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SOURCES OF U.S. LONGEVITY INCREASE, 1960 -1997

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Abstract

Between 1960 and 1997, life expectancy at birth of Americans increased approximately 10% – from 69.7 to 76.5 years – and it has been estimated that the value of life extension during this period nearly equaled the gains in tangible consumption. While life expectancy has tended to increase, there have been substantial fluctuations in the rate of increase. In this paper we investigate whether an aggregate health production function can help to explain the annual time-series behavior of U.S. longevity since 1960. We view longevity as the output of the health production function, and output fluctuations as the consequence of fluctuations in medical inputs (expenditure) and technology. We estimate longevity models using annual U.S. time-series data on life expectancy, health expenditure, and medical innovation. Reliable annual data are available for only one type of innovation – new drugs – but pharmaceutical R&D accounts for a significant fraction of total biomedical research. The empirical analysis provides strong support for the hypothesis that both medical innovation (in the form of new drug approvals) and expenditure on medical care (especially public expenditure) contributed to longevity increase during the period 1960-1997. Increased drug approvals and health expenditure per person jointly explain just about 100% of the observed long-run longevity increase. The estimates provide strong evidence against the null hypothesis that public health expenditure has no effect on longevity, but not against the null hypothesis that private health expenditure has no effect on longevity. This is at least partly attributable to the fact that public health expenditure exhibited much greater variability during the sample period than private health expenditure. The estimates imply that the medical expenditure needed to gain one life-year is about $11,000, and that the pharmaceutical R&D expenditure needed to gain one life-year is about $1,345. This suggests that increased development of new drugs may be a more cost-effective way of increasing life expectancy than increased medical expenditure in general. Previous researchers have estimated that the average value of a life-year is approximately $150,000. This figure implies that the benefit-cost ratio of general medical expenditure is 13.6, and that the ratio for pharmaceutical R&D exceeds 100.

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Between 1960 and 1997, life expectancy at birth increased approximately 10%, from 69.7 to 76.5 years.[1] Nordhaus (1999) estimates that the value of life extension during this period nearly equaled the gains in tangible consumption.[2]

While life expectancy has tended to increase since 1960, as Figure 1 indicates, there have been substantial fluctuations in the rate of increase. Life expectancy increased

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[1] Life expectancy (e_x)--the average number of years of life remaining for persons who have attained a given age (x)--is the most frequently used life table statistic. Calculation of the complete life table is derived from the probability of death (q_x), which depends on the number of deaths (D_x) and the midyear population (P_x) for each single year of age (x) observed during the calendar year of interest. There are two types of life tables—the generation or cohort life table and the current life table.

The generation life table provides a ''longitudinal'' perspective in that it follows the mortality experience of a particular cohort, all persons born in the year 1900, for example, from the moment of birth through consecutive ages in successive calendar years. Based on age-specific death rates observed through consecutive calendar years, the generation life table reflects the mortality experience of an actual cohort from birth until no lives remain in the group. To prepare just a single complete generation life table requires data over many years. It is not feasible to construct generation life tables entirely on the basis of actual data for cohorts born in this century. It is necessary to project data for the incomplete period for cohorts whose life spans are not yet complete.

The better-known current life table may, in contrast, be characterized as ''cross-sectional.'' Unlike the generation life table, the current life table does not represent the mortality experience of an actual cohort. Rather, the current life table considers a hypothetical cohort and assumes that it is subject to the age-specific death rates observed for an actual population during a particular period. Thus, for example, a current life table for 1997 assumes a hypothetical cohort subject throughout its lifetime to the age-specific death rates prevailing for the actual population in 1997. The current life table may thus be characterized as rendering a ''snapshot'' of current mortality experience, and shows the long-range implications of a set of age-specific death rates that prevailed in a given year. The life expectancy data analyzed in this paper are based on the current life table, not on the generation life table.

[2] Nordhaus (1999), along with Murphy and Topel (1999), offer parallel estimates of the value of recent increases in longevity. To the casual observer it hardly seems possible – and may seem morally offensive – to put a dollar value on human life. But modern economics has devised a credible way around these imponderables, inferring the value people put on life from what they must be "bribed" in everyday settings to incur small but predictable increases in the risk of death. Let's say that moving from a factory line to outdoor construction increases a worker's chance of a fatal accident by one in 10,000 each year. In other words, if 10,000 workers made the shift, expected on-the-job fatalities would rise by one per year. Suppose further that to induce 10,000 workers to play this death lottery voluntarily, an employer would have to pay an extra $500 annually to each worker for a total of $5 million. One of these new construction workers is likely to die in return for the group gaining $5 million. Thus the value of one life in this example is said to be $5 million.

Estimates from the dozen or so work-related studies since the mid-1970s put the value of a statistical life in the relatively narrow $3 million-to-$7 million range. Using the relatively conservative estimate of $3 million for the average value of avoiding one death to calculate the value of extending life, Nordhaus estimates that in the 1975-1995 period the value of life extension nearly equaled the gains in tangible consumption.

at an average annual rate of 0.25%; it increased more than 0.70% in 1961, 1974, and 1975, and declined more than 0.25% in 1963, 1968, 1980, and 1993. Measurement error is unlikely to account for much of the fluctuations in life expectancy: as noted in Anderson (1999, p. 34), "the annual life tables are based on a complete count of all reported deaths," and there are about 2 million deaths per year.

Nor does growth in real per capita income (GDP) appear to offer a plausible explanation for the increase in life expectancy. As Figure 2 indicates, the period in which life expectancy increased most rapidly (1973-75) was a period of dismal macroeconomic performance. Indeed, there is a significant *negative* correlation (p-value = .04) between the annual rates of life expectancy increase and GDP increase.

