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The life expectancy gains from pharmaceutical drugs: a critical appraisal of the literature [#]

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Abstract: Several studies suggest that, on the basis of life expectancy (LE) regressions, new pharmaceutical drugs are responsible for some of the marked gains in LE observed over the last 50 years. We critically appraise these studies. We point out several modeling issues, including disentangling the contribution of new drugs from advances in disease management, changes in the distribution of health care and other confounding factors. We suggest that the studies estimates of pharmaceutical productivity are implausibly high. Some of the models have very large forecast errors. Finally, the models that we replicated were found to be sensitive to seemingly innocuous changes in specification. We conclude that it is difficult to estimate the bio-medical determinants of LE using aggregate data. Analyses using individual level data or perhaps disease specific data will likely produce more compelling results.

JEL Classifications: I12, I18
Keywords: pharmaceuticals, life expectancy, health production, treatment effects

Résumé: Plusieurs études suggèrent, en se basant sur des analyses multivariées de l'espérance de vie (EV), que l'arrivée de nouveaux produits pharmaceutiques soit responsable des gains de l'EV observés au cours des cinquante dernières années. Nous évaluons ces études en portant un regard critique. Nous avons mis en évidence plusieurs problèmes de modélisation. Par exemple, nous tentons de démêler la contribution liée à l'arrivée de nouveaux médicaments, de celle liée aux progrès en matière de gestion de la maladie, aux changements de la répartition des soins de santé ainsi que d'autres facteurs de confusion. Nous pensons que les estimations élevées de la productivité pharmaceutique présentées dans ces études sont fort peu plausibles. Certains modèles souffrent d'erreurs de prédiction importantes. Finalement, les modèles que nous avons répliqués sont sensibles à des variations en apparence anodines de leurs spécifications. Nous arrivons à la conclusion qu'il est difficile d'évaluer les déterminants bio-médicaux de l'EV en s'appuyant sur des données agrégées. Des analyses reposant sur des données individuelles ou des données particulières à une maladie vont sûrement produire des résultats plus convaincants.

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Introduction

Life expectancy (LE) gains in many developed countries since WWII have been dramatic. In Canada, for instance, LE at birth increased by about 11 years over the period 1950-2002, from 70.8 to 82.1 years for females and from 66.3 to 77.2 years for males (Beaudet, Tully and St-Arnaud 2005). What has caused these recent LE increases? Cutler, Deaton and Lleras-Muney (2006) point to advances in medical technology. There is a growing body of evidence that new pharmaceutical drugs, in particular, are responsible for some of this LE increase. This evidence comes from several sources: (i) clinical trials of new drugs, (ii) studies of trends in disease prevalence, and (iii) econometric LE models. As examples: (i) clinical trials indicate that drugs can help prevent heart attack, stroke and heart failure, which are among the leading causes of death and disability (Welsfeldt and Zleman 2007); (ii) Lichtenberg (2003) has shown that the introduction of drugs for HIV-AIDs coincided with a dramatic reduction in mortality from the disease; and (iii) Lichtenberg (2004), Crémieux, Meilleur, Ouellette *et al*. (2005) (hereafter CMO), Frech and Miller (2004), and Shaw, Horrace, and Vogel (2005) (hereafter Shaw) have estimated the LE gain from drug spending and new drug launches.

The econometric LE models suggest that drugs are remarkably effective. Based on annual U.S. time-series data on LE at birth, health expenditure, and new molecular entities (NMEs) approved each year during the period 1960–2001, Lichtenberg estimates that an additional year of life expectancy can be purchased for US$ 926 (1982 dollars) in per capita pharmaceutical research and development (R&D) expenditure, which is cheap compared to his estimated cost of buying a life year using public health expenditure ($9,640). Using annual data on LE, drug and other types of health care spending in each of the ten Canadian provinces over the period 1981-1998, CMO estimate remarkably large drug expenditure-LE elasticities. For instance, a one percent increase in private drug expenditure was found to increase male LE at birth by 0.015%. Evaluating this response at sample means, this estimate implies that one more year of life expectancy can be had for additional per capita drug expenditure of only CDN$ 186 (1997 dollars). This is a bargain: consensus estimates of the value of a life year are orders of magnitude larger (Viscusi and Aldy 2003). Shaw, and Frech and Miller provide corroborating evidence. Shaw regressed LE in 19 OECD countries in 1997 on country-wide drug spending 12 years earlier and a battery of control variables; he estimates that male LE at age 40 increases by 1 year for every $175 increase in drug spending. Frech and Miller regressed 'disability adjusted' LE in 18 OECD countries in 1999 on drug spending in 1990 and several control variables; their estimates imply that male disability adjusted LE at birth increases by 1 year for every $186 increase in drug spending.

