs which extends their lives. While we do not have evidence on the potential impact of drug coverage for Medicare FFS patients, we believe it will be similar since the methods of service provision are similar across the two types of plans. Apart from their role in the provision of drug benefits, HMOs appear to have little effect on mortality, although there is weak evidence that their cancer treatment quality may be better than Medicare FFS but that their heart disease treatment quality may be worse than Medicare FFS.

Our results suggest several policy implications. Most directly, they imply that policies to extend drug coverage to the elderly would decrease elderly mortality. Following our sample period, the introduction of Medicare Part D in 2006 significantly expanded drug coverage to the elderly and our results implies that between 2005 and 2006 we should observe a significant drop in the elderly mortality relative to the non-Medicare population. Between 2005 and 2006, the mortality rate for the over-65 population dropped 3% (from .0486 to .0472) while the age 55 to 64 mortality rate declined only 2.2% (from .0091 to .0089). While this simple analysis cannot determine if the introduction of Medicare Part D caused the differential drop in mortality for the Medicare eligible population, the pattern is consistent with our results.

In addition, our results imply that moving Medicare enrollees towards managed care plans is not likely to have any adverse mortality consequences. The passage of the 2003 MMA and 2010 PPACA is evidence that Congress made similar assessments. Our results also imply that there are substantial selection differences between Medicare HMOs and Medicare FFS and, in particular, that M+C non-drug plans tend to attract enrollees with a higher than average unobserved health status. The unobserved nature of the selection also suggests that any attempts by the Medicare administration to better adjust payments for observed patient risk status may not completely eliminate the adverse selection. However, reimbursement schemes that paid plans more to provide drug benefits to enrollees would alleviate this selection problem and potentially help the market function more efficiently.

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