The effect of pharmaceutical innovation on longevity: patient-level evidence from the 1996-2002 Medical Expenditure Panel Survey and Linked Mortality Public-use Files

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Abstract

This study uses patient-level data to analyze the effect of technological change embodied in pharmaceuticals on the longevity of elderly Americans. Previous patient-level studies could not control for important patient attributes such as education, income, and race; they did not provide estimates of the effect of using newer drugs on life expectancy, or of the overall cost-effectiveness of new drugs relative to old drugs; and they were not based on nationally representative samples of individuals. Our data, primarily derived from the Medical Expenditure Panel Survey and the Linked Mortality Public-use Files, enable us to overcome those limitations.

We investigate the effect of the vintage (year of FDA approval) of the prescription drugs used by an individual on his or her survival and medical expenditure, controlling for a number of demographic characteristics and indicators and determinants of health status. When we only control for age, sex, and interview year, we estimate that a one-year increase in drug vintage increases life expectancy by 0.52%. Controlling for a much more extensive set of other attributes (the mean year the person started taking his or her medications, and dummy variables for activity limitations, race, education, family income as a percent of the poverty line, insurance coverage, Census region, BMI, smoking and over 100 medical conditions) has virtually no effect on the estimate of the effect of drug vintage on life expectancy.

Between 1996 and 2003, the mean vintage of prescription drugs increased by 6.6 years. This is estimated to have increased life expectancy of elderly Americans by 0.38 years. This suggests that 63% of the 0.6-year increase in the life expectancy of elderly Americans during 1996-2003 was due to the increase in drug vintage. The 1996-2003 increase in drug vintage is also estimated to have increased annual drug expenditure per elderly American by $194, and annual total medical expenditure per elderly American by $286. This implies that the incremental cost-effectiveness ratio (cost per life-year gained) of pharmaceutical innovation was about $15,000. This estimate of the cost per life-year gained from the use of newer drugs is a small fraction of leading economists' estimates of the value of (willingness to pay for) an additional year of life. It is also consistent with estimates from clinical trials.

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Longevity increase is increasingly recognized by economists to be an important part of economic growth and development.[1] Economists have also recognized for many years that, in the long run, economic "growth…is driven by technological change that arises from intentional [research and development (R&D)] investment decisions made by profit-maximizing agents" (Romer (1990)) and by public organizations such as the National Institutes of Health. In principle, technological change could be either disembodied or embodied in new goods. Solow (1960) hypothesized that most technological change is embodied: to benefit from technological progress, one must use newer, or later vintage,[2] goods and services. Bresnahan and Gordon (1996) argued that "new goods are at the heart of economic progress," and Hercowitz (1998, p. 223) also reached the "conclusion…that 'embodiment' is the main transmission mechanism of technological progress to economic growth."

In this paper, I will use patient-level data to analyze the effect of technological change embodied in pharmaceuticals on the longevity of elderly Americans.[3] The basic approach will be to investigate whether patients using newer drugs in a given year remain alive longer than patients using older drugs, controlling for many important patient characteristics.

Two previous studies have examined the effect of pharmaceutical innovation on longevity using patient-level data. Lichtenberg et al (2009) analyzed medical and pharmacy claims data on elderly patients enrolled in Quebec's provincial health plan (Régie de l'assurance maladie du Québec), during the period 1997-2006. Lichtenberg (2010) analyzed medical and pharmacy claims data from Puerto Rico's Medicaid program during the period 2000-2002. Both studies found that the use of newer medications was associated with a statistically significant mortality risk reduction, relative to older medications.

While these two studies were useful, they were subject to several limitations. First, they controlled for some important patient characteristics, such as age, sex, region, and the presence of various medical conditions, but, since they were based entirely on administrative data, they were unable to control for other characteristics, such as education, income, and race. Second, both studies provided estimates of the effect of using newer drugs on the probability of surviving

[1] See e.g. Nordhaus (2002) and Murphy and Topel (2005).
[2] According to the Merriam Webster dictionary, one definition of vintage is "a period of origin or manufacture (e.g. a piano of 1845 vintage)". http://www.merriam-webster.com/dictionary/vintage
[3] According to the National Science Foundation, the pharmaceutical and medical devices industries are the most research intensive industries in the economy.

a certain period of time,[4] but neither provided estimates of the effect of using newer drugs on mean time till death (life expectancy), or of the overall cost-effectiveness of new drugs relative to old drugs. And third, neither study was based on nationally representative samples of individuals.

The present study will overcome these limitations. The data we will use (primarily derived from the Medical Expenditure Panel Survey and the Linked Mortality Public-use Files) will enable us to control for a larger set of potentially important patient characteristics. We will provide estimates (which we believe to be quite robust and reliable) of the effect of using newer drugs on life expectancy per se, as well as its effect on pharmaceutical and overall medical expenditure. Hence, we can estimate the incremental cost-effectiveness (cost per life-year gained) of using newer drugs. These estimates will be based on a nationally representative sample of elderly U.S. community residents.[5]

General approach

Figure 1 illustrates the general approach we will use. We will investigate the effect of the vintage of the prescription drugs used by an individual on his or her survival and medical expenditure, controlling for a number of demographic characteristics and indicators and determinants of health status. We will do this by estimating models of the following general form:

$$survival_i = \beta\ rx\_vintage_i + \gamma\ Z_i + \varepsilon_i \qquad (1)$$

where

$survival_i$ = a measure based on individual i's survival time (number of years until death)
$vintage_i$ = a measure of the vintage of prescription drugs used by individual i
Z_i = a vector of other attributes of individual i
ε_i = a disturbance

First, I will discuss the measures of survival and appropriate estimation methods. Then I will discuss the measurement of drug vintage. Third, I will describe the other individual attributes I

[4] Lichtenberg (2010) examined whether Puerto Rico Medicaid beneficiaries using newer drugs during January– June 2000 were less likely to die by the end of 2002, conditional on the covariates.
[5] Nursing home residents, which account for about 4% of the elderly population (http://www.cdc.gov/nchs/data/hus/hus09.pdf#105), are not included in our sample.

will control for. Fourth, I will consider why there is likely to be substantial variation in drug vintage, even controlling for all of these attributes.

A. Measures of survival and appropriate estimation methods

Most of the data we will use were obtained from the 1996-2002 waves of the Medical Expenditure Panel Survey (MEPS), a set of large-scale surveys of families and individuals, their medical providers, and employers across the United States. MEPS is the most complete source of data on the cost and use of health care and health insurance coverage. The Household Component (HC) of MEPS provides data from individual households and their members, which is supplemented by data from their medical providers. It collects data from a sample of families and individuals in selected communities across the United States, drawn from a nationally representative subsample of households that participated in the prior year's National Health Interview Survey (conducted by the National Center for Health Statistics). During the household interviews, MEPS collects detailed information for each person in the household on the following: demographic characteristics, health conditions, health status, use of medical services, charges and source of payments, access to care, satisfaction with care, health insurance coverage, income, and employment. For example, MEPS provides data about (including the 11-digit National Drug Codes of) all of the prescription drugs used by a patient during a calendar year.

