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PHARMACEUTICAL INNOVATION AND THE LONGEVITY OF AUSTRALIANS: A FIRST LOOK

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ABSTRACT

We examine the impact of pharmaceutical innovation on the longevity of Australians during the period 1995-2003. Due to the government's Pharmaceutical Benefits Scheme, Australia has much better data on drug utilization than most other countries. We find that mean age at death increased more for diseases with larger increases in mean drug vintage. The estimates indicate that increasing the mean vintage of drugs by 5 years would increase mean age at death by almost 11 months. The estimates also indicate that using newer drugs reduced the number of years of potential life lost before the ages of 65 and 70 (but not before age 75). During the period 1995-2003, mean age at death increased by about 2.0 years, from 74.4 to 76.4. The estimates imply that, in the absence of any increase in drug vintage, mean age at death would have increased by only 0.7 years. The increase in drug vintage accounts for about 65% of the total increase in mean age at death. We obtain a rough estimate of the cost per life-year gained from using newer drugs. Under our assumptions, using newer drugs (increasing drug vintage) increased life expectancy by 1.23 years and increased lifetime drug expenditure by $12,976; the cost per life-year gained from using newer drugs is $10,585. An estimate made by other investigators of the value of a statistical Australian life-year ($70,618) is 6.7 times as large as our estimate of the cost per life-year gained from using newer drugs. We discuss several reasons why our estimate of the cost per life-year gained from using newer drugs could be too high or too low.

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In previous papers, Lichtenberg (2005a, 2005b) has examined the impact of pharmaceutical innovation on longevity in the United States and in a group of developed and developing countries. Due to data limitations, the measure of pharmaceutical innovation used in these studies was the cumulative number of drugs launched. These studies provided support for the hypothesis that the introduction of new drugs has played an important role in increasing longevity.

In this paper, we will examine the impact of pharmaceutical innovation on the longevity of Australians during the period 1995-2003. In one important respect, the data available for Australia are much better than those used in the previous studies. Rather than merely knowing whether a given drug has been launched in Australia by a certain date, we know how frequently that drug is used in each year. Combining these data with data from other sources enables us to calculate the mean *vintage*[1] of drugs utilized in Australia, by disease and year.

Section I contains a discussion of the "embodied technological progress hypothesis". Section II describes an econometric model to test this hypothesis. Data sources and descriptive statistics are presented in Section III. Empirical results are presented in Section IV. Section V contains a summary and discussion.

I. Embodied technological progress hypothesis

Economists believe that the development of new products is the main reason why people are better off today than they were several generations ago. In their 1993 book, *Innovation and Growth in the Global Economy*, Grossman and Helpman argued that "innovative goods are better than older products simply because they provide more 'product services' in relation to their cost of production." In their 1996 book, *The Economics of New Goods*, Bresnahan and Gordon stated simply that "new goods are at the heart of economic progress." In a recent paper, *Measuring the Growth from Better*

[1] The dictionary contains several different definitions of vintage. The definition we use is: "a period of origin or manufacture". We define the vintage of a drug as the year in which the U.S. Food and Drug Administration (FDA) first approved the drug's active ingredient. (The FDA, which has been in existence since 1938, provides the most complete data on drug vintage.) For example, the vintage of Pharmaceutical Benefits Scheme items 8213G, 8214H, 8215J, and 8521L is 1997, the year the active ingredient of all these items (atorvastatin calcium) was approved by the FDA. (These items correspond to 10, 20, 40, and 80 mg tablets, respectively.)

and Better Goods, Bils (2004) makes the case that "much of economic growth occurs through growth in quality as new models of consumer goods replace older, sometimes inferior, models."

We seek to test the hypothesis that, ceteris paribus, people using newer, or later vintage, drugs will be in better health, and will therefore live longer (die later). This hypothesis is predicated on the idea that pharmaceuticals, like other R&D intensive products, are characterized by *embodied technological progress*.

A number of econometric studies (Bahk and Gort (1993), Hulten (1992), Sakellaris and Wilson (2001, 2004)) have investigated the hypothesis that capital equipment employed by U.S. manufacturing firms embodies technological change, i.e. that each successive vintage of investment is more productive than the last. Equipment is expected to embody significant technical progress due to the relatively high R&D-intensity of equipment manufacturers. The method that has been used to test the equipment-embodied technical change hypothesis is to estimate manufacturing production functions, including (mean) vintage of equipment as well as quantities of capital and labor. These studies have concluded that technical progress embodied in equipment is a major source of manufacturing productivity growth.

Although most previous empirical studies of embodied technical progress have focused on equipment used in manufacturing, embodied technical progress may also be an important source of economic growth in health care. One important input in the production of health—pharmaceuticals—is even more R&D-intensive than equipment. According to the National Science Foundation, the R&D intensity of drugs and medicines manufacturing is 74% higher than the R&D intensity of machinery and equipment manufacturing. Therefore, it is quite plausible that there is also a high rate of pharmaceutical-embodied technical progress.

The hypothesis that technical progress is embodied in pharmaceuticals may be tested in two alternative ways. One approach is to investigate whether the health and longevity of people with a given disease is positively related to the number of drugs that have been approved to treat that disease.[2] Lichtenberg adopted this approach in several

[2] In his model of endogenous technological change, Romer (1990) hypothesized the production function $Y = (AL)^{1-\alpha} K^{\alpha}$, where Y = output, A = the "stock of ideas", L = labor used to produce output, K = capital, and

studies (2005a, 2005b, 2005c); in all of them, he found that increases in the cumulative number of drugs improved health. This approach allows one to distinguish between the effects of approval of "priority-review" drugs—drugs that the FDA considers to offer significant improvements over existing therapies—and approval of "standard-review" drugs—drugs that the FDA considers to be similar to previously approved drugs. In two studies, Lichtenberg distinguished between the effects of priority-review and standard-review drug approvals. The results of distinguishing between the two were mixed. Lichtenberg (2005a) found that that approval of standard-review drugs had no effect on longevity, but that approval of priority-review drugs had a significant positive impact on longevity. But most of the results in Lichtenberg (2005c) indicated that the difference between the effect of priority-and standard-review drugs on ability to work was not statistically significant.