In this paper we investigate whether an *aggregate health production function* can help to explain the annual time-series behavior of U.S. life expectancy since 1960. We view life expectancy as the *output* of the health production function. In general, production functions have two types of arguments: inputs and technology. Increases in input result in *movements along* the production function. Improvements in technology result in productivity increases, or *shifts* of the production function.[3]

We hypothesize that life expectancy in year t is a function of the stock of medical innovations available in year t and the stock of of real per capita health expenditure in year t, assumed to be a distributed-lag function of real per capita health expenditure in year t – i (i = 0, 1, 2,…):

$$LE_t = f(EXP\_STOCK_t, INV\_STOCK_t) + u_t \qquad (1)$$

where

LE = life expectancy at birth
$EXP\_STOCK_t = \Sigma_{i=0} (1 - \delta_1)^i EXP_{t-i}$ = the "health expenditure stock"

EXP = real per capita health expenditure

δ_1 = the "depreciation" rate of medical expenditure ($0 < \delta_1 < 1$)

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[3] $\text{output} = \frac{\text{output}}{\text{input}} * \text{input} = \text{productivity} * \text{input}$, and productivity = f(technology).

$INV\_STOCK_t = \Sigma_{i=0} (1 - \delta_2)^i INV_{t-I}$ = the "medical innovation stock"

INV = the number of innovations

δ_2 = the "depreciation" (or obsolescence) rate of medical innovations ($0 \leq \delta_2 < 1$)[4]

u = disturbance.[5]

This specification imposes the restriction that the coefficients on lagged innovations and expenditures decline geometrically with respect to time. As Greene (1997, pp. 786-96) notes, estimation of the unrestricted finite distributed lag model is likely to be ineffective because (1) the typical time series is fairly short, so the unrestricted model will consume an excessive number of degrees of freedom, and (2) multicollinearity is likely to be quite severe. These considerations have led researchers to formulate compact parametric models that allow infinite lags, but require only a small number of parameters. The geometric lag model is a common choice. This model incorporates infinite lags but assigns arbitrarily small weights to the distant past.

If equation (1) were linear ($LE_t = \alpha + \beta_1 EXP\_STOCK_t + \beta_2 INV\_STOCK_t + u_t$), and the innovation and expenditure depreciation rates were identical ($\delta_1 = \delta_2 = \delta$), then one could reformulate equation (1) in autoregressive form as follows[6]:

$$LE_t = \alpha\delta + (1 - \delta) LE_{t-1} + \beta_1 EXP_t + \beta_2 INV_t + v_t \qquad (2)$$

where $v_t = u_t - (1 - \delta) u_{t-1}$. The regression of life expectancy on its own lagged value, current health expenditure, and the current flow of innovations, will yield estimates of the parameters δ, β_1, and β_2. From these one can estimate both the short-run and long-run impacts on longevity of changes in health expenditure and innovation flows. For

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[4] Whether or not medical innovations are subject to depreciation or obsolescence (i.e., whether $\delta_2 > 0$) is an issue discussed below.

[5] In our empirical analysis, we also include per capita income (GDP) and a time trend as control variables.

[6] If the disturbances of eq. (1) are serially independent, the disturbances of eq. (2) are serially dependent, which has implications for the estimation procedure. When $\delta_1 \neq \delta_2$, the autoregressive equation also includes LE_{t-2}, EXP_{t-1}, and INV_{t-1} as regressors. See Johnston (1984, p. 347).

example, the longevity impact of a sustained unit increase in the number of innovations is (β_2 / δ).

We don't really believe that longevity is a *linear* function of the expenditure and innovation stocks. It seems more likely that these stocks have diminishing marginal effects on longevity, i.e. that the relationship is log-linear. However if one specifies eq. (1) to be log-linear, due to the linear form of the accumulation equations one can no longer derive a simple autoregressive equation like eq. (2). The theoretically more appropriate functional form does not yield a tractable estimating equation.

Instead of estimating eq.(2) using data on the *levels* of the variables LE, EXP, and INV, we will estimate it using data on the logarithms of these variables. In this setting, β_2 is the short-run elasticity of longevity with respect to innovation, and (β_2 / δ) is the long-run elasticity.

Data

Estimation of the longevity model (2) requires data on health expenditure and medical innovation as well as on life expectancy. We were able to find annual U.S. time-series data on each of these variables for the period 1960-1997. We describe the sources and properties of the data below.

Longevity. Annual data on life expectancy at birth are given in Anderson (1999, Table 12). That publication also provides data on life expectancy at ages greater than 0 (1, 5, 10, 15,…,100), but only at decennial frequency. Of course, changes in life expectancy at ages greater than zero are reflected, to varying degrees, in changes in life expectancy at birth. Figure 3 reveals that during the sample period, movements in life expectancy at birth tracked those in life expectancy at age 40 very closely.

Anderson (1999, Table 12) also provides annual data on life expectancy at birth, by race (white vs. black). Figure 4 shows data on the life expectancy of blacks at birth as a percent of the life expectancy of whites at birth. On average, white longevity is about 10% greater. The relative longevity of blacks tended to decline from 1960 to 1970, increased steadily from 1970 to its peak in 1982, declined steadily from 1982 to 1989,

and increased slightly since then. In addition to estimating an overall longevity model, we will estimate separate models, by race.

Health expenditure. Annual data for 1960-97 on national health expenditures, in current dollars, by source of funds (public vs. private) are produced by the National Health Statistics Group, Office of the Actuary, Health Care Financing Administration. To obtain per capita expenditures in constant dollars, we deflated these figures using the BLS consumer price index for medical care (U.S. city average, base period: 1982-84=100, Series ID: CUSR0000SAM) and divided by the population.

Real public and private health expenditure per are shown in Figure 5. Public and private health expenditure exhibited different, sometimes opposite, behavior during the 1960-97 period. Between 1965 and 1967, there was a 64% increase in real per capita public health expenditure, due to the establishment of the Medicare[7] and Medicaid[8] programs. For each $100 increase in public spending, there was about a $39 reduction in private spending. The government's share of national health expenditure increased from under 25% to over 37% in two years. Public spending also increased more than private spending since 1967; by 1997, the government was financing almost half (46.2%) of national health expenditure.