These results, taken at face value, have important health policy implications. Indeed, Lichtenberg concludes that "increased development of new drugs may be a more cost-effective way of increasing life expectancy than increased public health expenditure." In other words, governments seeking to improve population health should realign spending priorities, reducing subsidies for physician and hospital services and increasing subsidies of pharmaceutical R&D. The other studies do not appraise the value of medical R&D *per se*, but provide evidence that drugs are among the most cost effective forms of health care.

Before governments act on these recommendations, it seems prudent to critically appraise these econometric analyses and ensure that the results standup upon re-examination of the data. This is precisely what we attempt to do in this paper. We point to several questionable assumptions underlying these empirical analyses, assess the plausibility of the estimates and re-analyze the data using what we feel are more appropriate econometric methods to see if the results stand up.

The previous studies

All four studies estimate LE 'production functions', although they take different approaches, both in the models estimated and the data used. The years of LE produced depends on both the amounts of productive inputs used and the technology that converts these inputs into LE outputs. Lichtenberg estimates the contribution of both medical technology (measured using the number of NMEs approved for use in the US each year) and the amounts of medical care and other inputs on LE at birth, whereas the others estimate the contribution of productive inputs alone. Lichtenberg includes in his models lagged real per capita (RPC) health care expenditures, and in some of the models he considers, RPC gross domestic product (GDP) and cigarette consumption as well. CMO consider a larger set of inputs. They model sex-specific LE, both LE at birth and at age 65, as a function of contemporaneous RPC public and private drug expenditures, RPC expenditures on other health services, various 'life style' factors (RPC expenditures on: food and non-alcoholic beverages, alcohol, and tobacco), RPC GDP and regional fixed effects. They also model sex-specific infant mortality rates using the same covariates. Shaw models sex-specific LE, at age 40, 60 and 65 in 1997 as a function of a set of covariates similar to those that CMO considered, except that Shaw used the 12 or 15 year lags of these covariates. Frech and Miller model sex-specific disability adjusted LE (DALE) at birth and at age 60 in 1999 as a function of 1990 values of RPC drug expenditures, RPC expenditures on other health services, alcohol consumption, and rates of smoking and obesity.

Problems with the previous analyses

Functional form issues

All studies assume Cobb-Douglas LE production:

$$LE = A^{\theta} \prod_k x_k^{\beta_k} \quad (1)$$

or in logs:

$$\ln LE = \theta \ln A + \sum_k \beta_k \ln x_k \quad (1A)$$

where A is the level of technology, x_k is the level of the kth input, and θ and the β_k are unknown parameters. In this model, the marginal productivity of an input is proportional to its average productivity:

$$\frac{\partial LE}{\partial x_k} = \beta_k \frac{LE}{x_k} \quad (2)$$

Because the marginal productivities (2) depend on LE, they depend on the levels of all inputs and the existing technology. While this model is plausible, it is somewhat restrictive. It implies that the optimal mix of inputs does not depend on the scale of LE production. One might question whether the restrictions inherent in (2) are consistent with the data and if not, how they can be relaxed. That being said, the paucity of observations available – especially in the analyses of OECD countries – requires that a lot of structure be placed on the model if estimates are to be produced with reasonable precision.

A second concern relates to the separability of the 'inputs' from the 'technology' in the empirical LE production models. The health care spending inputs considered in these models are not the homogenous inputs of the textbook production function because they embody – to varying degrees, depending on the input – the current technology. This is especially true of drugs. A dollar spent on drugs now likely yields more health gain than the (inflation adjusted) dollar spent in 1960, when the scope and effectiveness of drugs was much more limited. This presents a modeling problem: it is difficult to neatly partition the role of technology and inputs in the empirical model. In Lichtenberg's LE model, the influence of pharmaceutical technology is reflected in both the number of NMEs approved and total health care spending (which includes as a component pharmaceuticals spending).