The second way to test the hypothesis that technical progress is embodied in pharmaceuticals is to investigate whether the health and longevity of people with a given disease is positively related to the mean vintage (FDA approval year) of drugs used to treat the disease. We believe that the second approach is superior to the first approach. The drugs that have been approved to treat a given disease influence the therapy that a patient *could* receive, but his health and longevity depend on the therapy he actually *does* receive. The fact that a drug has been approved does not necessarily mean that it is commonly used. In this paper we will pursue the second approach.

Although we believe that mean vintage is a better measure of innovation than number of previously-approved drugs, proper accounting for the distinction between priority- and standard-review drugs when measuring drug vintage, while straightforward in theory, is difficult in practice. Suppose a (standard-review) drug approved in 2008 is "therapeutically equivalent" to a drug approved in 1998. Then the "effective vintage" of the drug is 1998, whereas its actual vintage is 2008. (The effective vintage of a priority-review drug is the same as its actual vintage.) If we could measure the effective vintage of all drugs, we would use mean effective vintage instead of mean actual vintage in our econometric model. However, although the FDA characterizes some drugs as

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$0 < \alpha < 1$. The cumulative number of drugs approved is analogous to the stock of (FDA-approved) ideas. Health and longevity may be considered outputs of a health production function.

therapeutically equivalent to previously approved drugs, it does not specify the drugs to which they are therapeutically equivalent. Hence measurement of mean effective vintage is not feasible.

II. Econometric model

To test the hypothesis that pharmaceutical innovation has increased the longevity of Australians, we will estimate the following econometric model:

$$Y_{it} = \beta \left[\Sigma_d N\_RX_{dit} FDA\_YEAR_d / \Sigma_d N\_RX_{dit}\right] + \alpha_i + \delta_t + \varepsilon_{it}$$

or
$$Y_{it} = \beta V_{it} + \alpha_i + \delta_t + \varepsilon_{it} \qquad (1)$$

where

Y_{it} = a measure based on the age distribution of deaths from disease i in year t
N_RX_{dit} = the number of times drug d was used to treat patients with disease i in year t
FDA_YEAR_d = the FDA approval year of the active ingredient of drug d
$V_{it} = \Sigma_d N_{dit} FDA\_YEAR_d / \Sigma_d N_{dit}$
= the mean vintage of drugs used to treat disease i in year t
α_i = fixed disease effects
δ_t = fixed year effects

There are both practical and theoretical reasons to define the vintage of a drug as the year the drug was approved by the U.S. FDA rather than the year the drug was listed (approved for reimbursement) in Australia's Pharmaceutical Benefits Sceme (PBS). Data on PBS listing dates are quite incomplete. We obtained unpublished data on listing dates of drugs listed by the PBS after 1990.[3] Based on a sample of 311 drugs for which both FDA approval dates and PBS listing dates were available, we estimate that the mean lag between FDA approval and PBS listing is 3.6 years. However we believe that the FDA approval date is theoretically superior to the PBS listing date as a measure of vintage (which is intended to indicate year of (global) market introduction or first use). The vintage of a wine is the year the wine was bottled, not the year it was opened!

[3] We are grateful to Kim Sweeny of Victoria University for sharing these data with us.

In principle, health and longevity may be affected by lagged as well as current mean drug vintage. However, including lagged vintage would substantially reduce the size of our sample since we have data on Y and V in only 9 years (1995-2003). Moreover, since vintage tends to be serially correlated, including lagged vintage terms would introduce multicollinearity. We will therefore only include contemporaneous vintage in the model.

We will estimate the model using 4 different dependent variables. The first is the mean age at death of Australians dying from disease i in year t:

$$AGE\_DEATH_{it} = \Sigma_a (a\ N\_DEATH_{ait}) / \Sigma_a\ N\_DEATH_{ait}$$

where N_DEATH_{ait} is the number of deaths at age a from disease i in year t.

The second is the logarithm[4] of potential years of life lost before age 75 from disease i in year t:

$$LPYLL75_{it} = \ln[\Sigma_a \max(75 - a, 0)\ N\_DEATH_{ait}]$$

The Australian Institute of Health and Welfare (AIHW) reports both mean age at death and potential years of life lost before age 75 in its General Records of Incidence of Mortality. It also notes that the limit to life of 75 years is "arbitrary". We will also estimate models using two lower thresholds, 70 and 65:[5]

$$LPYLL70_{it} = \ln[\Sigma_a \max(70 - a, 0)\ N\_DEATH_{ait}]$$

$$LPYLL65_{it} = \ln[\Sigma_a \max(65 - a, 0)\ N\_DEATH_{ait}]$$

All models will be estimated via weighted least squares. For the first model the weight is the number of deaths from disease i in year t: $N\_DEATH_{.it} = \Sigma_a\ N\_DEATH_{ait}$.

For the second model the weight is the mean number of potential years of life lost before age 75 from disease i during the 9 years 1995-2003: $(1/9)\ \Sigma_t \exp(LPYLL75_{it})$. Analogous weights will be used for the two lower age thresholds.

Due to the presence of fixed disease effects and year effects, eq. (1) is a difference-in-differences model. If the dependent variable is mean age at death, a positive and significant estimate of β would signify that there were above-average

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[4] The logarithmic specification embodies the assumption that equal increases in vintage result in equal *percentage* reductions in potential years of life lost.

[5] The 70-year threshold is the one used in the OECD Health Database for making international comparisons. The 65-year threshold is the "default choice" in the U.S. Center for Disease Control's Years of Potential Life Lost Reports <http://www.cdc.gov/ncipc/wisqars/fatal/help/definitions.htm>.

increases in mean age at death for diseases with above-average increases in mean vintage of drugs.