I am able to track a patient's vital status for up to 10 years after he or she was in the MEPS sample because (1) the set of households selected for each panel of the MEPS HC is a subsample of households participating in the previous year's National Health Interview Survey (NHIS) conducted by the National Center for Health Statistics,[6] and (2) the NHIS Linked Mortality Public-use Files provide mortality follow-up data for the NHIS years 1986-2004 from the date of interview through December 31, 2006.[7] Let

$interview\_date_i$ = the date individual i was interviewed
$death\_date_i$ = the date individual i died
$surv\_time_i$ = $death\_date_i$ - $interview\_date_i$ = the number of years individual i survived after being interviewed

[6] NHIS/MEPS Public Use Person Record Linkage files contain crosswalks that allows data users to merge MEPS full-year public use data files to NHIS person-level public use data files that contain data collected for MEPS respondents in the year prior to their initial year of MEPS participation; see http://www.meps.ahrq.gov/mepsweb/data_stats/more_info_download_data_files.jsp#hc-nhis

[7] http://www.cdc.gov/nchs/data_access/data_linkage/mortality/nhis_linkage_public_use.htm

If individual i did not die by December 31, 2006, his or her death date is unknown—we only know that the death date was or will be after December 31, 2006. Hence, the variable surv_time is right-censored. We will estimate versions of eq. (1), in which the dependent variable is surv_time, using a statistical procedure (the SAS LIFEREG procedure) that fits parametric models to failure time data that can be uncensored, right censored, left censored, or interval censored. I will assume that the number of years the patient lived after being interviewed (or the number of years till death) has the Weibull distribution, one of the most commonly used distributions in failure time analysis. The probability density function of a Weibull random variable *X* is:

$$f(x;\lambda,k) = \begin{cases} \frac{k}{\lambda}\left(\frac{x}{\lambda}\right)^{k-1} e^{-(x/\lambda)^k} & x \geq 0, \\ 0 & x < 0, \end{cases}$$

where $k > 0$ is the *shape parameter* and $\lambda > 0$ is the *scale parameter* of the distribution.[8] The mean of a Weibull random variable can be expressed as $\lambda\,\Gamma(1+(1/k))$ where $\Gamma(z)$ is the Gamma function:[9]

$$\Gamma(z) = \int_0^\infty t^{z-1} e^{-t}\,\mathrm{d}t\,.$$

We assume that the scale parameter λ depends on patient characteristics X as follows: $\lambda = \exp(\beta X)$. Hence $\ln \lambda = \beta X$, and ln(mean survival time) $= \beta X + \ln(\Gamma(1+(1/k)))$. Therefore the estimated coefficient on a patient characteristic X_1 indicates the percentage change in mean survival time attributable to a unit increase in X_1.

Our estimation procedure allows us to use right-censored survival data, but the precision of our estimates will be greater, the larger the fraction of the observations that are not censored. To increase the precision of the estimates, I will analyze people 65 years and older ("the elderly") who were interviewed during 1996-2000. The five-year mortality rate of elderly people is over nine times as high as the five-year mortality rate of nonelderly people (23.3% vs. 2.6%). Choosing 2000 as the final sample year ensures that each individual's vital status can be tracked

[8] The shape parameter is what gives the Weibull distribution its flexibility. By changing the value of the shape parameter, the Weibull distribution can model a wide variety of data. If k = 1, the Weibull distribution is identical to the exponential distribution; if k = 2, the Weibull distribution is identical to the Rayleigh distribution; if k is between 3 and 4 the Weibull distribution approximates the normal distribution. The Weibull distribution approximates the lognormal distribution for several values of k.

[9] See http://en.wikipedia.org/wiki/Weibull_distribution and http://www.engineeredsoftware.com/nasa/weibull.htm.

for a minimum of six years. The survival times of 38% of the people 65 years and older who were interviewed during 1996-2000 are not censored.

Unfortunately, MEPS did not obtain data on two potentially important behavioral risk factors prior to 2000: data on whether or not the individual currently smokes began in 2000, and data on body mass index began in 2001.[10] Therefore, including these variables in the surv_time model is not feasible. However, we can assess whether controlling for these variables affects our estimates of the effect of pharmaceutical innovation on survival by using an alternative measure of survival:

$surv\_3\_year_i$ = 1 if $surv\_time_i \geq 3$ years
= 0 if $surv\_time_i < 3$ years

When the dependent variable of eq. (1) is defined as surv_3_year, the model including the two behavioral risk factors can be estimated using data on people interviewed in 2001 and 2002. Since the dependent variable is binary, we will estimate this model as a probit model.

B. Measurement of drug vintage

MEPS Prescribed Medicines files provide data on all outpatient prescription drugs used by each individual.[11] We identified the active ingredients of all of the medications used by each respondent. We used data provided by the FDA (http://www.fda.gov/Drugs/InformationOnDrugs/ucm079750.htm) to determine the year in which the FDA first approved a product containing that active ingredient. From the Drugs@FDA database, we constructed the following variables:

$ingred\_year_a$ = the year in which the FDA first approved a product containing active ingredient a (a =1,…,800)
$post1975_a$ = 1 if $ingred\_year_a > 1975$
= 0 otherwise
$post1985_a$ = 1 if $ingred\_year_a > 1985$
= 0 otherwise

[10] CDC (2005) provides estimates of smoking-attributable mortality; Flegal et al (2005) provides estimates of the effects of obesity on U.S. mortality.
[11] MEPS does not provide information about provider-administered drugs, e.g. chemotherapy. Provider-administered drugs may account for about 15% of total U.S. drug expenditure.

MEPS provides data on n_rx_{ia}: the total number of medications used by individual i that contained active ingredient a. We combined the MEPS medication data with the FDA ingredient attribute data to construct the following variables characterizing the distribution of medications used by each individual:

rx_year_i = $(\Sigma_a\ n\_rx_{ia}\ ingred\_year_a) / \Sigma_a\ n\_rx_{ia}$ = the (weighted) mean vintage of medications used by individual i

$rx\_post1975\%_i$ = $(\Sigma_a\ n\_rx_{ia}\ post1975_a) / \Sigma_a\ n\_rx_{ia}$ = the fraction of medications used by individual i that contained active ingredients approved after 1975

$rx\_post1985\%_i$ = $(\Sigma_a\ n\_rx_{ia}\ post1985_a) / \Sigma_a\ n\_rx_{ia}$ = the fraction of medications used by individual i that contained active ingredients approved after 1985

C. Other individual attributes

The survival models we estimate will include an extensive set of demographic characteristics and indicators and determinants of health status. The effects of some of these variables on mortality and survival are well documented. For example, life tables published by the CDC (Arias et al (2008)) show how life expectancy depends on age, sex, and race. The life table figures can be used as a benchmark against which we can compare our estimates.