There is one final yet important modeling issue. CMO specify LE to be a function of current health care expenditures, when it seems plausible that health care spending might affect LE only after some time has elapsed. The other investigators, recognizing this, use lagged health care expenditures in their LE models.

Identification of pharmaceutical productivity

All studies estimate parameters using least squares (LS). For LS to be unbiased, covariates must be uncorrelated with variation in the outcome variable not explained by the model; the unexplained health determinants are captured in the model's 'error term'. Yet it is unlikely that this condition is satisfied in the empirical LE models, for several reasons. First, it is possible that health care inputs are endogenous: Governments and individuals alike may increase health care spending in regions/years in which age-specific death rates are particularly high. Lichtenberg's econometric specification overcomes this: He uses lagged health care spending (and lagged new drug introductions) to at once overcome issues of endogeneity and allow health inputs to affect LE only after a lag, which seems more sensible that forcing all effects to be contemporaneous. Shaw uses covariates lagged 12 or 15 years; Frech and Miller use covariates lagged 9 years. As mentioned earlier, CMO's specification is susceptible to this criticism.

Second, model covariates that are measured with error will induce correlation between the mismeasured covariate and the error term. Several covariates are mismeasured: CMO had to interpolate the values of alcohol, tobacco and dietary intake variables for a number of years. Frech and Miller, CMO and Shaw measured drug consumption using inflation-adjusted nominal drug expenditure. But this is a very noisy measure of drug input – larger inflation-adjusted expenditures do not necessarily translate into longer LE, for several reasons. One reason is that this expenditure variation could reflect differences in the vintage composition of drug use (with some provinces using a proportionately larger share of more recently introduced and more costly drugs), and the quantities used of drugs of all vintages. Two provinces with identical drug spending – with one using lots of older, less productive but cheaper drugs and the other using a smaller amount of newer drugs – might therefore achieve different health outcomes. Variation in inflation-adjusted drug spending might also reflect variation in other factors unrelated to 'drug input', including:

- payments to drug wholesalers and pharmacies
- waste: drugs dispensed but not taken as recommended (i.e. drug non-compliance), or the use of expensive drugs when less costly drugs work equally well (Morgan 2001).
- use of drugs that affect primarily morbidity not mortality (Shaw, Horrace and Vogel 2005)

- noise in the index used to adjust for drug price differences (which CMO never described), so that some of the variation in inflation-adjusted drug spending might actually reflect drug price differences.

Shaw, and Frech and Miller face the same measurement issues as do CMO plus the problem of reconciling drug spending denominated in different currencies and collected using different accounting standards (Gerdtham and Jonsson, 2000). Lichtenberg's econometric specification overcomes these difficulties. Implicitly he uses new drug introductions as an instrument for drug expenditures. Instead of using instrumental variables estimation of a 'structural' model, however, he estimates a reduced form model; that is, he regresses LE on new drug introductions.

Third, both LE and drug spending/new drug launches could be correlated with omitted variables, and these omitted variables would be contained in the error term. The list of suspects includes the state of surgical, diagnostic, disease management and other non-pharmaceutical medical technologies that are not explicitly embodied in the health care spending variables. Certainly, there have been important innovations in non-pharmaceutical medical innovation (Fuchs and Sox 2001) and these advances are often complementary to advances in pharmacotherapy. The management of diabetes is a good case in point. While there have been important advances in drug treatment for this disease (especially in the range of oral hypoglycemic drugs that are available), the promulgation of treatment guidelines and advances in patient self-management have likely also contributed to lowering death rates from the disease.

Also on our list of suspected omitted variables is the distribution of health inputs across individuals. Specifically, all studies specify an aggregate health production function using inputs that might apply at the individual level. Aggregate health, however, also depends on the distribution of inputs across individuals if the production function is non-linear. To illustrate, suppose that a fixed amount of healthcare is divided among 2 individuals *A* and *B*, so that the average per capita allocation is constant, regardless of how it is divided. Suppose further that the marginal productivity of healthcare declines, the more is used. Then average population health depends on the allocations to *A* and *B*; if initially *B* gets all the healthcare, then average health can be improved by a small transfer from *B* to *A* – because health is a concave function of healthcare, the improvement in *A*'s health outweighs the decline in *B*'s health. Estimates from both studies are suspect if changes in mean values of health care inputs are associated with changes in the distribution of these inputs.