III. Data sources and descriptive statistics

Mortality data. The AIHW has compiled long-term mortality data on selected causes of death by age and sex for each year from the beginning of the 20th century, and published them in its GRIM (**G**eneral **R**ecord of **I**ncidence of **M**ortality) books. These are interactive Excel workbooks updated annually containing comprehensive long-term mortality data on selected causes of death by age and sex for each year. The GRIM books have been grouped together by chapters as adopted by the 10th Revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10). Each workbook contains mortality data, population data, derived data items (e.g. age-specific and age-standardised rates), summary measures (e.g. mean age at death, potential years of life lost, lifetime risk of dying), birth cohort information and graphs.

The following table shows annual mortality data for all causes of death combined for the period 1995-2003.

Year	Number of deaths	Mean age at death	Years of life lost before age 75	Years of life lost before age 75 per 1,000 population
1995	125,133	71.8	966,458	56.2
1996	128,719	72.2	963,160	55.3
1997	129,350	72.4	959,548	54.6
1998	127,202	72.4	941,793	53.1
1999	128,102	72.6	938,078	52.4
2000	128,291	73.0	908,058	50.2
2001	128,544	73.3	881,733	48.2
2002	133,707	73.8	876,770	47.4
2003	132,292	73.9	866,298	46.4

Pharmaceutical utilization data. Data on pharmaceutical utilization were obtained from the National Social Health Statistical Data Library (HealthWIZ)[6] a database on CD-ROM that is used to disseminate comprehensive population health related statistical datasets, across the Australian health services sector, for the purposes of clinical research, policy

[6] http://www.health.gov.au/internet/wcms/publishing.nsf/Content/Healthwiz-1

development and health services planning, particularly in regional areas. Several datasets contained in HealthWIZ are derived from the Australian Government's Pharmaceutical Benefits Scheme (PBS).[7]

For nearly 60 years, the PBS has provided reliable, timely and affordable access to a wide range of medicines for all Australians. Many medicines cost the Government much more than the price paid by the patient – some cost hundreds, even thousands of dollars, but the government provides a subsidy so that patients pay much less. The patient receives the benefit of this subsidy when she has her prescription for a medicine filled under the PBS. Current provisions governing the operations of the PBS are embodied in Part VII of the National Health Act 1953 together with the National Health (Pharmaceutical Benefits) Regulations 1960 made under the Act. The scheme has proven itself to be one of the best drug subsidy systems in the world and around 80% of prescriptions dispensed in Australia are subsidized under the PBS.[8] Every time a patient fills a prescription for a PBS medicine, she receives a subsidy. From 1 January 2006, the patient pays up to $29.50 for most PBS medicines or $4.70 if she has a concession card. The Australian Government pays the remaining cost. The PBS covered around 170 million prescriptions in the year to June 2005. This equates to about eight prescriptions per person in Australia for the year. With new and more effective medicines helping us to lead longer and healthier lives, the PBS is growing each year. The cost of the PBS is currently around $6.0 billion per year.

HealthWIZ provides data on the number of prescriptions filled under the PBS, by drug and year, 1995-2004. This dataset contained information about approximately 700 drugs. The Anatomical Therapeutic Chemical (ATC) Classification System is used for the classification of drugs. It is controlled by the WHO Collaborating Centre for Drug Statistics Methodology, and was first published in 1976. Drugs are divided into different groups according to the organ or system on which they act and/or their therapeutic and

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[7] http://www.health.gov.au/internet/wcms/publishing.nsf/Content/health-pbs-general-aboutus.htm

[8] Some of the reasons why a medicine may not be available on the PBS are: (1) the manufacturer has not registered its product to treat a particular condition with the Therapeutic Goods Administration; (2) the manufacturer did not apply to the government's independent expert committee – the Pharmaceutical Benefits Advisory Committee (PBAC) – to list the medicine on the PBS; and (3) the manufacturer hasn't supplied sufficient evidence, or the evidence supplied does not support a recommendation by the PBAC. http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pbs-general-faq.htm-copy2

chemical characteristics. In the system drugs are classified into groups at 5 different levels. There are 14 main groups at the first level.

To illustrate the pharmaceutical utilization data, the following is a list of the top 10 cardiovascular system drugs, ranked by number of prescriptions in 2004:

Drug	**Number of PBS rx's in 2004**
atorvastatin	7,207,717
simvastatin	5,756,278
irbesartan	3,278,440
atenolol	2,952,209
irbesartan with hydrochlorothiazide	2,807,419
ramipril	2,663,857
perindopril	2,578,733
amlodipine	2,201,328
pravastatin	1,978,913
perindopril and diuretics	1,522,659

The following is a list of the top 10 antineoplastic and immunomodulating agents, ranked by number of prescriptions in 2004:

Drug	**Number of PBS rx's in 2004**
tamoxifen	193,340
methotrexate	149,107
leflunomide	108,144
azathioprine	104,236
goserelin	53,556
letrozole	36,837
anastrozole	36,268
cyclophosphamide	35,232
interferon beta-1b	32,282
fluorouracil	29,160

Pharmaceutical vintage data. We used data from the Drugs@FDA database[9] and Mosby's Drug Consult[10] to determine the year in which each active ingredient was first approved by the FDA.

Descriptive statistics on the mean vintage of PBS prescriptions. As the following table shows, during the period 1995-2004 the mean vintage of PBS prescriptions increased by

[9] http://www.fda.gov/cder/drugsatfda/datafiles/default.htm
[10] http://www.mosbysdrugconsult.com/

about 1 year per year, from 1977.8 to 1986.7. The average PBS prescription is for a 17 year-old drug.

year	Number of rx's	Mean FDA approval year
1995	122,224,901	1977.8
1996	125,904,079	1978.8
1997	124,980,656	1979.8
1998	125,365,284	1980.9
1999	133,455,864	1981.9
2000	142,877,869	1983.1
2001	150,924,801	1984.4
2002	158,172,125	1985.3
2003	161,192,358	1986.1
2004	170,253,375	1986.7

The level and growth rate of vintage varies considerably across ATC groups. Figure 1 depicts the mean vintage of two classes of drugs during 1995-2003. Cardiovascular drugs tend to be much newer than antineoplastic and immunomodulating agents; in 2003 the vintage of the latter was almost 10 years lower. The mean vintage of cardiovascular system drugs increased almost twice as much during the first half of this period (1995-1999) as it did during the second half (1999-2003). In contrast, the mean vintage of antineoplastic and immunomodulating agents increased over four times as much in the second half as it did in the first half.