A person's life expectancy also clearly depends on the medical conditions a person has. For example, during 2001-2007, the 5-year relative survival rate of a person with breast cancer was 89.1%, whereas the 5-year relative survival rate of a person with pancreatic cancer was only 5.5%.[12] We will control for the presence or absence of over 100 (2-digit ICD9) medical conditions by including a dummy variable for each condition.[13] There are 4 possible reasons why a person could be defined as having a given medical condition: (1) the person responded affirmatively when specifically asked whether he or she has ever been diagnosed with the condition; (2) the condition was reported by the person as the reason for a particular medical event (hospital stay, outpatient visit, emergency room visit, home health episode, prescribed medication purchase, or medical provider visit); (3) the condition was reported as the reason for one or more episodes of disability days; and (4) the condition was reported by the person as a condition "bothering" the person during the reference period.[14]

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[12] SEER Cancer Statistics Review, 1975-2008, http://seer.cancer.gov/csr/1975_2008/results_merged/topic_survival.pdf

[13] The dummy variables were constructed using data in the MEPS Medical Conditions files.

[14] In some previous studies based on claims data, a person would be considered to have a medical condition only if the diagnosis code for that condition appeared in a medical claim.

A person's life expectancy may depend not just on *whether* he or she has a medical condition, but on *how long* he or she has had the condition. MEPS Medical Conditions files provide some information on the duration of medical conditions (i.e. the date the condition began), but this information is very incomplete. However, MEPS Prescribed Medicines files provide fairly complete information on the date the person started taking each medicine. Mean duration of medication use (or the mean year the person started taking his or her medications (began_med_year)) may serve as a reasonable proxy for mean duration of medical condition. We will include began_med_year as an explanatory variable.

In addition to data on medical conditions and their treatment, MEPS provides data on the functional status and activity limitations of individuals. In particular, MEPS respondents are asked whether or not they experience any limitation in work, housework, or school activities, and if so, whether they are completely unable to perform those activities. We constructed the following measure of activity limitations:

act_lim_i = 2 if person i was completely unable to work at a job, do housework, or go to school
= 1 if person i was limited in these activities but not completely unable to perform them
= 0 if person i was not limited in these activities

One would expect that people whose activities are more limited have lower life expectancy, ceteris paribus. However, if pharmaceutical innovation improves health (and reduces activity limitations), then estimates of β in eq. (1)—the effect of drug vintage on life expectancy—are likely to be conservative when Z includes act_lim. We will present estimates of eq. (1) both excluding and including act_lim.

We also include two measures of socioeconomic status—income and education—in the survival model. Many studies have found a positive correlation between these variables and life expectancy. However, cross-sectional correlations between longevity and either income or education may substantially overestimate the effect of socioeconomic status *per se* on longevity. For example, the positive correlation between income and longevity may reflect the effect of health on income ("reverse causality") as well as the effect of income on health. Almond and Mazumder (2006) argue that, "although it is well known that there is a strong association

between education and health, much less is known about how these factors are connected, and whether the relationship is causal."[15]

D. Why does drug vintage vary, controlling for these individual attributes?

We believe that heterogeneous pharmaceutical treatment of patients, controlling for their medical conditions, demographic characteristics, insurance coverage, and other factors, is primarily due to physician practice variation. Wennberg (2004) argues that "unwarranted [treatment] variation—variation not explained by illness, patient preference, or the dictates of evidence-based medicine—is a ubiquitous feature of U.S. health care." A large number of studies have documented the importance of unexplained variation in medical care in general and prescribing behavior in particular. Wennberg and Wennberg examined variation in nine drugs or classes of drugs among members of Blue Cross and Blue Shield of Michigan, and argued that "evidence does not, per se, ensure that pharmaceuticals are always used rationally." Lee et al (2008) showed that "pediatric and adult transplant physicians differed significantly in their management strategies for chronic myeloid leukemia, acute and chronic graft-versus-host disease, and choice of graft source for patients with aplastic anemia. Among adult transplant physicians, there was little agreement on the patient factors favoring reduced intensity conditioning or myeloablative conditioning." DeSalvo et al (2000) reported "wide variation…in assignment of reappointment interval with mean return intervals…ranging from 2.2 to 20.5 weeks. Sex was a significant provider independent variable…Female providers assigned earlier reappointment intervals for their patients." Solomon et al (2003) found that "established risk factors for NSAID-associated gastrointestinal toxicity were poor predictors of who was prescribed a selective COX-2 inhibitor; in contrast, physician prescribing preference was an important determinant." De Las Cuevas et al (2002) showed that "there is a remarkable degree of variation in antidepressant prescribing by psychiatrists and general practitioners; this is due to

[15] Lleras-Muney (2005) provided perhaps the strongest evidence that education has a causal effect on health. Using state compulsory school laws as instruments, Lleras-Muney found large effects of education on mortality. Almond and Mazumder (2006) revisited these results, noting they were not robust to state time trends, even when the sample was vastly expanded and a coding error rectified. They employed a dataset containing a broad array of health outcomes and found that when using the same instruments, the pattern of effects for specific health conditions appeared to depart markedly from prominent theories of how education should affect health. They also found suggestive evidence that vaccination against smallpox for school age children may account for some of the improvement in health and its association with education. This raised concerns about using compulsory schooling laws to identify the causal effects of education on health.

economic and social factors as much as to morbidity differences." Rochon et al (2007) found that "residents in facilities with high antipsychotic prescribing rates were about 3 times more likely than those in facilities with low prescribing rates to be dispensed an antipsychotic agent, irrespective of their clinical indication."[16] Zink et al (2001) found that "trends in the drug management of [rheumatoid arthritis] are adopted differentially by the members of the rheumatology community." Davis and Gribben (1995) found that "data from a survey of general practice in New Zealand confirm the existence of extensive variability in prescribing. Controlling for patient, diagnostic, and practitioner variables…does not reduce the extent of interpractitioner variability in prescribing rates." Moreover, de Jong et al (2009) found that decision support systems do not reduce variation in prescribing.