Stationarity

In addition to problems created by correlation between regressors and the error term, the LS estimator is also potentially adversely affected by non-stationarity of the regressors. CMO claim that, because their GLS model is asymptotically equivalent to a regression on the first-differenced variables, their estimator is robust to the presence of non-stationary variables. But it is not clear if this is the case. First, their sample size is modest so it is unclear if an asymptotic result would be a good approximation. Second, it is unclear if their model covariates are all integrated of the same order. Lichtenberg cannot rule out the hypothesis that LE and real health care spending are random walks although he presents evidence that the annual NMEs approved is stationary.

Plausability of Estimates

Granger (1999) argues that it is better to judge a model by its outputs, not inputs. After all, it is unclear to what extent the criticisms leveled against a model matter empirically. Hence, we assess the output of the empirical LE models – i.e. the marginal effects estimates and predictions. We first ask whether the models' estimates of pharmaceutical productivity are consistent with estimates obtained from other sources. We could not locate any other sources of data on the LE gains of pharmaceutical R&D expenditure *per se*, but we did locate a review of the estimates of cost per 'quality adjusted' life year, or QALY, produced by 251 different pharmaceuticals introduced over the period 1976-1997 (Neuman *et al*, 2000). Presumably it is more costly to produce a quality adjusted life year (a year of life spent in normal health) than a life year spent in perhaps compromised health, so the estimates reviewed by Neuman might overestimate the cost per (unadjusted) LE. On the other hand, the 251 different drugs Neuman reviewed might not be representative of all the 526 different drugs introduced into the US over the same period; in particular, these evaluations might be selected for formal evaluation and the results published due to particularly favorable cost effectiveness. Moreover, the economic appraisals typically rely of efficacy estimates generated from clinical trials, and thus might overstate treatment effectiveness in routine clinical conditions. On balance then, Neuman's estimates likely serve as a useful benchmark.

Neuman finds that the median cost per QALY produced by pharmaceuticals is US$ 11,000 (1998 dollars). How does this figure compare with the estimates produced by CMO? CMO did not report directly comparable estimates but it is possible to generate comparable figures from the marginal effects expression (2) assuming (2) holds for discrete changes, evaluated at sample means:

$$\Delta x_k = \frac{\Delta LE \times \bar{x}_k}{\beta_k \times \overline{LE}} \qquad (3)$$

This expression gives the change in the input Δx_k required to increase the LE measure by 1 year $(\Delta LE = 1)$, given CMO's elasticity estimate β_k, and the sample averages of x_k and LE ($\bar{x}_k$ and $\overline{LE}$, respectively). Estimates of Δx_k for each of CMO's 6 models (sex-specific models of LE at birth, LE at 65 and infant mortality rates per 1000 births) are presented in Table 1.

Table 1 Estimated change in real per capita health care spending required to increase health outcome by one unit, by type of health care spending. Estimates based on CMO (2005).

health outcome	sex	type of health care spending		
		private prescription drugs	public prescription drugs	total non-drug health care spending
infant mortality rate per 1000 live births	male	-145	-89	-440
	female	-153	-67	-1,111
life expectancy at birth	male	182	97	1,468
	female	419	109	22,932
life expectancy at 65	male	256	201	-2,439
	female	768	486	-8,175

Hence CMO's estimates, which are in the range $97 (the increase in RPC public drug spending required to increase male LE at birth by one year) to $768 (the increase in RPC private drug spending required to increase male LE at 65 by one year) are between 1% to 7% of Neuman *et al*'s median estimate of $11,000.[1] Shaw's estimates – between $152 and $231 – are in the same ballpark as CMO's.

Frech and Miller's estimates of drug spending on disability-adjusted LE are perhaps most directly comparable to Neuman's because they explicitly account for the potential effects of drugs on morbidity. Frech and Miller's estimates imply that an additional year of disability adjusted LE can be purchased for a figure in the range of $140 to $186.