Linkage of drugs to diseases. Estimation of eq. (1) requires linkage of drugs to the diseases they are used to treat. We used the following linkage of ATC drug groups to ICD-10 causes of death chapters:[11]

ATC Drug Group(s)	ICD-10 Cause of Death Chapter(s)
Alimentary tract and metabolism (A) + systemic hormonal preparations, excluding sex hormones and insulins (H)	Diseases of the digestive system (XI) + endocrine, nutritional and metabolic diseases (IV)
Blood and blood forming organs (B)	Diseases of the blood and blood-forming organs (III)
Cardiovascular system (C)	Diseases of the circulatory system (IX)

[11] The following ICD-10 chapters are excluded from our analysis: pregnancy, childbirth and the puerperium (XV); certain conditions originating in the perinatal period (XVI); congenital malformations, deformations and chromosomal abnormalities (XVII); symptoms, signs and abnormal clinical and laboratory findings (XVIII);injury, poisoning and certain other consequences of external causes (XIX); external causes of morbidity and mortality (XX); factors influencing health status and contact with health services (XXI); codes for special purposes (XXII).

Dermatologicals (D)	Diseases of the skin and subcutaneous tissue (XII)
Genito-urinary system and sex hormones (G)	Diseases of the genitourinary system (XIV)
Anti-infectives for systemic use (J) + antiparasitic products, insecticides and repellents (P)	Certain infectious and parasitic diseases (I)
Antineoplastic and immunomodulating agents (L)	Neoplasms (II)
Musculo-skeletal system (M)	Diseases of the musculoskeletal system and connective tissue (XIII)
Nervous system (N)	Diseases of the nervous system (VI) + mental and behavioural disorders (V)
Respiratory system (R)	Diseases of the respiratory system (X)
Sensory organs (S)	Diseases of the eye and adnexa (VII) + diseases of the ear and mastoid process (VIII)

IV. Empirical results

Estimates of eq. (1) with four different dependent variables are shown in Table 1.[12] The equations were estimated using annual data for the period 1995-2003 on the eleven groups of diseases shown above: N = 99 (11 diseases * 9 years). All equations include disease fixed effects and year fixed effects.

The dependent variable of the first equation is the mean age at death of Australians dying from disease i in year t. As shown in line 1, the coefficient on the mean vintage of drugs is positive and statistically significant (p-value = 0.0497). This indicates that mean age at death increased more for diseases with larger increases in mean drug vintage. The point estimate of β indicates that increasing the mean vintage of drugs by 5 years would increase mean age at death by almost 11 months. Additional implications of the estimates of the first model will be considered below. Before doing that, we will discuss estimates of the other three models.

As shown in line 11, when the dependent variable is the logarithm of potential years of life lost before age 75 from disease i in year t, the coefficient on the mean vintage of drugs is negative but not statistically significant (p-value = 0.1787). However, as shown in lines 21 and 31, when the age threshold is either 70 or 65, the coefficient on

[12] Data used to estimate eq. (1) are shown in Appendix Table 1.

the mean vintage of drugs is negative and statistically significant (p-value = 0.0488 and 0.0135, respectively). This implies that using newer drugs has reduced premature mortality—especially mortality before age 65—in the Australian population.[13] The estimates of the three potential years of life lost equations tend to confirm the estimates of the mean age at death equation.

We can use our estimates of the first equation to compare the actual increase in mean age at death during the period 1995-2003 to the increase that would have occurred in the absence of any increase in drug vintage.[14] As shown in Figure 2, during this period, mean age at death increased by about 2.0 years, from 74.4 to 76.4. The estimates imply that, in the absence of any increase in drug vintage, mean age at death would have increased by only 0.7 years. The increase in drug vintage accounts for about 65% of the total increase in mean age at death.

We can also obtain a rough estimate of the cost per life-year gained from using newer drugs. The calculations are shown in the following table.

		year		
		1995	**2003**	**change**
1	rx expenditure[15]	$2,672,000,000	$6,268,000,000	
2	population	18,071,758	19,872,646	
3	rx expenditure per capita ((2)/(1))	$148	$315	
4	life expectancy (mean age at death)	75.13	76.36	1.23
5	"lifetime" rx expenditure per capita ((4) * (3))	$11,109	$24,085	$12,976

Line 3 shows that per capita drug expenditure more than doubled in Australia from 1995 to 2003, from $148 to $315. For simplicity, suppose that *all* of this increase was due to the fact that the drugs used in 2003 were newer than those used in 1995. Line 4 shows the increase in "life expectancy" (mean age at death) that is attributable to increasing drug vintage. Line 5 shows "lifetime" drug expenditure per capita: annual expenditure

[13] The magnitude of the point estimate in line 31 is about 35% larger than the magnitude of the point estimate in line 21. But since the number of years of potential life lost before age 70 is about 47% higher than the number of years of potential life lost before age 65, these two models yield similar estimates of the absolute reduction in years of potential life lost from increasing drug vintage.

[14] The increase that would have occurred in the absence of any increase in drug vintage is measured by the differences between the year fixed effects shown in lines 2-10 of Table 1.

[15] Source: OECD Health Database.

times life expectancy. Under our assumptions, using newer drugs (increasing drug vintage) increased life expectancy by 1.23 years and increased lifetime drug expenditure by $12,976. The cost per life-year gained from using newer drugs is $10,585 (= $12,976/1.23).

Viscusi (2005), citing Kniesner and Leeth (1991), estimates that the value of a statistical Australian life is 4.2 million USD, which is equal to $A 5.4 million at the current exchange rate (1.2839 $A/USD). This implies that the value of a statistical Australian *life-year* is $70,618 (=$A 5.4 million / 76.4). This value is 6.7 times as large as our estimate of the cost per life-year gained from using newer drugs.

V. Summary and discussion

We have examined the impact of pharmaceutical innovation on the longevity of Australians during the period 1995-2003. Due to the government's Pharmaceutical Benefits Scheme, Australia has much better data on drug utilization than most other countries.