We will assess the extent of prescribing practice variation in our sample of individuals by estimating the following equation:

$$rx\_vintage_i = \pi Z_i + \varepsilon_i \qquad (2)$$

The R^2 of this equation indicates the fraction of the variance in prescription drug vintage that is explained by variation in medical conditions and other individual attributes. A low R^2 would be indicative of substantial practice variation.

Descriptive statistics

Now I will present some descriptive statistics. Following the sequence of the previous section, I will first present statistics about life expectancy. Next, I will present statistics about the vintage of prescription drugs. Third, I will present statistics about other patient attributes. Finally, I will present estimates of eq. (2), to assess the extent of prescribing practice variation in our sample.

[16] Using clinical and administrative data obtained from all facilities in a Department of Veterans Affairs integrated service network, Krein et al (2002) showed that there was variation in diabetes practice patterns at the primary care provider, provider group, and facility levels, and that the greatest amount of variance tended to be attributable to the facility level.

Life expectancy. Our sample consists of people age 65 and over interviewed in the MEPS during 1996-2000 who were eligible for mortality follow-up[17] and who had at least one prescription drug[18] during the interview year. The following table shows some sample statistics.

Sex	Number of observations	Mean age	Mean life expectancy based on 1999-2001 CDC life table[19]
Both	5230	74.3	11.9
Male	2084	73.6	11.0
Female	3146	74.8	12.5

We estimated survival functions for both sexes combined and separately by sex using the right-censored surv_time observations, and no explanatory variables (only an intercept). This provided the following estimates of the Weibull shape and scale parameters (k and λ) and of mean survival time ($\lambda\,\Gamma(1+(1/k))$.

Sex	λ	k	$\lambda\,\Gamma(1+(1/k))$
Both	14.7	1.2	13.7
Male	13.6	1.2	12.8
Female	15.4	1.3	14.3

Mean survival time computed from the right-censored surv_time observations is about 15% higher than mean life expectancy based on the 1999-2001 CDC life table. We can think of two possible reasons for this. One is that CDC life-table estimates of life expectancy are based on nursing-home residents as well as community residents, whereas our sample only includes community residents, who are certainly healthier. A second, less important reason is that the CDC life table is a period life table, whereas the parameter estimates are, in effect, estimates of the cohort life table.[20]

The life-table and parametric estimates of the percentage differential between female and male life expectancy are quite similar: 13.5% and 11.5%. Overall, our statistical procedure seems to yield plausible estimates of the survival distribution.

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[17] Less than half of MEPS respondents were eligible for mortality follow-up. See http://www.cdc.gov/nchs/data/datalinkage/nhis_frequency_of_selected_variables_public_2010.pdf.

[18] In 2000, 88% of elderly MEPS respondents had at least one prescription drug during the year.

[19] http://www.cdc.gov/nchs/data/nvsr/nvsr57/nvsr57_01.pdf

[20] The Social Security Administration publishes both period and cohort U.S. life tables (http://www.ssa.gov/oact/NOTES/as120/LifeTables_Body.html). The estimate of life expectancy of 70-year-olds in 2000 from the (1930 birth) cohort life table is higher than the estimate of life expectancy of 70-year-olds in 2000 from the period life table, but only about 2% higher.

Drug vintage. Statistics describing the vintage of prescription drugs used by elderly MEPS respondents during the period 1996-2008 are shown in Table 1.[21] Not surprisingly, drug vintage increased over time. rx_post1975% (the fraction of medications that contained active ingredients approved after 1975) increased from 57% in 1996 to 75% in 2008, and rx_post1985% increased from 26% in 1996 to 57% in 2008. rx_year (the weighted mean FDA approval year) increased by 8 years during this period.[22] However, the average annual rate of increase was three times as high from 1996 to 2003 as it was from 2003 to 2008 (0.9 vs. 0.3 years/year). The post-1995 decline in the number of new drugs approved by the FDA, illustrated in Figure 2, probably contributed to the decline in the rate of increase of drug vintage.

Other variables. Frequency distributions of many of the individual attributes shown in Figure 1 are presented in Table 2.

Prescribing practice variation. To assess the degree of unexplained variability in prescription drug use, we estimated eq. (2): a regression of rx_year on a large set of individual attributes: dummy variables for interview year, age, sex, race, marital status, Census region, insurance coverage, education, income (poverty group category), and presence of 100+ medical conditions. Table 3 shows the Type III (marginal) sums of squares for all factors except the medical conditions. Several factors (interview year, region, sex, and education) are statistically significant. The significance of interview year is not surprising, since we already saw in Table 1 than drug vintage tends to increase over time. The estimates in Table 3 indicate that drugs used in the western U.S. are about 2 years older than drugs used in the rest of the country; drugs used by women are about a year older than drugs used by men; and that highly educated people tend to use newer drugs than less educated people. Drug vintage is not significantly related to several important individual attributes, including insurance coverage, race, and family income as a percent of the poverty line. The R^2 of this regression is only 12%, which indicates that only 1/8 of the variation in drug vintage is explained by the presence of 100+ medical conditions and key demographic and socioeconomic variables. This is consistent with the literature documenting substantial variation in prescribing and drug use.

[21] These statistics describe the vintage of prescription drugs used by all elderly MEPS respondents, including those not eligible for mortality follow-up.

[22] In 1996, the mean age (number of years since FDA approval) of drugs consumed was 20.1 years; in 2008, it was 24.1 years.

Empirical results

Estimates of three versions of eq. (1) are shown in Table 4. In all three versions, the dependent variable is the right-censored variable surv_time (the number of years individual i survived after being interviewed), and the measure of drug vintage is rx_year (the (weighted) mean vintage of medications used by individual i). The three models differ in terms of the other attributes (Z) that are included. In model 1, the only other attributes included are dummy variables for age, sex, and interview year. In this model, the rx_year coefficient (β) is positive and highly significant (p-value = .001); the point estimate (.0052) indicates that a one-year increase in drug vintage increases life expectancy by 0.52%. Estimates of the other parameters seem reasonable: life expectancy declines with age, is higher for women than for men, and was lower in 1996-1997 than it was in 1998-2000.

Model 2 includes a much more extensive set of other attributes: began_med_year (the mean year the person started taking his or her medications, which may serve as a proxy for mean duration of medical conditions); race; education; family income as a percent of the poverty line; insurance coverage; Census region; and over 100 medical condition dummy variables (whose coefficients are not shown to conserve space). The coefficient on began_med_year is not statistically significant. Also, the difference in life expectancy between people with and without private health insurance is not significant. Regional differences are also insignificant. The race coefficients indicate that the life expectancy of blacks is 9% lower than that of whites (p-value = .078), and that the life expectancy of Asians is 39% higher than that of whites (p-value = .022, despite the fact that only 1.4% of sample members are Asians). The income and education group variables indicate that, overall, people in higher income categories and with more education have longer life expectancy. However, controlling for this much more extensive set of other attributes has virtually no effect on the estimate of β; indeed, the drug vintage coefficient is about 10% *larger* in model 2 than it is in model 1.