Table 2 Estimated change in real per capita health care spending required to increase health outcome by one unit, by type of health care spending. Estimates based on Frech and Miller (2004).

health outcome	sex	type of health care spending	
		drugs	total non-drug health care spending
disability adjusted life expectancy at birth	male	186	1,012
	female	173	940
disability adjusted life expectancy at 60	male	172	2,534
	female	140	2,059

Lichtenberg provides estimates of the expenditure on pharmaceutical R&D and publicly financed healthcare required to increase LE by one year. Evaluating his elasticity estimates at his sample means, in equilibrium, one additional year of LE at birth can be purchased for $171 in per capita public healthcare expenditure or just $16 in per capita outlays on pharmaceutical R&D.[2] Our $171 estimate is a fraction of Lichtenberg's estimate of $9,640 – the reason for the difference is that Lichtenberg assumes that only newborns would benefit from increased healthcare spending, while we assume that everyone benefits. Our estimate is certainly too low and Lichtenberg's too high, but we feel our estimate is closer to the truth. The LE measure used in the literature is 'period' LE, which is a function of age-specific death rates for a given year. If it were the case that healthcare affected just infant mortality rates then Lichtenberg's approach would be correct. But health care use tends to increase with age, suggesting that the benefit of healthcare use does as well.

Another way of assessing the plausibility of the LE production models is to compare the out-of-sample forecasts from these models with actual values. We therefore gathered post-sample data on the covariates used in the respective models (1999-2004 for CMO's models, 2002-2004 for Lichtenberg's

[1] Interestingly, CMO find that non-drug spending has deleterious effects on the LE of those who have reached 65 years of age: Every $2400 increase in RPC non-drug spending was found to decrease male LE at 65 by one year.
[2] This $16 estimate uses Lichtenberg's assumptions re: the cost of developing a new drug – $500 million – and the average size of the US population – 224 million – during his sample period.

model #3, and 1985-1993 and 1980-1988 for the covariates in Shaw's models), and using these data, forecasted values of the outcome variables. Because the models are log-linear, we compared post-sample forecasted growth rates in the outcome variable with actual growth rates.

The prediction performance of CMO's models varied depending on the outcome being modeled (Table 3). Life expectancy at birth was predicted with the greatest degree of accuracy; forecast errors were between 21%-50% of observed LE growth Life expectancy at 65 was less well predicted – forecast errors were between 85%-139% of observed values. Female LE was predicted to decline when it actually grew. The infant mortality models performed worst; the models predicted reductions in the order of 33% and 21% for males and females, respectively, while male IMRs declined by 4% and females actually increased by 4%. Lichtenberg's model fared better. It over-predicted the 0.71% growth in LE observed 2001-2004 but the prediction error was only 10% of observed growth.

Table 3 Observed and predicted growth in life expectancy and infant mortality in Canada, 1998-2004

Outcome	Sex	Observed growth 1998-2004	Predicted growth 1998-2004*	Error = Observed-Predicted	100*Error / Observed
Life expectancy at birth					
	Males	2.37%	1.87%	0.50%	21%
	Females	1.35%	0.67%	0.68%	50%
Life expectancy at 65					
	Males	8.59%	1.27%	7.32%	85%
	Females	4.48%	-1.76%	6.24%	139%
Infant mortality rate					
	Males	-3.51%	-33.14%	29.63%	-844%
	Females	4.17%	-20.85%	25.01%	600%

Notes

$$\text{Predicted Growth} = \sum_{k=1}^{K} \eta_k \times \frac{x_{k,2004} - x_{k,1998}}{x_{k,1998}} \times 100$$

where k indices the covariates used in CMO's models, and η_k indicates the estimated elasticity associated with the kth covariate.

Shaw's models produced forecasts errors in the order of 11.7% of observed growth of female LE at age 40 to 27.2% of observed growth of male LE at age 40. Forecast errors for the models of LE at 65 were about 15% of observed growth.

Do the marginal productivity estimates stand up to alternative model specifications?

A model is more credible if its outputs are robust to seemingly innocuous changes in its specification. We therefore assembled the data used in the CMO, Shaw and Lichtenberg studies, updated them where possible and re-estimated modified versions of the models.