We found that mean age at death increased more for diseases with larger increases in mean drug vintage. The estimates indicated that increasing the mean vintage of drugs by 5 years would increase mean age at death by almost 11 months. The estimates also indicated that using newer drugs reduced the number of years of potential life lost before the ages of 65 and 70 (but not before age 75).

During the period 1995-2003, mean age at death increased by about 2.0 years, from 74.4 to 76.4.[16] The estimates implied that, in the absence of any increase in drug vintage, mean age at death would have increased by only 0.7 years. The increase in drug vintage accounts for about 65% of the total increase in mean age at death.

We obtained a rough estimate of the cost per life-year gained from using newer drugs. Under our assumptions, using newer drugs (increasing drug vintage) increased life expectancy by 1.23 years and increased lifetime drug expenditure by $12,976; the cost

[16] Lichtenberg (2005a) found that, in the U.S., *within-disease* increases in mean age at death accounted for about 80% of the aggregate long-term increase in mean age at death; the remaining 20% was due to a shift in the distribution of fatal diseases.

per life-year gained from using newer drugs is $10,585.[17] An estimate made by other investigators of the value of a statistical Australian life-year ($70,618) is 6.7 times as large as our estimate of the cost per life-year gained from using newer drugs.

For several reasons, our estimate of the cost per life-year gained from using newer drugs could be too high or too low. Studies based on U.S. data (Lichtenberg (2001, 2005c, 2006)) indicate that use of newer drugs reduces admissions to hospitals and nursing homes, and increases ability to work. By not accounting for this, we may have overestimated the cost per Australian life-year gained.

Use of newer drugs may have cross-disease spillover effects: using newer drugs for one disease may either increase or decrease mortality from other diseases (in part due to "competing risks"). Such spillovers could be either negative or positive. For example, using a newer drug to treat cancer might reduce cancer mortality but increase life-years lost due to cardiovascular disease. On the other hand, using a newer drug to treat depression and other mental disorders might lead to better management of cardiovascular disease.

Finally, innovation in medical devices and procedures, as well as in drugs, have undoubtedly contributed to Australian longevity increase.[18] The models we have estimated control (via year fixed effects) for device/procedure innovation that is common to all diseases, but not for disease-specific device/procedure innovation: measuring disease-specific device/procedure innovation is far more challenging than measuring disease-specific drug innovation. Since device/procedure innovation may either substitute for or complement drug innovation, controlling for disease-specific device/procedure innovation could either decrease or increase our estimate of the cost per life-year gained from using newer drugs.

Our findings, which are based on aggregate data, are broadly consistent with previous findings based on individual-level data. Lichtenberg and Virabhak (2007) examined the impact of drug vintage on health and longevity using data on (American) individuals before and after the drugs were consumed. They found that people who used

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[17] This is an estimate of the cost per life-year gained from using newer drugs *in general*. It is likely that the cost per life-year gained from some newer drugs is higher, and from other newer drugs is lower, than this average.

[18] However, the biopharmaceutical industry is much more R&D-intensive than the medical device and equipment industry.

newer drugs had better post-treatment health than people using older drugs for the same condition, controlling for pre-treatment health, age, sex, race, marital status, education, income, and insurance coverage: they were more likely to survive, their perceived health status was higher, and they experienced fewer activity, social, and physical limitations. Most of the health measures indicated that the effect of drug vintage on health is higher for people with low initial health than it is for people with high initial health. This suggests that pharmaceutical-embodied technical progress has a tendency to reduce inequality as well as promote economic growth, broadly defined.

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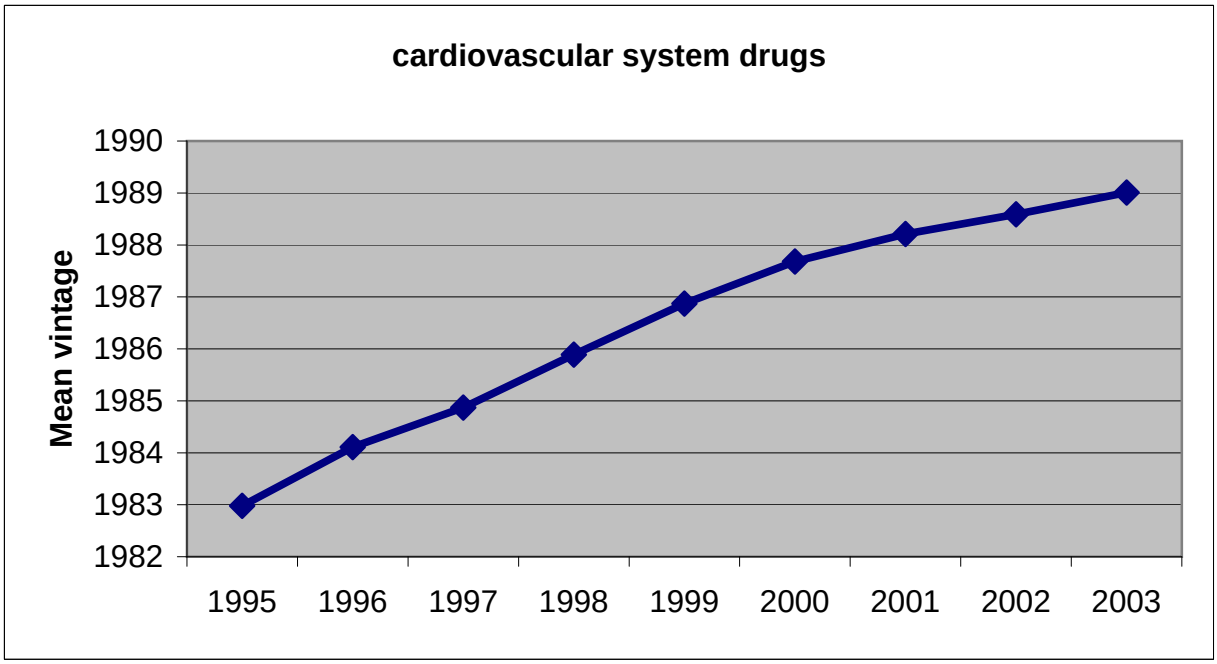
Figure 1
Mean vintage of two classes of drugs, 1995-2003