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[7] As part of the Social Security Amendments of 1965, Title XVIII of the Social Security Act established a health insurance program, commonly known as "Medicare", for persons 65 and over, to complement the retirement, survivors and disability insurance benefits under Title II of the Act. Medicare was first implemented in 1966, and by the end of that year, 3.7 million persons had received at least some health care services covered by Medicare. Today Medicare is the nation's largest health insurance program, covering approximately 39 million Americans. Medicare consists of two primary parts: Hospital Insurance (HI), also known as "Part A," and Supplementary Medical insurance (SMI), also known as "Part B". In 1973, other groups became eligible for Medicare benefits: persons who are entitled to Social Security or Railroad Retirement disability benefits for at least 24 months; persons with end-stage renal disease (ESRD) requiring continuing dialysis or kidney transplant; and certain otherwise non-covered aged persons who elect to *buy into* Medicare.

[8] Title XIX of the Social Security Act established a program which provides medical assistance for certain individuals and families with low incomes and resources. The program, known as Medicaid, became law in 1965 as a jointly funded cooperative venture between the Federal and State governments to assist States in the provision of adequate medical care to eligible needy persons. Medicaid is the largest program providing medical and health-related services to America's poorest people.

The share of health expenditure that is publicly funded varies across demographic groups. Data from the 1996 Medical Expenditure Panel Survey indicate that the fraction of health expenditure that was publicly funded was 32.4% for whites and 50.3% for blacks. Virtually all of this difference was due to a difference in the proportion of expenditures paid for by Medicaid: 6.8% for whites and 24.7% for blacks.

As noted above, we can estimate the longevity model (eq. (2)), by race; we can also disaggregate the expenditure variable EXP into its public and private components:

$$LE_t = \alpha\delta + (1 - \delta)\, LE_{t-1} + \beta_{1G}\, EXP\_G_t + \beta_{1P}\, EXP\_P_t + \beta_2\, INV_t + v_t \qquad (3)$$

where EXP_G = government-funded health expenditure and EXP_P = privately-funded health expenditure. Given the greater relative importance of publicly-funded health expenditure to blacks, one would expect the ratio (β_{1G} / β_{1P}) to be larger in the black longevity equation than it is in the white longevity equation.

We recognize that causality between longevity and average medical expenditure is likely to run in both directions. In any given year, old people tend to spend more on medical care than young people. Figure 6 illustrates this tendency using cross-sectional data from the 1977 National Medical Care Expenditure Survey. In 1977, the average 75-84 year-old spent 27% more than the average 65-74 year old; the average 85+ year-old spent 62% more than the average 65-74 year old.[9] As life expectancy increases, the average age of the population (and the proportion above a high age such as 65) increases, so per capita medical expenditure increases.[10] To ensure that we are measuring the effect of expenditure on longevity rather than the effect of longevity (age) on expenditure, we will include only *previous* health expenditure on the right-hand side of the longevity equation.[11]

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[9] Moreover, the age/medical expenditure profile was steeper in 1996 than it was in 1977.

[10] However, the aging of the population accounts for a very small fraction of the total observed increase in per capita medical expenditure.

[11] Since this equation also includes lagged longevity, significance of the lagged expenditure term would allow us to reject the null hypothesis that expenditure does not Granger-cause longevity. See Greene (1997, pp. 816-817) for a discussion of Granger causality testing.

Medical innovation. There are many kinds of medical innovations, including new drugs, medical devices, and surgical and diagnostic procedures. However drugs is the only type of innovation for which reliable annual data are available. We obtained from the FDA a list of all new molecular entities (NMEs) approved during the period 1950-1993. Using data posted on the FDA website, we were able to extend the coverage of this list to the period 1950-1999.

Although new drugs represent only one type of medical innovation, pharmaceutical R&D accounts for a significant fraction of total biomedical research.[12] In 1993, pharmaceutical industry R&D accounted for 61.3% of industry-funded health R&D, and for 31.0% of total health R&D. Moreover, new drugs are usually thought to embody knowledge generated by both publicly- and privately-funded research.

If the rate of introduction of new drugs were very stable from year to year, it would be hard to discern the effect of pharmaceutical innovation on outcomes and expenditure. In practice, however, as Figure 7 indicates, the innovation rate fluctuates considerably. Part of this is due to the inherent randomness of the drug development and approval process. But major changes in government policy also clearly influence the number of new drugs approved. Policy-induced and other changes in the rate of new drug approval facilitate statistical inference about the impact of pharmaceutical innovation (and the policies themselves) on longevity. We will briefly describe four such policy changes.

1962 Kefauver-Harris Amendment. As a result of the thalidomide tragedy, in 1962, Congress passed the Kefauver-Harris Amendment to the Federal Food, Drug, and Cosmetic Act (FDCA), which required extensive animal pharmacological and toxicological testing before a drug could be tested in humans. The data from these studies must be submitted in the form of an IND ("Notice of Claimed Investigational Exemption for a New Drug") and approved by the FDA before clinical studies can begin. The amendment also required that manufacturers submit to the FDA "substantial evidence" of the unapproved (investigational) drug's efficacy, as well as safety, in the form of an NDA

[12] Company R&D, drugs and medicines industry, 1993: $9,625 m. (Source: NSF); Industry-funded health R&D, 1993: $15,711 m. (Source: NIH, reported in *Health, U.S.*); Total health R&D, 1993: $31,032 m. (Source: NIH, reported in *Health, U.S.*)

("New Drug Application"). Therefore, in addition to safety, the manufacturer was now required to demonstrate efficacy (effectiveness), as well.[13] Passage of this amendment appears to have led to a significant, roughly 10-year decline in the number of new drugs approved.

Title XVIII of the Social Security Amendments of 1965 (Medicare). Although Medicare does not pay for most prescription drugs, Medicare Part B (Supplementary Medical insurance) pays part of the cost of a service that is complementary with (necessary to receive) prescription drugs: doctor visits.[14] The data shown in Figure 8 suggests that Medicare had a significant effect on utilization of ambulatory care by the elderly. Between 1964—immediately before Medicare was established—and 1990, the probability that a person over 65 had not seen a doctor in the last two years declined from 21.0% to 8.0%. The corresponding probability for people under 65 (who are generally not covered by Medicare) also declined, but by much less.

Title XIX of the Social Security Amendments of 1965 (Medicaid). Prescription drugs are covered under Medicaid. In 1964, less than 4% of national expenditure on prescription drugs was publicly funded. In 1998, over 20% was publicly funded, and the Medicaid program accounted for over 80% of this funding.