Model 3 includes one additional individual attribute: act_lim, the index of activity limitations. We argued above that estimates of the effect of drug vintage on life expectancy are likely to be conservative when we control for act_lim. As one would expect, the life expectancy of people with activity limitations is lower than that of people without activity limitations.

Controlling for activity limitations reduces the estimate of β, but only slightly (β = .0052, p-value = .003 than in model 3).

We also estimated two models (models 4 and 5) similar to model 3 in which we replaced the drug vintage measure rx_year by one of the alternative measures (rx_post1975% or rx_post1985%). The estimates of the drug vintage coefficients in these models are:

Model	rx_vintage measure	Estimate	StdErr	ChiSq	ProbChiSq
4	rx_post1975%	0.177	0.050	12.4	0.000
5	rx_post1985%	0.112	0.056	4.0	0.045

The coefficient on rx_post1975% is more significant than the coefficient on rx_post1985%, but both coefficients are positive and statistically significant (p-value < .05). Since the choice of new-drug/old-drug threshold year (e.g. 1975 or 1985) is arbitrary, and rx_year (based on the continuous ingredient vintage measure ingred_year) presumably conveys more information than the other two measures (based on the discrete ingredient vintage measures post1975 and post1985), the remainder of our analysis will be based on the rx_year measure of drug vintage.

The models of survival time shown in Table 4 did not include two potentially important behavioral risk factors—BMI and smoking—as covariates because, as discussed earlier, data on whether or not the individual currently smokes began in 2000, and data on body mass index began in 2001. However, we can assess whether controlling for these variables affects our estimates of the effect of pharmaceutical innovation on survival by estimating a model of an alternative measure of survival—the 3-year survival rate—using data on people interviewed in 2001 and 2002. Table 5 shows estimates of two probit models of the 3-year survival rate. Both models include all of the individual attributes included in model 3. Model 6 does not include controls for BMI and smoking. In this model, the coefficient on rx_year is positive and significant (p-value = .038), indicating that drug vintage had a positive effect on the 3-year survival rate. Model 7 includes controls for smoking and BMI.[23] The coefficient on current_smoker is negative but not significant. The BMI dummy variables are jointly significant (p-value = 0.0003), but, contrary to expectations, they indicate that overweight and obese people

[23] 11.1% of respondents were current smokers. The BMI distribution is: underweight (BMI < 19) 4.1%; healthy weight (19 ≤ BMI < 25) 34.9%; overweight (25 ≤ BMI < 30) 36.4%; obese (30 ≤ BMI) 24.7%.

had *higher* survival rates than people with healthy weight. However, controlling for the two behavioral risk factors has virtually no effect on the drug vintage coefficient.

To summarize, when we only controlled for age, sex, interview year, we estimated that a one-year increase in drug vintage increases life expectancy by 0.52%. Controlling for a much more extensive set of other attributes (the mean year the person started taking his or her medications, and dummy variables for activity limitations, race, education, family income as a percent of the poverty line, insurance coverage, Census region, BMI, smoking and over 100 medical condition dummy variables) had virtually no effect on the estimate of β.

As shown in Table 1, between 1996 and 2003, the mean value of rx_year increased by 6.6 years, from 1975.9 to 1982.5. The estimate of β (.0052) from model 3 in Table 4 implies that the 1996-2003 increase in mean vintage increased life expectancy of elderly community residents by 3.4% (= .0052 * 6.6). According to CDC life tables, life expectancy at age 75 increased from 11.1 years in 1996 to 11.7 years in 2003, so the 1996-2003 increase in mean vintage increased life expectancy of elderly community residents by not less than 0.38 years (= 3.4% * 11.1 years). This suggests that 63% of the 0.6-year increase in the life expectancy of elderly Americans during 1996-2003 was due to the increase in drug vintage.

Since new drugs tend to be more expensive than old drugs, the increase in drug vintage is likely to have increased pharmaceutical expenditure, and may have increased total medical expenditure as well. As indicated in Figure 1, we can assess the impact of pharmaceutical innovation on medical expenditure by estimating models similar to eq. (1), in which the dependent variable is a measure of medical expenditure rather than a survival measure. We estimated models similar to model 3 in Table 4, in which the dependent variable was either $\ln(rx\_expend_i)$ or $\ln(tot\_expend_i)$, where

rx_expend_i = prescription drug expenditure by (or on behalf of) individual i
tot_expend_i = total medical expenditure by (or on behalf of) individual i

The estimated coefficients on rx_year in these two models were:

Model	Dependent	Parameter	Estimate	StdErr	tValue	Probt
6	$\ln(rx\_expend_i)$	rx_year	0.0294	0.0014	20.46	0.0000
7	$\ln(tot\_expend_i)$	rx_year	0.0068	0.0015	4.47	0.0000

These estimates imply that a 1-year increase in drug vintage increases pharmaceutical expenditure by 2.9%, and total medical expenditure by 0.7%. The sample mean values of rx_expend and tot_expend were $1000 and $6352, respectively. Therefore, the 6.6-year increase in drug vintage that occurred between 1996 and 2003 is estimated to have increased annual drug expenditure per elderly American by $194 (= 6.6 * 2.9% * $1000), and annual total medical expenditure per elderly American by $286 (= 6.6 * 0.7% * $6352).

We can use these estimates to calculate the incremental cost-effectiveness ratio (ICER) of pharmaceutical innovation, defined as follows:

$$\text{ICER} = \frac{\Delta \text{ lifetime medical expenditure}}{\Delta \text{ life expectancy}} = \frac{\Delta \text{ lifetime medical expenditure} / \Delta \text{ rx\_year}}{\Delta \text{ life expectancy} / \Delta \text{ rx\_year}}$$

The effect of the 1996-2003 increase in drug vintage on (undiscounted) lifetime medical expenditure is calculated in the following table.

	Life expectancy (1)	Annual medical expenditure (2)	Lifetime medical expenditure (1) * (2)
Baseline	11.10	$6,352	$70,512
Baseline + effect of 6.6-year increase in drug vintage	11.48	$6,639	$76,207
Effect of 6.6-year increase in drug vintage	0.38	$286	$5,695

The use of newer drugs is estimated to have increased lifetime medical expenditure by $5695, and life expectancy by 0.38 years. Therefore the ICER (cost per life-year gained) is estimated to be $15,008 (= $5695 / 0.38).