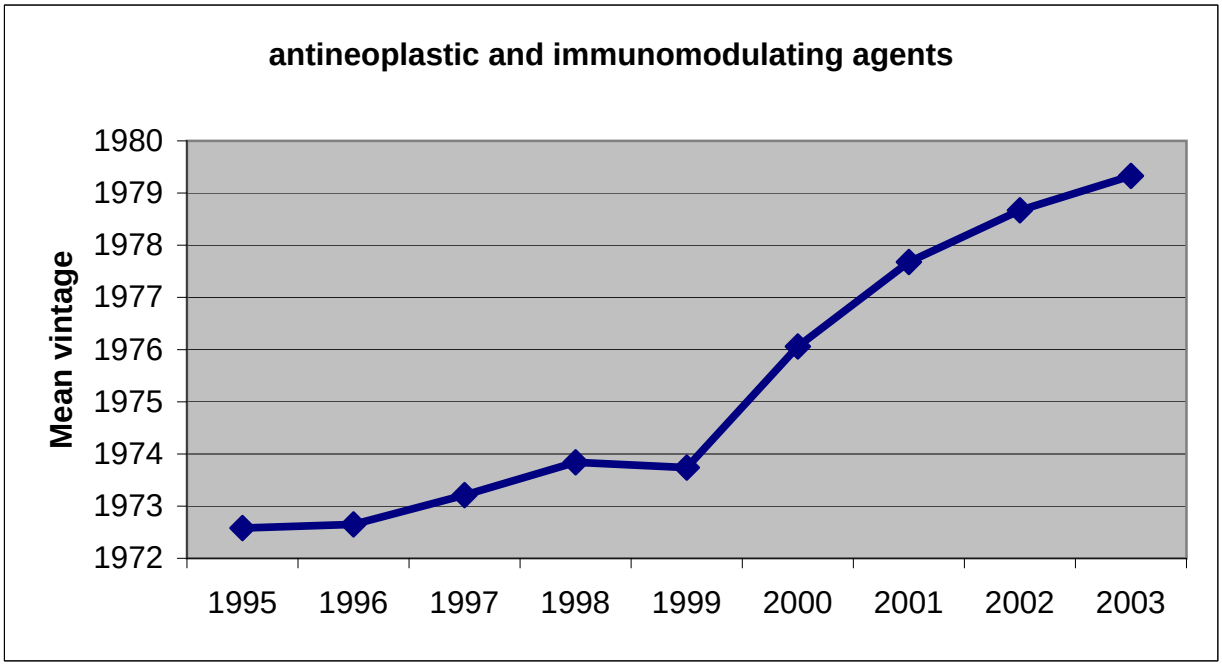
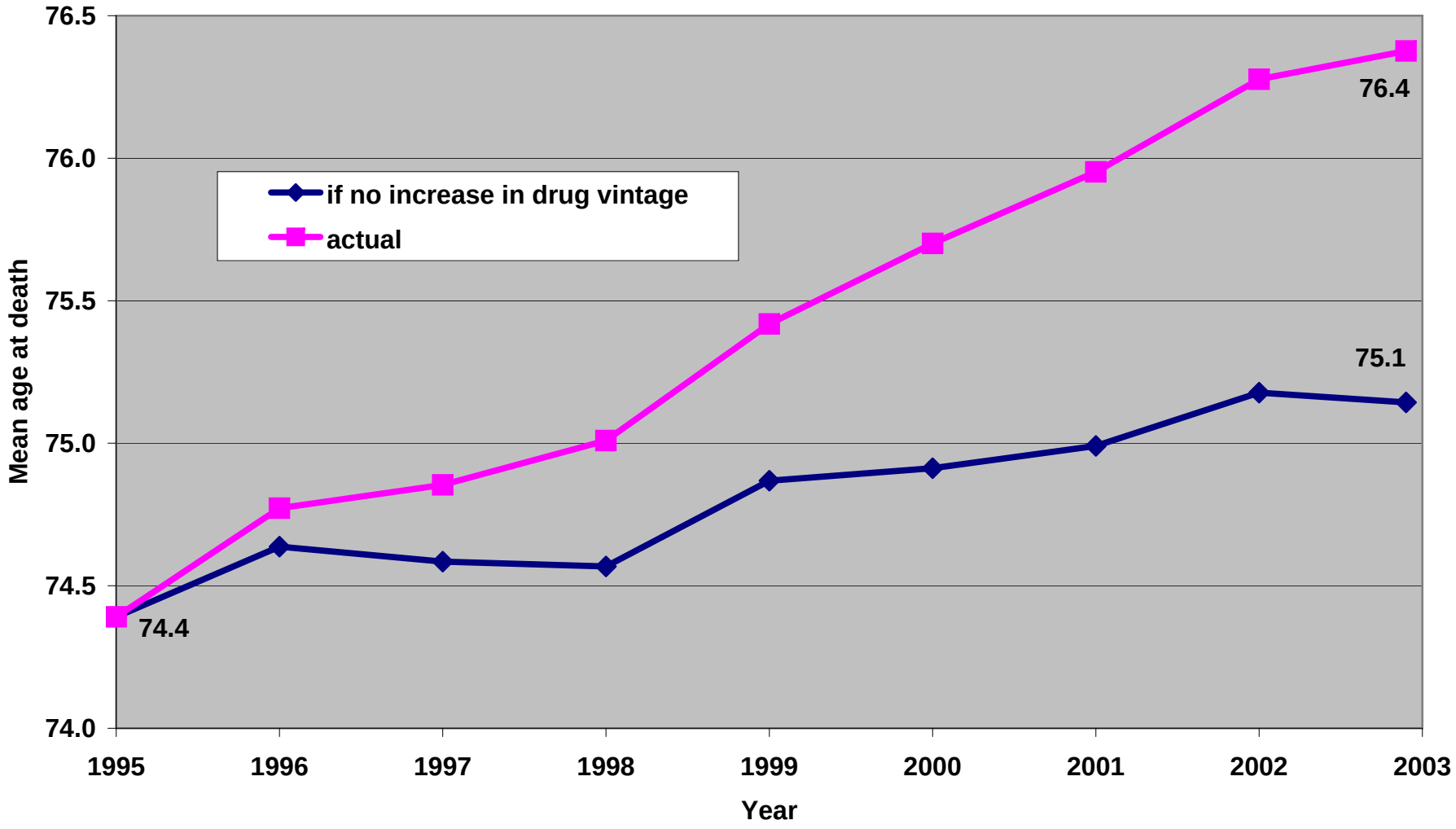