1992 Prescription Drug User Fee Act. This act caused a (temporary) increase in drug approvals due to a 60% reduction in mean drug approval times.

Recall that the stock of medical innovations was defined as a weighted sum of past innovations ($INV\_STOCK_t = \Sigma_{i=0} (1 - \delta_2)^i INV_{t-i}$), where the weights depend on the "depreciation" (or obsolescence) rate of medical innovations, δ_2. In principle, the depreciation rate might be zero, so that $INV\_STOCK_t = \Sigma_{i=0} INV_{t-i}$: the innovation stock is the unweighted sum of all past innovations. According to that hypothesis, longevity in

[13] "Substantial evidence" is defined by Section 505 of the FD&C Act as "evidence consisting of adequate and well-controlled investigations, including clinical investigations, by experts qualified by scientific training and experience to evaluate the effectiveness of the drug involved, on the basis of which it could be fairly and responsibly concluded by such experts that the drug will have the effect it purports or is represented to have under the conditions of use prescribed, recommended, or suggested in the labeling thereof." FDCA [505(d)].

[14] At least one prescription drug is prescribed in about 60% of doctor visits; this percentage is even higher for the Medicare population.

year t would depend on the number of drugs ever approved up until year t (regardless of when they were approved).

We have evidence, however, that casts doubt on the hypothesis of zero depreciation. As mentioned above, the FDA provided us with a list of all drugs (new molecular entities) approved since 1950. We also have a comprehensive database (Multum's Lexicon[15]) of all drugs marketed in 1999. By comparing these two lists, we can determine how many drugs approved in each year beginning in 1950 were no longer being marketed in 1999. As Figure 9 reveals, about 20% of the drugs approved during 1950-93 were no longer on the market in 1999. The earlier the drug was approved, the lower the probability of being on the market in 1999: 28.6% of drugs approved in the 1950s had disappeared by 1999.

If the eventual disappearance of drugs from the market were inevitable—if it occurred regardless of the pace of subsequent innovation—then the fact that obsolescence occurs implies that a temporary increase in the number of innovations would have only a temporary impact on longevity. A permanent increase in the number of new drugs approved would be required to achieve a permanent longevity increase. However evidence presented in Lichtenberg and Philipson (2000) suggests that a drug's eventual disappearance (or the decline of its sales as it ages) is not automatic or exogenous, but is the result of "creative destruction": the entry of new, superior, products. Changes in the number of innovations will affect the obsolescence rate. Consequently, a temporary increase in the number of innovations could perhaps have a permanent impact on longevity.

Empirical results

Summary statistics for the 1960-1997 sample period are reported in Table 1.[16] Maximum likelihood estimates of longevity equations are presented in Table 2. All variables are expressed in natural logarithms, and all equations are estimated with a correction for first-order serial correlation of disturbances.

[15] See http://www.multum.com/Lexicon.htm.
[16] The underlying data are presented in Appendix Table 1.

The first column presents estimates of the most basic model, the regression of life expectancy at birth in year t (le) on its own lagged value (le_{-1}), the number of new molecular entities approved in year t (nme), and real per capita health expenditure in year t (exp). The coefficients on nme and exp are positive and highly statistically significant, which suggests that longevity is positively affected by current and lagged drug approvals and health expenditure.

To guard against the possibility that the positive partial correlation between longevity and health expenditure may be due to the effect of the former on the latter, rather than the reverse, in column (2) we substitute lagged health expenditure for current health expenditure.[17] This substitution has very little effect on the estimates (it actually *increases* the expenditure coefficient by about 10%), which seems to cast doubt on the reverse-causality hypothesis.

In column (3), we include two additional variables as "controls": a time trend, and real per capita income (GDP). Neither of these variables has statistically significant effects on longevity, and their inclusion scarcely changes the innovation and health expenditure coefficients.

These estimates permit us to perform a "growth accounting" exercise, i.e. to compute how much of the long-run growth in longevity during 1960-97 may be attributed to long-run growth in new drug approvals and in per capita health expenditure. To calculate the contribution of each of the two sources to longevity increase, we multiply the average rate of growth (from Table 1) by the respective long-run elasticity (from column 2 of Table 2).[18] These calculations are shown below.

[17] This specification corresponds to the hypothesis that current longevity depends on last year's accumulated stock of health expenditure.

[18] We use the estimates from column 2 rather than column 3 because neither lagged gdp nor the time trend were statistically significant.

Source of longevity growth	mean growth rate	long run elasticity (Table 2, col. 2)	contribution to longevity growth rate	contribution as % of actual longevity growth rate
Number of new molecular entities approved	0.0279	0.024	0.000679	27.0%
Real health expenditure per capita	0.0260	0.074	0.001918	76.1%

Increased drug approvals and health expenditure per person jointly explain just about 100%--slightly more: 103%--of the observed long-run longevity increase. The contribution of health expenditure is about three times as large as the contribution of new drug approvals. However the average level of health expenditure is much larger than the average level of (R&D) expenditure associated with new drug approvals. In 1993, the ratio of health R&D funding from all sources to national health expenditure was 3.5%; the ratio of pharmaceutical industry R&D expenditure to national health expenditure was probably just over 1%. The fact that the estimated relative longevity contribution of new drugs is about 30 times as large as relative expenditure on drug research suggests that a dollar of drug research tended to increase life expectancy more than a dollar of general medical expenditure. We will return to this point after presenting the rest of our estimates.

In column (4), we disaggregate total health expenditure per person into its two components, public and private health expenditure per person (exp_g and exp_p, respectively). If the longevity effect of an additional dollar of public health expenditure were the same as that of an additional dollar of private expenditure, the ratio of the exp_g coefficient to the exp_p coefficient should be equal to the average ratio of public to private expenditure, which is about 61%. The ratio of these two coefficients is higher than this—79%. This suggests that public health expenditure has a higher marginal effect on longevity than private health expenditure. The difference between these two ratios is not statistically significant, however.

Although the point estimate of the exp_p coefficient is larger than that of the exp_g coefficient, only the latter is statistically significantly different from zero. Thus,

the estimates provide strong evidence against the null hypothesis that public health expenditure has no effect on longevity, but not against the null hypothesis that private health expenditure has no effect on longevity.