This rough assessment of the overall cost-effectiveness of pharmaceutical innovation may be compared to evidence from clinical trials as reported in the CEA Registry[24], a comprehensive database of cost-utility analyses on a wide variety of diseases and treatments. A search of the registry found (1) 545 pharmaceutical interventions that decreased cost and improved health (in which case the ICER is negative); (2) 771 pharmaceutical interventions that increased cost and improved health at a cost of less than $16,173 per QALY; and (3) 1481 pharmaceutical interventions that increased cost and improved health at a cost of more than $16,173 per QALY.

[24] The CEA Registry (https://research.tufts-nemc.org/cear4/) is produced by the Center for the Evaluation of Value and Risk in Health, part of the Institute for Clinical Research and Health Policy Studies at Tufts Medical Center in Boston, MA.

Therefore, our estimate of the ICER is not very far from the median of the estimates reported in the CEA Registry.

Summary

This study used patient-level data to analyze the effect of technological change embodied in pharmaceuticals on the longevity of elderly Americans. Previous patient-level studies could not control for important patient attributes such as education, income, and race; they did not provide estimates of the effect of using newer drugs on life expectancy, or of the overall cost-effectiveness of new drugs relative to old drugs; and they were not based on nationally representative samples of individuals. Our data, primarily derived from the Medical Expenditure Panel Survey and the Linked Mortality Public-use Files, enabled us to overcome those limitations.

We investigated the effect of the vintage (year of FDA approval) of the prescription drugs used by an individual on his or her survival and medical expenditure, controlling for a number of demographic characteristics and indicators and determinants of health status. When we only controlled for age, sex, and interview year, we estimated that a one-year increase in drug vintage increases life expectancy by 0.52%. Controlling for a much more extensive set of other attributes (the mean year the person started taking his or her medications, and dummy variables for activity limitations, race, education, family income as a percent of the poverty line, insurance coverage, Census region, BMI, smoking and over 100 medical conditions) had virtually no effect on the estimate of the effect of drug vintage on life expectancy.

Between 1996 and 2003, the mean vintage of prescription drugs increased by 6.6 years. This is estimated to have increased life expectancy by 0.38 years. This suggests that 63% of the 0.6-year increase in the life expectancy of elderly Americans during 1996-2003 was due to the increase in drug vintage. The 1996-2003 increase in drug vintage is also estimated to have increased annual drug expenditure per elderly American by $194, and annual total medical expenditure per elderly American by $286. This implies that the incremental cost-effectiveness ratio (cost per life-year gained) of pharmaceutical innovation was about $15,000. This estimate of the cost per life-year gained from the use of newer drugs is a small fraction of leading

economists' estimates of the value of (willingness to pay for) an additional year of life. It is also consistent with estimates from clinical trials.

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Figure 1
General approach

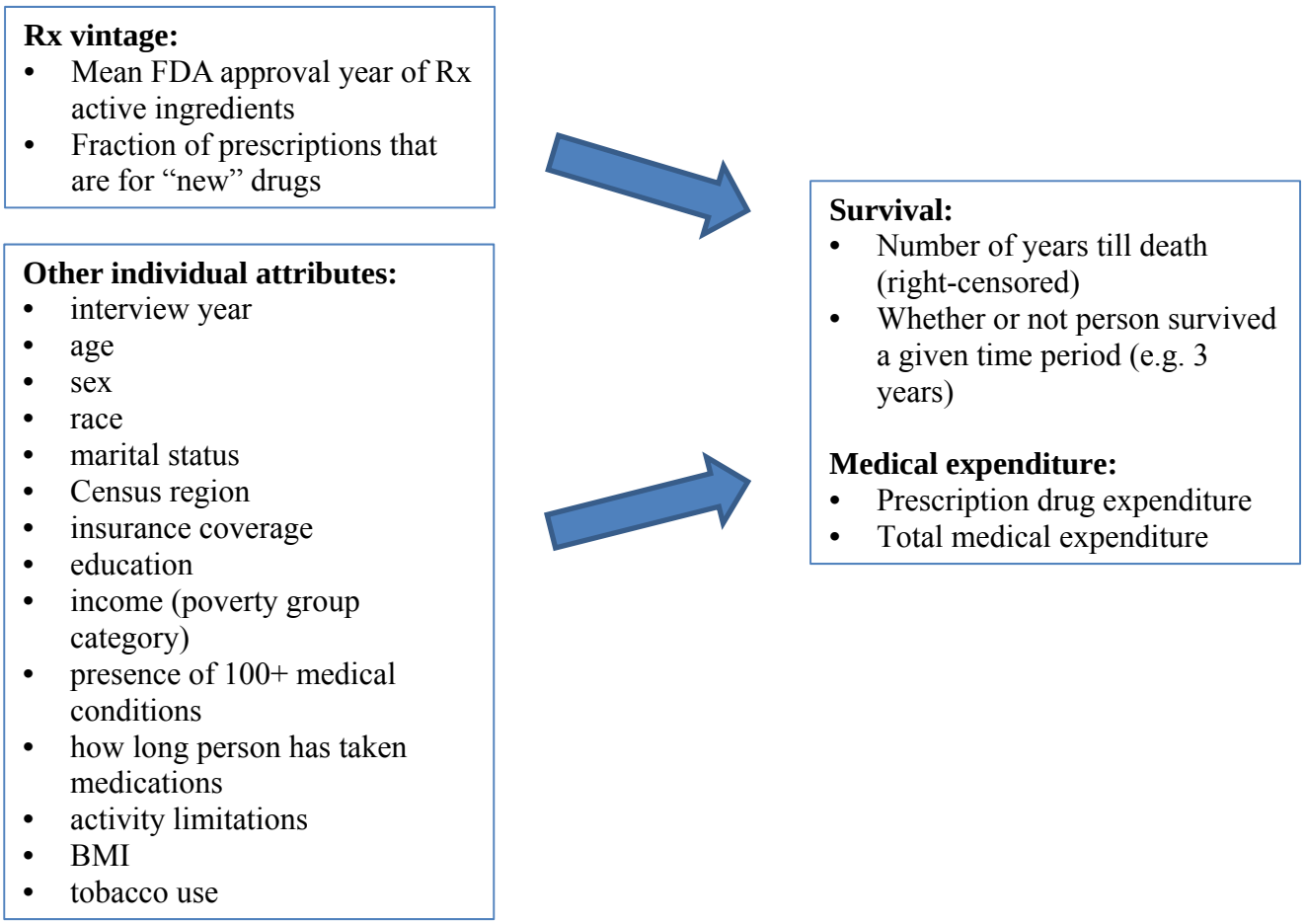

Figure 2

Pharmaceutical research expenditure and new pharmaceuticals approved by the FDA, 1995-2010