Table 1
Estimates of eq. (1)

Line	Parameter	Estimate	StdErr	tValue	Probt
	dep. var. = AGE_DEATH_{it}; weight = N_DEATH_{it}				
1	fda_year	0.182	0.091	1.99	0.0497
2	year 1995	-0.752	0.688	-1.09	0.2777
3	year 1996	-0.506	0.627	-0.81	0.4219
4	year 1997	-0.559	0.569	-0.98	0.3291
5	year 1998	-0.576	0.498	-1.16	0.2514
6	year 1999	-0.275	0.456	-0.60	0.5487
7	year 2000	-0.231	0.374	-0.62	0.5386
8	year 2001	-0.153	0.329	-0.46	0.6440
9	year 2002	0.034	0.304	0.11	0.9112
10	year 2003	0.000	.	.	.
	dep. var. = $LPYLL75_{it}$; weight = $(1/9)\ \Sigma_t \exp(LPYLL75_{it})$				
11	fda_year	-0.015	0.011	-1.36	0.1787
12	year 1995	-0.008	0.089	-0.09	0.9262
13	year 1996	-0.001	0.084	-0.01	0.9910
14	year 1997	0.013	0.077	0.16	0.8708
15	year 1998	0.000	0.069	0.00	0.9984
16	year 1999	-0.032	0.065	-0.49	0.6274
17	year 2000	-0.023	0.053	-0.44	0.6640
18	year 2001	-0.025	0.046	-0.54	0.5940
19	year 2002	-0.008	0.043	-0.19	0.8463
20	year 2003	0.000	.	.	.
	dep. var. = $LPYLL70_{it}$; weight = $(1/9)\ \Sigma_t \exp(LPYLL70_{it})$				
21	fda_year	-0.024	0.012	-2.00	0.0488
22	year 1995	-0.087	0.098	-0.89	0.3763
23	year 1996	-0.072	0.092	-0.78	0.4349
24	year 1997	-0.047	0.084	-0.55	0.5819
25	year 1998	-0.044	0.076	-0.58	0.5609
26	year 1999	-0.075	0.072	-1.05	0.2958
27	year 2000	-0.050	0.059	-0.85	0.3982
28	year 2001	-0.046	0.052	-0.88	0.3816
29	year 2002	-0.021	0.049	-0.43	0.6714
30	year 2003	0.000	.	.	.
	dep. var. = $LPYLL65_{it}$; weight = $(1/9)\ \Sigma_t \exp(LPYLL65_{it})$				
31	fda_year	-0.033	0.013	-2.53	0.0135
32	year 1995	-0.147	0.107	-1.37	0.1741
33	year 1996	-0.125	0.101	-1.24	0.2175
34	year 1997	-0.087	0.093	-0.94	0.3483
35	year 1998	-0.069	0.083	-0.83	0.4094
36	year 1999	-0.100	0.079	-1.28	0.2058
37	year 2000	-0.063	0.066	-0.97	0.3355
38	year 2001	-0.059	0.058	-1.01	0.3139
39	year 2002	-0.029	0.055	-0.53	0.5961
40	year 2003	0.000	.	.	.

Figure 2
Comparison of actual increase in mean age at death to the increase that would have occurred in the absence of any increase in drug vintage



Appendix Table 1
Data used to estimate eq. (1)

Disease	Year	Number of rx's	Mean vintage of rx's	Number of deaths	Mean age at death	Potential years of life lost before age 65
Digestive & Endocrine	1995	16,316,361	1982.8	7,448	73.0	23,090
Digestive & Endocrine	1996	17,133,504	1983.4	7,783	73.1	24,213
Digestive & Endocrine	1997	17,533,563	1984.0	8,154	72.7	28,023
Digestive & Endocrine	1998	17,918,165	1984.7	7,932	72.8	27,585
Digestive & Endocrine	1999	19,188,559	1985.1	8,321	73.4	26,993
Digestive & Endocrine	2000	20,147,558	1985.5	8,301	74.0	23,785
Digestive & Endocrine	2001	20,385,191	1986.1	8,403	74.2	24,213
Digestive & Endocrine	2002	22,400,650	1987.4	9,125	74.2	27,298
Digestive & Endocrine	2003	23,330,710	1988.7	9,222	74.4	26,658
Blood	1995	1,782,830	1953.2	871	54.8	13,788
Blood	1996	1,889,272	1953.3	433	72.4	1,995
Blood	1997	1,950,002	1953.7	372	73.9	1,658
Blood	1998	2,167,689	1954.2	436	72.5	2,358
Blood	1999	2,636,979	1954.8	450	71.1	2,655
Blood	2000	3,366,276	1958.9	413	72.5	2,143
Blood	2001	3,726,524	1964.2	408	72.3	1,818
Blood	2002	4,407,413	1967.9	428	74.1	1,870
Blood	2003	5,053,317	1970.2	454	74.5	1,670
Circulatory	1995	29,274,934	1983.0	53,407	77.6	65,548
Circulatory	1996	31,445,238	1984.1	53,990	77.9	64,778
Circulatory	1997	33,112,134	1984.9	53,636	78.1	65,435
Circulatory	1998	34,601,496	1985.9	51,787	78.2	62,868
Circulatory	1999	38,246,147	1986.9	51,303	78.5	61,273
Circulatory	2000	42,380,643	1987.7	49,687	78.7	59,848
Circulatory	2001	45,401,307	1988.2	49,326	78.8	61,038
Circulatory	2002	48,340,917	1988.6	50,294	79.1	58,803
Circulatory	2003	50,585,429	1989.0	48,835	79.1	61,090
Skin	1995	4,158,948	1964.6	250	80.5	260
Skin	1996	3,935,264	1966.3	175	80.9	130
Skin	1997	3,189,000	1964.9	240	78.9	515
Skin	1998	2,748,965	1964.8	260	80.5	178
Skin	1999	2,919,539	1966.3	289	79.1	433
Skin	2000	3,003,996	1967.2	252	80.0	253
Skin	2001	2,969,818	1968.0	265	80.6	203
Skin	2002	2,870,937	1968.1	334	80.0	455
Skin	2003	2,757,778	1968.4	305	80.7	183
Genitourinary	1995	6,272,147	1976.6	2,074	79.2	1,878
Genitourinary	1996	6,239,411	1976.6	2,244	79.6	1,890
Genitourinary	1997	5,471,427	1978.3	2,588	80.1	2,095
Genitourinary	1998	5,323,027	1979.3	2,697	80.3	2,230
Genitourinary	1999	5,701,087	1979.5	2,768	80.7	2,195
Genitourinary	2000	5,878,884	1980.0	2,692	80.6	2,043
Genitourinary	2001	6,013,677	1980.8	2,812	81.0	1,868
Genitourinary	2002	5,423,044	1981.2	2,983	81.1	1,850