The exp_g coefficient is estimated much more precisely than the exp_p coefficient (the standard error of the former is only 29% as large as that of the latter) because public health expenditure exhibited much greater variability during the sample period than private health expenditure. The standard deviation of the growth rate of public health expenditure was 1.7 times as great, and whereas private health expenditure growth ranged between –7% to +8%, public health expenditure growth ranged from –2% to +25%.

Annual growth in the number of new drugs approved exhibited even greater volatility than growth in public health expenditure: the standard deviation of the growth rate of the number of new drugs approved was over 7 times as great as the standard deviation of private health expenditure growth. This volatility (relatively low serial correlation) of drug approvals suggests that it might be feasible to relax the assumption of strictly geometric decay of coefficients on lagged drug approvals, by including lagged as well as current values of nme in the model. Column (5) displays an equation including nme_{-1} as well as nme. Both coefficients are highly statistically significant, and their magnitudes indicate that a new drug approval in year t has about the same impact on longevity in year t+1 as it has on longevity in year t.[19] However inclusion of nme_{-1} has virtually no effect on the estimate of the long-run elasticity of longevity with respect to new drug approvals, which is about .036.

The low serial correlation of new drug approvals also provides us with the opportunity to investigate the direction of causality between approvals and longevity. In column (6) we include the number of drugs approved in year t+1 in the year-t longevity equation. If the coefficient on this variable were positive and significant, this would suggest that (for some reason) increases in longevity this year cause more drugs to be approved next year, rather than the reverse. But the coefficient on nme_{+1}, in contrast to

[19] When additional lagged values of nme (nme_{-2}, nme_{-3},…) are included, the coefficients on them are not statistically significant.

those on nme and nme_{-1}, is not significantly different from zero, suggesting that causality runs in only one direction, from new drug approvals to longevity.[20]

All of the estimates presented so far have been of models of longevity of the entire population, i.e. of whites and blacks. In the last two columns of Table 2, we report estimates of longevity models estimated separately, by race. The new drug approvals coefficient is highly statistically significant in both equations, indicating that the longevity of both races is increased by pharmaceutical innovation. The estimated long-run elasticity of longevity with respect to new drugs approved is almost three times as large for blacks as it is for whites, a result we did not expect and do not, at present, have an explanation for. Another (possibly related) puzzle is the significant negative coefficient on the time trend in the black longevity equation. This implies that, in the absence of health expenditure growth and new drug approvals, black longevity would have *declined* during this period.

The point estimates of the private health expenditure coefficient are almost identical in the white and black longevity equations, but the coefficient is statistically significant only in the former equation. Thus, we can reject the null hypothesis that privately-funded health expenditure does not affect longevity in the case of whites, but not in the case of blacks. The public health expenditure coefficient is significant in both equations, but its magnitude is over twice as large in the black longevity equation as it is in the white longevity equation. This is not surprising, given the fact that the proportion of health expenditures that are publicly funded is larger for blacks than it is for whites. Indeed, as Figure 10 shows, for both races the ratio of the exp_g coefficient to the exp_p coefficient is very close to the 1996 ratio of public to private expenditure. This is consistent with the hypothesis that, for both races, the longevity effect of an additional dollar of public health expenditure is similar to that of an additional dollar of private expenditure.

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[20] Granger causality testing within a vector autoregression (VAR) framework yields the same conclusion. When the growth rates of le and nme are both regressed on the first three lagged growth rates of both variables, the null hypothesis that nme does not Granger-cause le is easily rejected (p-value = .02), but the null hypothesis that le does not Granger-cause nme cannot be rejected (p-value = .93). Similar results are obtained when the VAR includes health expenditure growth and gdp growth. I am grateful to Charles Himmelberg for helping me with the VAR analysis.

We think that the preceding analysis provides strong support for the hypothesis that both medical innovation (in the form of new drug approvals) and expenditure on medical care (especially public expenditure) contributed to longevity increase during the period 1960-1997. Now we will use these estimates to calculate the "bang per buck", or its inverse: the medical care expenditure or pharmaceutical R&D expenditure per life-year gained.

These calculations are summarized in Table 3. Medical care expenditure per life-year gained is calculated in the first column. The starting point is the long-run elasticity of longevity with respect to total health expenditure, estimated in column (3) of Table 2 to be .0906. This means that a permanent 1% increase in per capita health expenditure would lead to a .0906% increase in life expectancy at birth. The sample mean value of per capita health expenditure is $1306 (in 1982-84 dollars), and the sample mean value of life expectancy is 73.1 years. Evaluating the elasticity at the sample mean implies that a permanent $1 increase in per capita health expenditure would increase life expectancy at birth by .0051 years (1.9 days). There are approximately 4 million Americans born each year, so the total number of life-years gained per year from a permanent $1 increase in per capita health expenditure is 20,274. The annual cost of a permanent $1 increase in per capita health expenditure is $1 times the U.S. population, which averaged about 224 million during the sample period. Hence the cost of medical care per life-year gained is about $11,000.

In the second column, we calculate pharmaceutical R&D expenditure per life-year gained. The long-run elasticity of longevity with respect to the number of new drugs approved is estimated in column (3) of Table 2 to be .0265. The sample mean number of drugs approved per year is 20.8. Evaluating the elasticity at the sample mean implies that, if one additional drug were approved every year, life expectancy at birth would increase by .093 years (just over a month). The total number of life-years gained per year from a permanent unit increase in new drug approvals is about 372 thousand. The average cost of obtaining FDA approval of a new drug is generally thought to be in the neighborhood of $500 million. Hence we estimate pharmaceutical R&D expenditure per life-year gained to be about $1345.

This suggests that increased development of new drugs may be a more cost-effective way of increasing life expectancy than increased medical expenditure in general: pharmaceutical R&D expenditure per life-year gained is about one eighth of the cost of medical care per life-year gained. Although overall medical care appears to be less cost-effective, recent research by Nordhaus and by Topel and Murphy suggests that the benefits to it greatly exceed the costs. They estimate that the average value of a life-year is approximately $150,000. This figure implies that the benefit-cost ratio of general medical expenditure is 13.6, and that the ratio for pharmaceutical R&D is over 100! (111.5).