CMO

We estimated modified versions of CMO's models using province and year level data over the period 1981-2004. We modified their model in three ways. First, instead of using contemporaneous values of covariates, we used one period lags of on RPC spending on pharmaceuticals, hospitals, physicians, a composite of all other health care spending, and RPC GDP. Second, we used province-specific dummies to control for time-invariant provincial differences in the outcome variables; CMO used regional dummies. Third, we used year dummies to control for the influence of time-varying confounders. This approach avoids the problems associated with identifying, measuring and modeling the impact of time-varying confounder variables. Specifically, one would likely miss important determinants; moreover, the factors that one might attempt to control for explicitly – alcohol and cigarette consumption, for instance – are likely mismeasured. The assumption underlying our approach – common year effects – strikes us as being defensible for the case of medical technology, which Cutler *et al* suggest as being the primary driver of recent LE gains. Presumably medical technology is equally accessible in all the provinces. This assumption is perhaps less defensible for the case of the distribution of alcohol, cigarette consumption and other lifestyle factors – the distribution of these factors could evolve differently in the different provinces. We therefore estimate alternative models that control for time-varying confounders using province-specific quadratic time trends.

The revised models yield dramatically different estimates of the expenditure required to increase LE by one year or increase infant mortality rates by one death per 1,000 live births. The models estimated using year dummies indicate that drug spending *increases* infant mortality rates. Drug spending is still found to increase LE, but the expenditure required to extend LE by 1 year is now much greater than that implied by CMO. Indeed expenditure requirements are between 5 and 72 times larger than corresponding estimates from CMO's models. The model that uses province-specific quadratic time trends produces estimates in the range of 10 and 527 times those produced by CMO. This model suggests, however, that drug spending reduces infant mortality rates.

Table 4 Estimated change in real per capita health care spending required to increase health outcome by one unit, by type of health care spending. Time varying confounders controlled for using year dummy variables.

health outcome	sex	type of health care spending			
		private prescription drugs	public prescription drugs	Hospitals	Physician services
infant mortality rate per 1000 live births	Male	1,607	649	-1,357	930
	female	1,044	280	-1,389	783
life expectancy at birth	Male	10,086	6,726	32,422	-5,222

health outcome	sex	type of health care spending			
		private prescription drugs	public prescription drugs	Hospitals	Physician services
	female	22,021	7,861	57,377	-7,120
life expectancy at 65	male	3,455	1,831	16,358	-1,781
	female	4,720	2,233	57,175	-3,467

Note: cost estimates obtained by evaluating elasticity estimates at sample means. GLS estimator used to allow for province-specific heteroskedasticity and common AR(1) errors.

Table 5 Estimated change in real per capita health care spending required to increase health outcome by one unit, by type of health care spending. Time varying confounders controlled for using province specific quadratic time trends.

health outcome	Sex	type of health care spending			
		private prescription drugs	public prescription drugs	hospitals	Physician services
infant mortality rate per 1000 live births	Male	-3,425	-227	22,945	1,368
	Female	-2,582	-323	-9,558	2,524
life expectancy at birth	Male	96,061	4,974	23,285	-7,217
	Female	164,769	4,384	20,730	-18,149
life expectancy at 65	Male	5,733	6,442	10,856	-6,717
	Female	29,059	4,883	7,955	-9,477

Note: cost estimates obtained by evaluating elasticity estimates at sample means. GLS estimator used to allow for province-specific heteroskedasticity and common AR(1) errors.

Shaw

Whereas Shaw estimated his models using a cross section of 19 OECD countries in 1997, we used panel data on the LE of selected OECD countries (Australia, Belgium, Canada, Denmark, Finland, France, Germany, Ireland, Japan, Netherlands, New Zealand, Norway, Sweden, UK, US) over the period 1962-2004. The primary advantage of our approach, of course, is that we can control for time-invariant country-specific differences in LE through the use of country dummy variables. The second advantage is sample size – we estimated parameters using as many as 370 observations; Shaw had just 19 observations to work with. We used lagged values of the same covariates that Shaw used, but we considered various lag lengths. Shaw used a 15 year lag; we tried 15 years and shorter lags (of 1, 2, 3, 4, and 5 years) as well. Finally, we considered models with and without year dummies included to assess the robustness of the estimates to the presence of time-varying confounders.

Our OLS estimates, appearing in Appendix 1 and 2, imply completely different values of pharmaceutical productivity compared to those implied by Shaw. Regardless of the covariate lag length chosen or the inclusion of year dummies, the models suggest that pharmaceutical spending *shortens* LE.