Disease	Year	Number of rx's	Mean vintage of rx's	Number of deaths	Mean age at death	Potential years of life lost before age 65
Genitourinary	2003	4,244,974	1981.4	3,001	80.8	2,350
Infectious	1995	17,079,435	1972.3	1,070	68.7	7,463
Infectious	1996	16,263,453	1972.7	1,638	62.1	18,278
Infectious	1997	15,199,509	1973.1	1,522	67.1	12,308
Infectious	1998	14,470,051	1973.5	1,454	68.7	10,513
Infectious	1999	13,523,718	1974.5	1,603	69.9	10,458
Infectious	2000	13,504,891	1974.7	1,646	70.4	10,290
Infectious	2001	13,487,633	1974.6	1,675	70.6	10,413
Infectious	2002	13,096,864	1974.8	1,790	71.0	10,578
Infectious	2003	12,745,328	1975.1	1,754	72.0	8,958
Neoplasms	1995	487,908	1972.6	34,368	70.1	115,888
Neoplasms	1996	534,042	1972.7	35,252	70.3	118,093
Neoplasms	1997	570,489	1973.2	35,363	70.3	117,395
Neoplasms	1998	605,012	1973.8	35,609	70.5	117,388
Neoplasms	1999	697,621	1973.7	35,856	70.9	114,630
Neoplasms	2000	829,760	1976.1	36,374	71.2	111,575
Neoplasms	2001	923,797	1977.7	37,497	71.3	115,190
Neoplasms	2002	1,008,548	1978.7	38,426	71.5	115,128
Neoplasms	2003	1,049,238	1979.3	38,392	71.5	115,990
Musculoskeletal	1995	5,956,861	1978.6	734	75.5	1,698
Musculoskeletal	1996	5,833,568	1978.7	794	76.6	1,438
Musculoskeletal	1997	5,636,378	1978.9	792	75.6	1,815
Musculoskeletal	1998	5,430,948	1978.9	751	75.1	1,805
Musculoskeletal	1999	5,669,394	1979.0	862	76.3	1,735
Musculoskeletal	2000	6,784,075	1983.7	852	76.2	1,900
Musculoskeletal	2001	9,639,186	1990.2	896	77.4	1,315
Musculoskeletal	2002	11,380,343	1991.5	1,015	77.5	1,715
Musculoskeletal	2003	12,012,146	1992.0	999	77.6	1,790
Mental & Nervous	1995	22,947,117	1973.6	6,142	72.5	36,365
Mental & Nervous	1996	23,993,807	1974.7	6,631	73.6	35,608
Mental & Nervous	1997	24,577,524	1976.6	6,591	71.9	40,780
Mental & Nervous	1998	25,121,805	1978.1	6,589	71.8	41,660
Mental & Nervous	1999	26,843,646	1979.5	6,698	73.6	33,388
Mental & Nervous	2000	28,269,319	1980.9	7,113	73.2	38,765
Mental & Nervous	2001	30,211,670	1982.6	6,908	75.9	25,475
Mental & Nervous	2002	31,017,021	1983.9	7,794	76.8	24,618
Mental & Nervous	2003	31,579,863	1984.8	7,565	76.9	24,383
Respiratory	1995	11,063,249	1981.1	9,431	75.6	17,180
Respiratory	1996	11,875,706	1982.0	10,294	76.5	15,228
Respiratory	1997	11,297,442	1982.5	10,349	76.8	15,028
Respiratory	1998	10,649,965	1984.3	9,614	76.8	14,543
Respiratory	1999	11,232,232	1985.2	9,613	77.2	13,365
Respiratory	2000	11,419,935	1986.1	10,907	77.9	13,695
Respiratory	2001	10,362,935	1986.0	10,626	77.7	14,515
Respiratory	2002	10,165,471	1986.9	11,668	78.3	13,790
Respiratory	2003	9,751,515	1988.8	11,892	78.7	14,380
Eye & Ear	1995	5,857,233	1969.4	16	47.5	440

Disease	Year	Number of rx's	Mean vintage of rx's	Number of deaths	Mean age at death	Potential years of life lost before age 65
Eye & Ear	1996	5,734,316	1968.4	18	53.1	390
Eye & Ear	1997	5,499,593	1968.6	9	69.7	75
Eye & Ear	1998	5,478,050	1970.0	15	71.8	93
Eye & Ear	1999	5,927,943	1973.2	11	64.3	143
Eye & Ear	2000	6,298,017	1975.9	10	59.5	143
Eye & Ear	2001	6,671,412	1977.5	10	74.5	40
Eye & Ear	2002	6,847,396	1978.7	8	63.1	113
Eye & Ear	2003	6,820,565	1979.4	15	64.5	213