Conclusions

Between 1960 and 1997, life expectancy at birth increased approximately 10%, from 69.7 to 76.5 years. Nordhaus (1999) estimates that the value of life extension during this period nearly equaled the gains in tangible consumption.

While life expectancy has tended to increase since 1960, there have been substantial fluctuations in the rate of increase. In this paper we investigate whether an aggregate health production function can help to explain the annual time-series behavior of U.S. life expectancy since 1960. We view life expectancy as the *output* of the health production function. In general, production functions have two types of arguments--inputs and technology—and output fluctuations are attributable to fluctuations in both of these.

We estimate the longevity model using annual U.S. time-series data on life expectancy, health expenditure, and medical innovation for the period 1960-1997. Reliable annual data are available for only one type of innovation: new drugs. Although new drugs represent only one type of medical innovation, pharmaceutical R&D accounts for a significant fraction of total biomedical research. In 1993, pharmaceutical industry R&D accounted for 61.3% of industry-funded health R&D, and for 31.0% of total health R&D.

The empirical analysis provides strong support for the hypothesis that both medical innovation (in the form of new drug approvals) and expenditure on medical care

(especially public expenditure) contributed to longevity increase during the period 1960-1997. Increased drug approvals and health expenditure per person jointly explain just about 100% of the observed long-run longevity increase. The fact that the estimated relative longevity contribution of new drugs is about 30 times as large as relative expenditure on drug research suggests that a dollar of drug research tended to increase life expectancy more than a dollar of general medical expenditure.

The estimates provide strong evidence against the null hypothesis that public health expenditure has no effect on longevity, but not against the null hypothesis that private health expenditure has no effect on longevity. This is at least partly attributable to the fact that public health expenditure exhibited much greater variability during the sample period than private health expenditure. The longevity of both races was increased by pharmaceutical innovation.

The cost of medical care per life-year gained is about $11,000. We estimate pharmaceutical R&D expenditure per life-year gained to be about $1345. This suggests that increased development of new drugs may be a more cost-effective way of increasing life expectancy than increased medical expenditure in general: pharmaceutical R&D expenditure per life-year gained is about one eighth of the cost of medical care per life-year gained. Topel and Murphy estimate that the average value of a life-year is approximately $150,000. This figure implies that the benefit-cost ratio of general medical expenditure is 13.6, and that the ratio for pharmaceutical R&D exceeds 100.

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U.S. Bureau of Labor Statistics, http://www.bls.gov/cpihome.htm

Figure 1
Life expectancy at birth, 1960-1997: trend and fluctuations

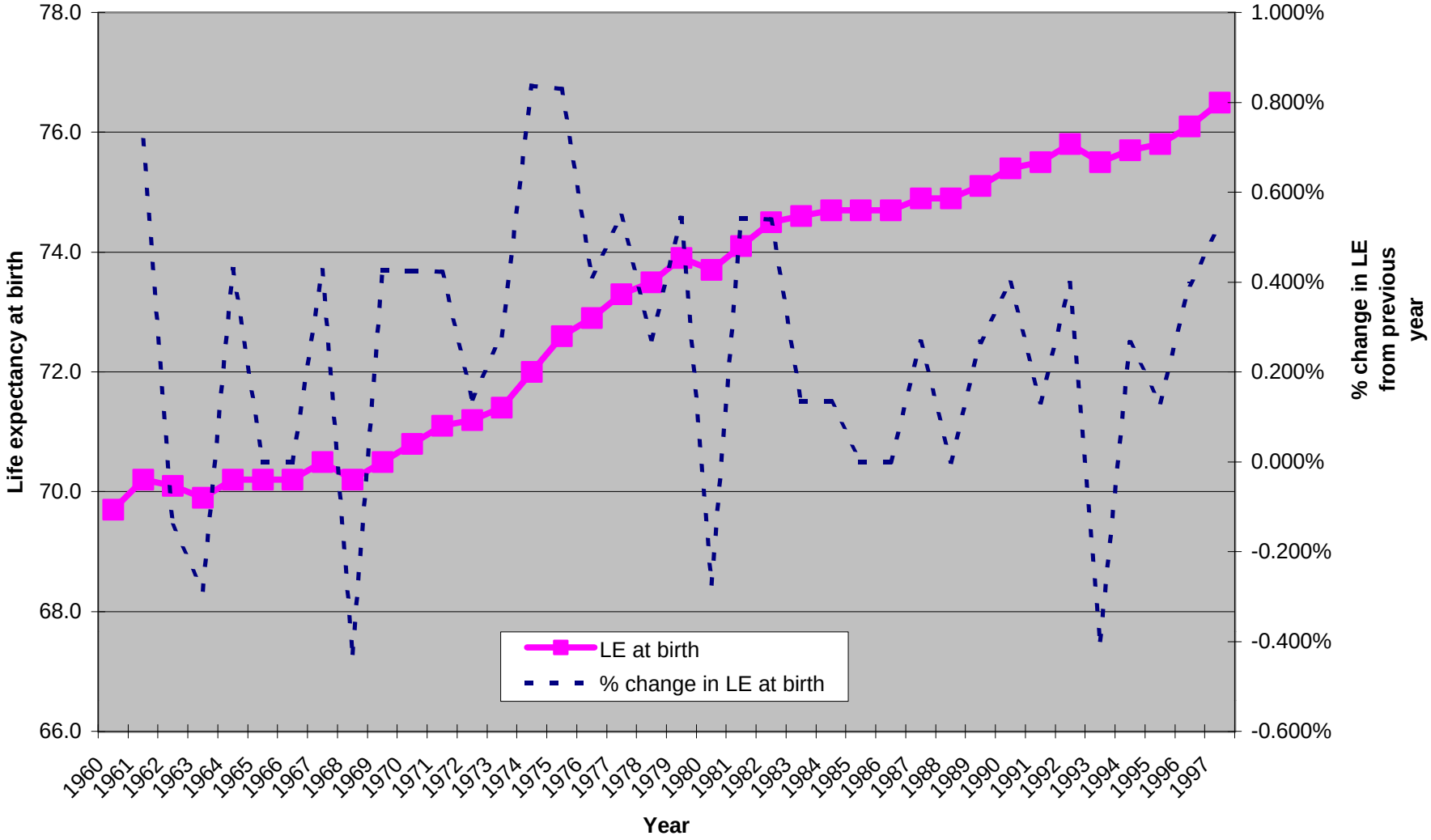


Figure 2
Annual growth rates of life expectancy at birth and real GDP per capita, 1960-1997