Lichtenberg

Recall that Lichtenberg regressed LE on its lagged value, current and lagged values of the number of NMEs approved and lagged RPC public healthcare spending. (All variables were in log units.) Clearly NME approvals do not affect LE directly; they affect LE only to the extent that they are consumed. Lichtenberg's model can thus be interpreted as a reduced form of a 'structural' model in which LE depends on drug consumption and drug consumption depends on the number of NMEs approved. When viewed from this perspective, Lichtenberg's model specification should be adjusted slightly. In particular, the health care spending variable he uses should reflect non-pharmaceutical health care spending since drug spending would not appear in a reduced form model. We therefore re-estimated Lichtenberg's model with two modifications. First, we used up-to-date data (data were collected for the period 1960-2004). Second, we replaced lagged RPC public healthcare spending with lagged RPC total spending on hospitals, physicians, and public health. Estimates were obtained using generalized least squares allowing for AR(1) errors; given the modest sample size we included the Prais-Winsten transformed first observation. Using these estimates, which appear in Appendix 3, we determined the expenditure on the various types of health care required to increase LE at birth by one year in long run equilibrium. We also determined the number of NMEs required to accomplish the same thing. We computed the per capita cost of developing the requisite number of NMEs by multiplying the estimate by $500M (the average cost per NME) and dividing by the average size of the US population during the sample period, 225M.

Table 6 Estimated change in real per capita spending required to increase LE at birth by one year, in longrun equilibrium, by type of intervention.

Intervention	per capita cost to increase LE by one year
develop NME	526.24
expenditures on public health	0.03
expenditures on physicians	-1386.26
expenditures on hospitals	-5215.51

The estimates indicate that the per capita outlays of $526 on drug development are required to increase LE at birth by one year. This estimate is substantially more than the $16 estimate derived from Lichtenberg's model but it is nonetheless still less than consensus estimates of the value of a life year. According to the model, an even less expensive option would be to invest in public health; an expected life year can be acquired for only $0.03. The model suggests that spending on physicians and hospitals is ill advised as these are found to decrease LE.

Discussion

New pharmaceutical drugs have undoubtedly contributed to the marked increases in LE observed over the last 50 years; the question is how much have they contributed? We argue that it is difficult to resolve this question using aggregate data. There are several hurdles, including disentangling the contribution of new drugs from advances in disease management, changes in the distribution of health

care and other confounding factors; measuring drug inputs using inflation-adjusted drug spending data; and dealing with non-stationary time series data. While it is unclear to what extent these modeling issues matter empirically, we present evidence that the several empirical models that have attempted to identify the contribution of pharmaceuticals to LE are not plausible.

These models assess the partial correlations between drug use or new drug launches and LE using cross sections of OECD countries, province-year level data and annual data for the US. We are skeptical of these models for several reasons. First, these studies produce estimates of pharmaceutical productivity that we feel are implausibly high. One study implies that a year of disability-adjusted LE at birth can be purchased for under US$ 200. Cost effectiveness studies of individual drugs, by contrast, indicate that the median cost of a quality-adjusted life year is in the order of US$ 11,000. Second, some of the models estimated using province-year level LE data have very large forecast errors. Third, the models that we replicated were found to be sensitive to seemingly innocuous changes in specification. The model we estimated using province-year level LE data for Canada suggests that pharmaceuticals are indeed productive, but not as productive as implied in the literature. Our estimates of the increase in per capita pharmaceutical expenditure needed to increase LE by one year is in the range $1,800 to $22,000; the original study implied estimates in the range $97 to $768. Another model estimated using LE of a cross-section of OECD countries found drugs to be highly effective. However, when we re-estimated the model exploiting within-country variation in drug expenditure we reached the opposite conclusion. This suggests that among OECD countries, drug spending is correlated with some unobserved determinant of LE. Finally, we found that modeling annual US LE using categories of healthcare spending (instead of an aggregate of all healthcare spending) produced drastically different policy implications. The original study found that new drug introductions were markedly more cost effective than public health care spending. We found just the opposite – indeed per capita outlays on public health measures in the order of $0.03 were found to increase LE by one year.

The conclusion that we draw from these analyses is that it is difficult to estimate the bio-medical determinants of LE using aggregate data. Analyses using individual level data or perhaps disease specific data will likely produce more compelling results.