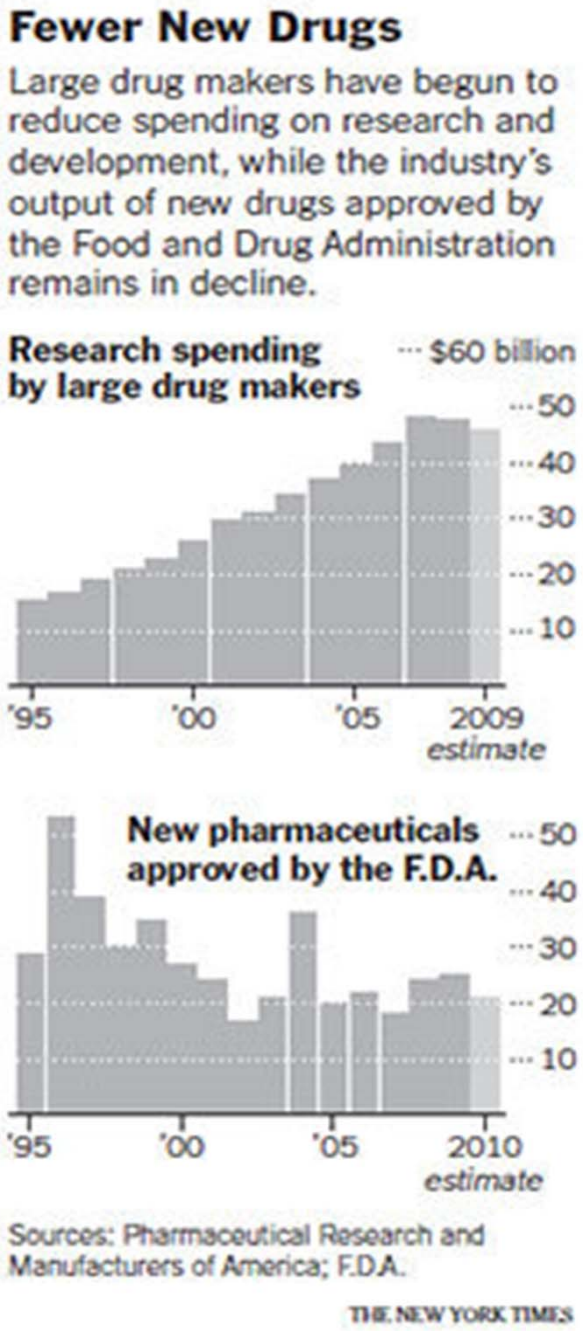

Table 1

Vintage of prescription drugs used by elderly MEPS respondents, 1996-2008

year	Number of prescriptions	rx_year	rx_post1975%	rx_post1985%
1996	38,902	1975.9	57%	26%
1997	64,220	1976.4	58%	30%
1998	46,019	1977.2	60%	34%
1999	45,691	1978.5	63%	38%
2000	47,828	1979.5	65%	42%
2001	68,829	1980.6	67%	46%
2002	82,460	1981.6	69%	49%
2003	69,150	1982.5	71%	52%
2004	81,502	1982.4	70%	52%
2005	92,149	1983.2	73%	54%
2006	106,273	1983.5	74%	54%
2007	89,989	1983.9	74%	56%
2008	82,851	1983.9	75%	57%

Table 2

Frequencies of individual characteristics

Age	
65-69	28.4%
70-74	27.0%
75-79	21.5%
80-84	14.0%
85-89	6.7%
90+	2.5%
Interview year	
1996	31.7%
1997	19.2%
1998	15.4%
1999	18.7%
2000	15.0%
Race	
WHITE	85.6%
BLACK	12.5%
ASIAN OR PACIFIC ISLANDER	1.4%
AMERICAN INDIAN	0.5%
ALEUT, ESKIMO	0.0%
Family income as % of poverty line	
POOR/NEGATIVE	16.0%
NEAR POOR	7.4%
LOW INCOME	19.8%
MIDDLE INCOME	28.1%
HIGH INCOME	28.7%
Activity limitation	
able to do activity	75.6%
somewhat unable to do activity	9.1%
completely unable to do activity	15.3%
Insurance coverage	
ANY PRIVATE	57.7%
PUBLIC ONLY	42.1%
UNINSURED	0.2%
Education	
ELEMENTARY GRADES 1 - 8	24.7%
HIGH SCHOOL GRADES 9 - 11	17.5%
GRADE 12	30.8%
1-3 YEARS COLLEGE	13.6%
4 YEARS COLLEGE	7.7%
5+ YEARS COLLEGE	5.6%
Marital status	
MARRIED IN ROUND	53.5%
WIDOWED IN ROUND	34.6%
DIVORCED	5.3%
NEVER MARRIED	3.9%
DIVORCED IN ROUND	1.5%
SEPARATED	0.9%
SEPARATED IN ROUND	0.4%
Region	
NORTHEAST	20.2%
MIDWEST	22.4%
SOUTH	37.3%
WEST	20.1%

Table 3

Estimates of drug vintage model (eq. (2)): $rx\_vintage_i = \pi Z_i + \varepsilon_i$

Type III (marginal) sums of squares for all factors except the medical conditions

Source	DF	SS	MS	FValue	ProbF
year	4	10176.16	2544.041	28.49705	1.74E-23
region	3	3079.03	1026.343	11.49657	1.64E-07
SEX	1	1121.655	1121.655	12.56421	0.000396
educyr	5	1137.461	227.4922	2.548251	0.026038
race2	4	609.3991	152.3498	1.706545	0.145522
age	5	276.7843	55.35686	0.620079	0.684515
POVCAT	4	146.2116	36.55291	0.409447	0.801976
marry	6	157.6191	26.26985	0.294261	0.939921
inscov	2	7.682661	3.84133	0.043029	0.957884