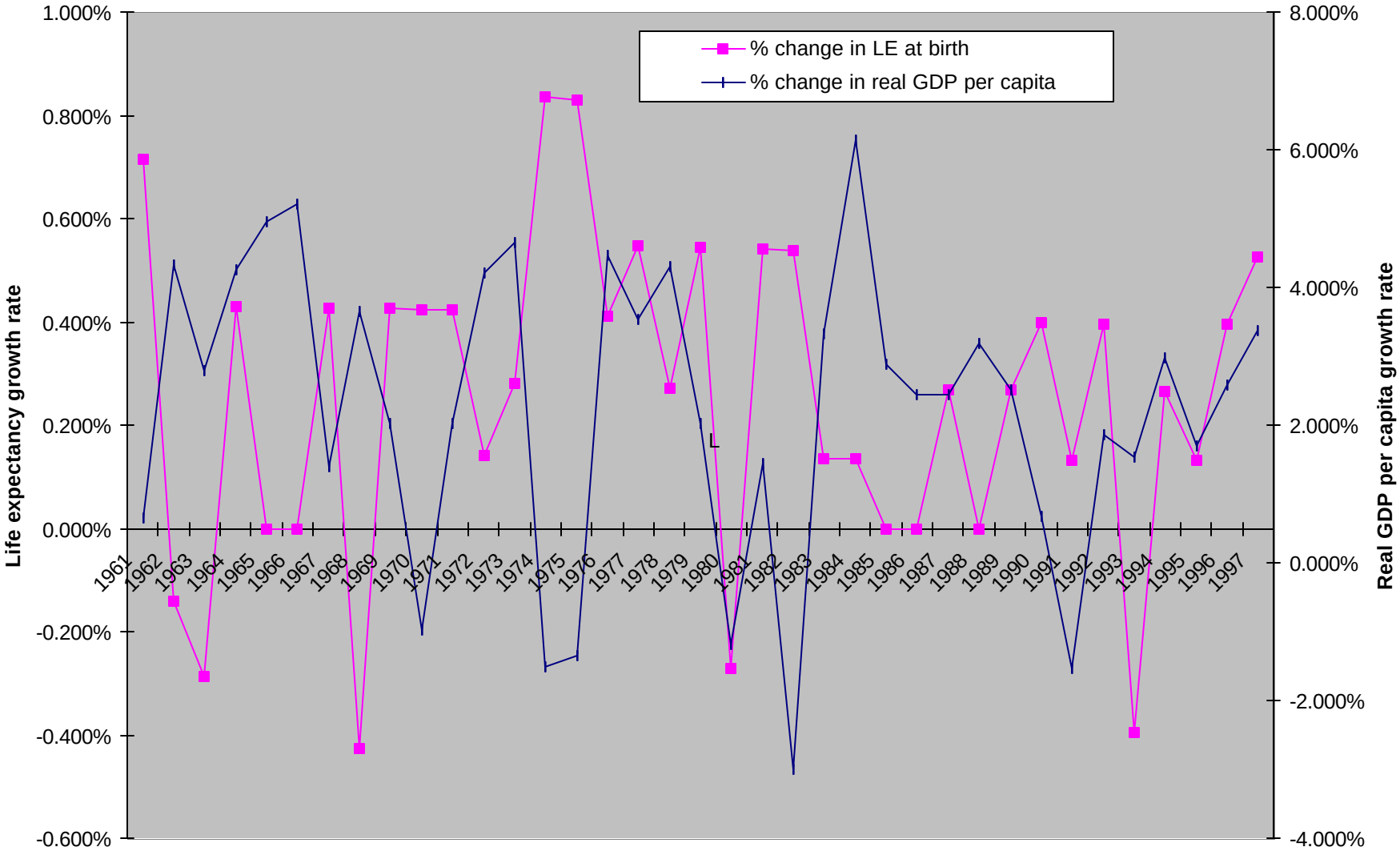


Figure 3
Life expectancy at birth and at age 40

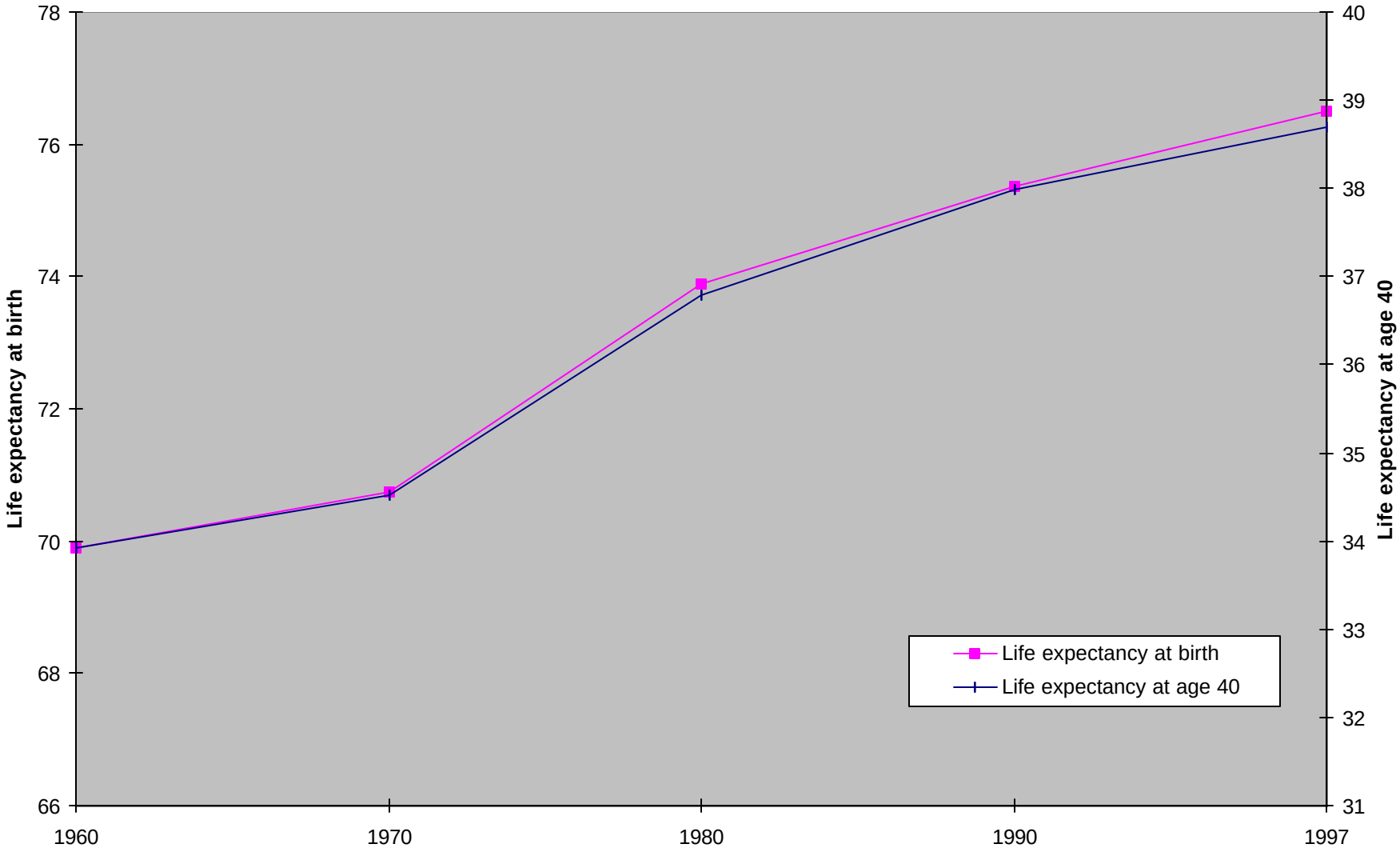


Figure 4