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Appendix 1: Shaw's models estimated using panel OECD data. Results for Male life expectancy

Model specification							Males											
Covariates lagged:						Year fixed effects included?	LE at 40				LE at 60				LE at 65			
							PHARM		HEALTH		PHARM		HEALTH		PHARM		HEALTH	
1 yr	2 yrs	3 yrs	4 yrs	5 yrs	16 y		coef.	signif.	coef.	signif.	coef.	signif.	coef.	signif.	coef.	signif.	coef.	signif.
x						x	-0.048	***	0.006		-0.073	***	0.022	*	-0.077	***	0.018	
x							-0.022	***	-0.041	***	-0.029	***	-0.056	***	-0.030	***	-0.058	***
	x					x	-0.046	***	0.007		-0.072	***	0.023	*	-0.073	**	0.019	
	x						-0.022	***	-0.037	***	-0.028	***	-0.053	***	-0.027	**	-0.058	***
		x				x	-0.047	***	0.004		-0.074	***	0.018		-0.075	***	0.011	
		x					-0.023	***	-0.039	***	-0.029	***	-0.061	***	-0.026	**	-0.070	***
			x			x	-0.047	***	0.004		-0.075	***	0.019		-0.075	***	0.010	
			x				-0.019	***	-0.040	***	-0.024	**	-0.060	***	-0.021	*	-0.072	***
				x		x	-0.042	***	-0.001		-0.067	**	0.012		-0.066	***	0.006	
				x			-0.018	***	-0.039	***	-0.021	**	-0.060	***	-0.018	*	-0.068	***
					x	x	-0.010	*	-0.001		-0.018	*	-0.004		-0.023	**	-0.002	
					x		-0.009		-0.021	**	-0.016		-0.050	***	-0.021	*	-0.056	***

legend: * p<0.05; ** p<0.01; *** p<0.001

Appendix 2: Shaw's models estimated using panel OECD data. Results for Female life expectancy

Model specification							Females											
Covariates lagged:						Year fixed effects included?	LE at 40				LE at 60				LE at 65			
							PHARM		HEALTH		PHARM		HEALTH		PHARM		HEALTH	
1 yr	2 yrs	3 yrs	4 yrs	5 yrs	16 y		coef.	signif.	coef.	signif.	coef.	signif.	coef.	signif.	coef.	signif.	coef.	signif.
x						x	-0.037	***	0.005		-0.051	***	0.013		-0.049	***	0.007	
x							-0.027	***	-0.004		-0.036	***	-0.004		-0.034	***	-0.004	
	x					x	-0.035	***	0.006		-0.048	***	0.013		-0.042	***	0.002	
	x						-0.025	***	-0.002		-0.032	***	-0.001		-0.028	***	-0.004	
		x				x	-0.036	***	0.002		-0.050	***	0.008		-0.044	***	-0.008	
		x					-0.024	***	-0.007		-0.030	***	-0.012		-0.025	**	-0.020	
			x			x	-0.036	***	0.002		-0.050	***	0.009		-0.048	***	-0.003	
			x				-0.019	***	-0.009		-0.022	**	-0.016		-0.018	*	-0.023	
				x		x	-0.031	***	0.001		-0.043	***	0.010		-0.038	***	0.003	
				x			-0.016	***	-0.010		-0.017	*	-0.016		-0.012		-0.018	
					x	x	-0.014	**	0.002		-0.023	**	0.016		-0.024	*	0.011	
					x		-0.011	*	-0.014		-0.016		-0.021		-0.015		-0.028	*

legend: * p<0.05; ** p<0.01; *** p<0.001

Appendix 3 Estimated reduced form model of LE at birth based on annual US data 1960-2004.

Covariate	estimate
Lag log real per capita expenditure on physicians	-0.010
	-1.650
Lag log real per capita expenditure on hospitals	-0.005
	-1.170
Lag log real per capita expenditure on public health	0.021
	3.420
Lag log life expectancy at birth	0.800
	15.390
Lag log number of new molecular entities approved	0.0005
	0.400
Log number of new molecular entities approved	0.004
	3.320
Constant	0.857
	3.970
Rho	-0.090
N	44

Note: *t*-statistics appear in italics.

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