Selected parameter estimates

Parameter		Estimate	StdErr	tValue	Probt
year	1996	-3.69891	0.3916	-9.44563	4.98E-21
year	1997	-3.57032	0.428951	-8.32338	1.05E-16
year	1998	-2.40202	0.450419	-5.33287	1E-07
year	1999	-1.51924	0.428207	-3.54791	0.000391
year	2000	0			
region	MIDWEST	1.864643	0.386697	4.821978	1.46E-06
region	NORTHEAST	2.054372	0.397907	5.162941	2.51E-07
region	SOUTH	1.695443	0.353876	4.791065	1.7E-06
region	WEST	0			
SEX	Female	-1.07884	0.30436	-3.5446	0.000396
SEX	Male	0			
educyr	00 - 08 ELEMENTARY GRADES 1 - 8	-0.21074	0.616176	-0.34201	0.732359
educyr	09 - 11 HIGH SCHOOL GRADES 9 - 11	-0.70005	0.621605	-1.1262	0.260127
educyr	12 GRADE 12	-0.16585	0.581327	-0.28529	0.775432
educyr	13-15 1-3 YEARS COLLEGE	-0.72332	0.625526	-1.15635	0.247587
educyr	16 4 YEARS COLLEGE	1.040067	0.685669	1.516866	0.129355
educyr	17 5+ YEARS COLLEGE	0			

Table 4: Estimates of survival model (eq. 1) using right-censored data

		Model 1				Model 2				Model 3			
Parameter	Level1	Estimate	StdErr	ChiSq	ProbChiSq	Estimate	StdErr	ChiSq	ProbChiSq	Estimate	StdErr	ChiSq	ProbChiSq
rx_year		0.0052	0.002	10.5	0.001	0.0058	0.002	11.0	0.001	0.0052	0.002	8.7	0.003
age	65-69	1.6628	0.087	364.5	0.000	1.4534	0.090	260.6	0.000	1.3707	0.090	230.5	0.000
age	70-74	1.3560	0.083	264.0	0.000	1.1917	0.086	193.9	0.000	1.1032	0.086	164.3	0.000
age	75-79	0.9759	0.081	145.0	0.000	0.8792	0.083	113.3	0.000	0.8002	0.083	92.7	0.000
age	80-84	0.6113	0.081	56.9	0.000	0.5439	0.081	44.8	0.000	0.4777	0.082	34.3	0.000
age	85-89	0.3829	0.087	19.2	0.000	0.3729	0.087	18.5	0.000	0.3093	0.087	12.6	0.000
age	90+	0.0000				0.0000				0.0000			
SEX	Female	0.2788	0.033	71.2	0.000	0.3399	0.038	78.1	0.000	0.3319	0.038	75.4	0.000
SEX	Male	0.0000				0.0000				0.0000			
year	1996	-0.1009	0.057	3.1	0.078	-0.1510	0.057	6.9	0.009	-0.1281	0.057	5.0	0.025
year	1997	-0.1470	0.061	5.8	0.016	-0.1140	0.060	3.6	0.058	-0.0748	0.060	1.6	0.213
year	1998	0.0314	0.066	0.2	0.637	0.0365	0.065	0.3	0.576	0.0571	0.065	0.8	0.379
year	1999	0.0316	0.065	0.2	0.627	0.0235	0.063	0.1	0.708	0.0389	0.063	0.4	0.535
year	2000	0.0000				0.0000				0.0000			
began_med_yea						-0.0004	0.003	0.0	0.890	-0.0002	0.003	0.0	0.956
act_lim	0 able to do activity									0.3339	0.043	61.0	0.000
act_lim	1 somewhat unable to do activity									0.0878	0.056	2.5	0.114
act_lim	2 completely unable to do activity									0.0000			
race	ALEUT, ESKIMO					14.5886	14093.131	0.0	0.999	14.4703	14185.711	0.0	0.999
race	AMERICAN INDIAN					-0.0118	0.206	0.0	0.954	-0.0262	0.207	0.0	0.899
race	ASIAN OR PACIFIC ISLANDER					0.3894	0.170	5.2	0.022	0.3770	0.169	5.0	0.026
race	BLACK					-0.0854	0.048	3.1	0.078	-0.0790	0.048	2.7	0.103
race	WHITE					0.0000				0.0000			
income group	POOR/NEGATIVE					-0.0555	0.053	1.1	0.294	-0.0292	0.053	0.3	0.581
income group	NEAR POOR					-0.1497	0.063	5.7	0.017	-0.1402	0.062	5.1	0.024
income group	LOW INCOME					-0.0624	0.048	1.7	0.198	-0.0530	0.048	1.2	0.273
income group	MIDDLE INCOME					-0.0005	0.045	0.0	0.990	-0.0065	0.045	0.0	0.885
income group	HIGH INCOME					0.0000				0.0000			
insurance	ANY PRIVATE					-0.8202	0.723	1.3	0.257	-0.8491	0.720	1.4	0.238
insurance	PUBLIC ONLY					-0.8752	0.723	1.5	0.226	-0.8823	0.720	1.5	0.220
insurance	UNINSURED					0.0000				0.0000			
education	00 - 08 ELEMENTARY GRADES 1 - 8					-0.2022	0.089	5.2	0.023	-0.1585	0.088	3.2	0.072
education	09 - 11 HIGH SCHOOL GRADES 9 - 11					-0.2909	0.090	10.5	0.001	-0.2554	0.089	8.2	0.004
education	12 GRADE 12					-0.2097	0.087	5.9	0.015	-0.1850	0.086	4.6	0.031
education	13-15 1-3 YEARS COLLEGE					-0.1427	0.092	2.4	0.122	-0.1343	0.092	2.1	0.143
education	16 4 YEARS COLLEGE					-0.1072	0.101	1.1	0.287	-0.0952	0.100	0.9	0.343
education	17 5+ YEARS COLLEGE					0.0000				0.0000			
region	MIDWEST					0.0367	0.049	0.6	0.450	0.0325	0.048	0.5	0.502
region	NORTHEAST					0.0275	0.051	0.3	0.589	0.0177	0.051	0.1	0.727
region	SOUTH					-0.0025	0.045	0.0	0.956	0.0050	0.045	0.0	0.910
region	WEST					0.0000				0.0000			
100+ medical conditon dummies´		No				Yes				Yes			

Table 5

Estimates of probit models of the 3-year survival rate based on data for 2001 and 2002

		Model 6					Model 7			
Parameter	Level1	Estimate	StdErr	ChiSq	ProbChiSq		Estimate	StdErr	ChiSq	ProbChiSq
rx_year		0.008	0.004	4.3	0.038		0.008	0.004	4.3	0.038
current_smoker							-0.071	0.121	0.3	0.557
bmi	0 underweight						-0.297	0.185	2.6	0.108
bmi	1 healthy weight						-0.301	0.104	8.3	0.004
bmi	2 overweight						0.068	0.107	0.4	0.524
bmi	3 obese						0.000			

Both models include began_med_year (the mean year the person started taking his or her medications) and dummy variables for act_lim (the index of activity limitations), age, sex, interview year, race, education, family income as a percent of the poverty line, insurance coverage, Census region, and over 100 medical condition dummy variables

The estimates are based on 2805 observations: 2480 people survived; 325 people did not survive 3 years.