Life expectancy of blacks at birth as a % of life expectancy of whites at birth

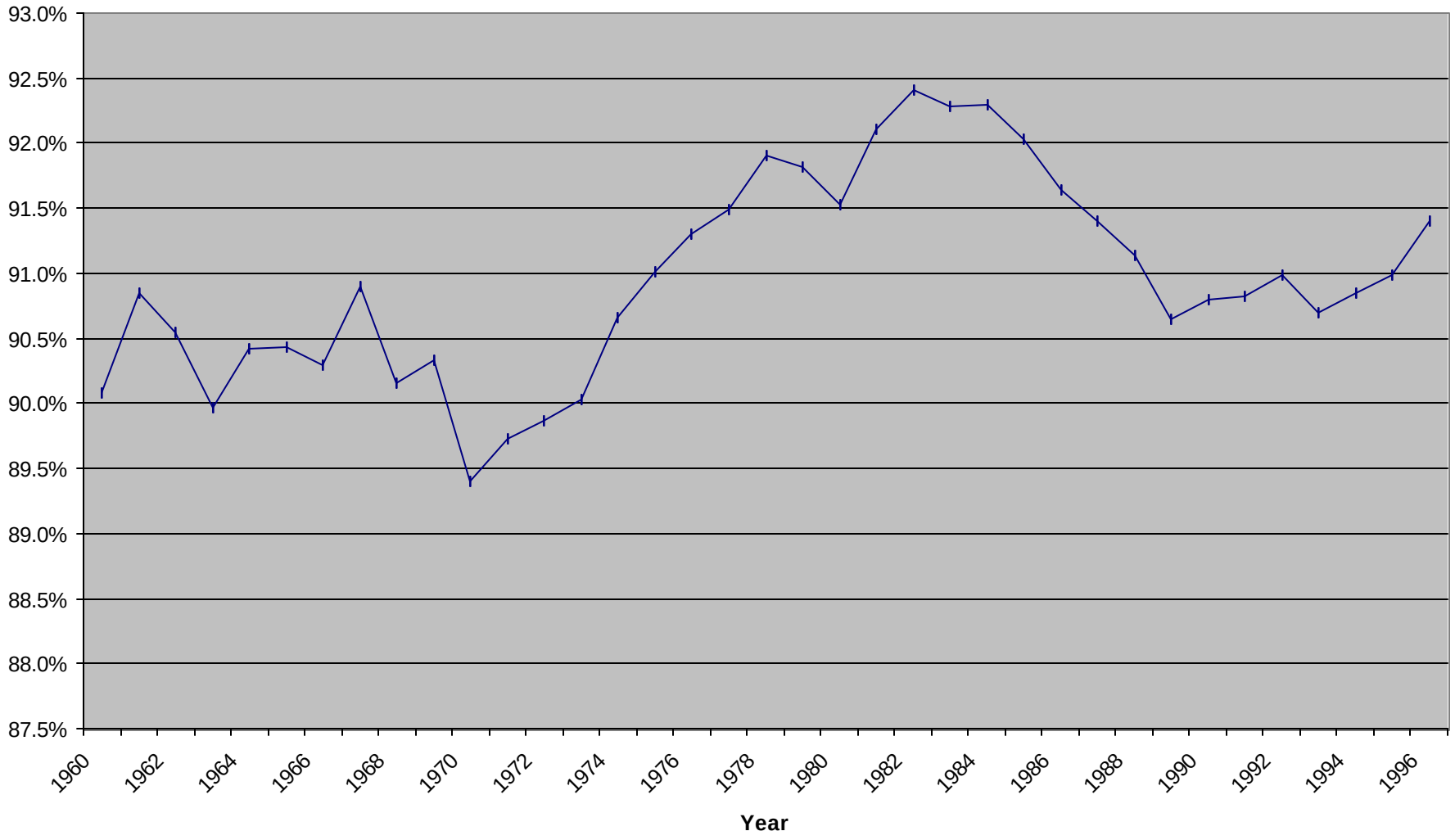


Figure 5
Real public and private health expenditure per capita

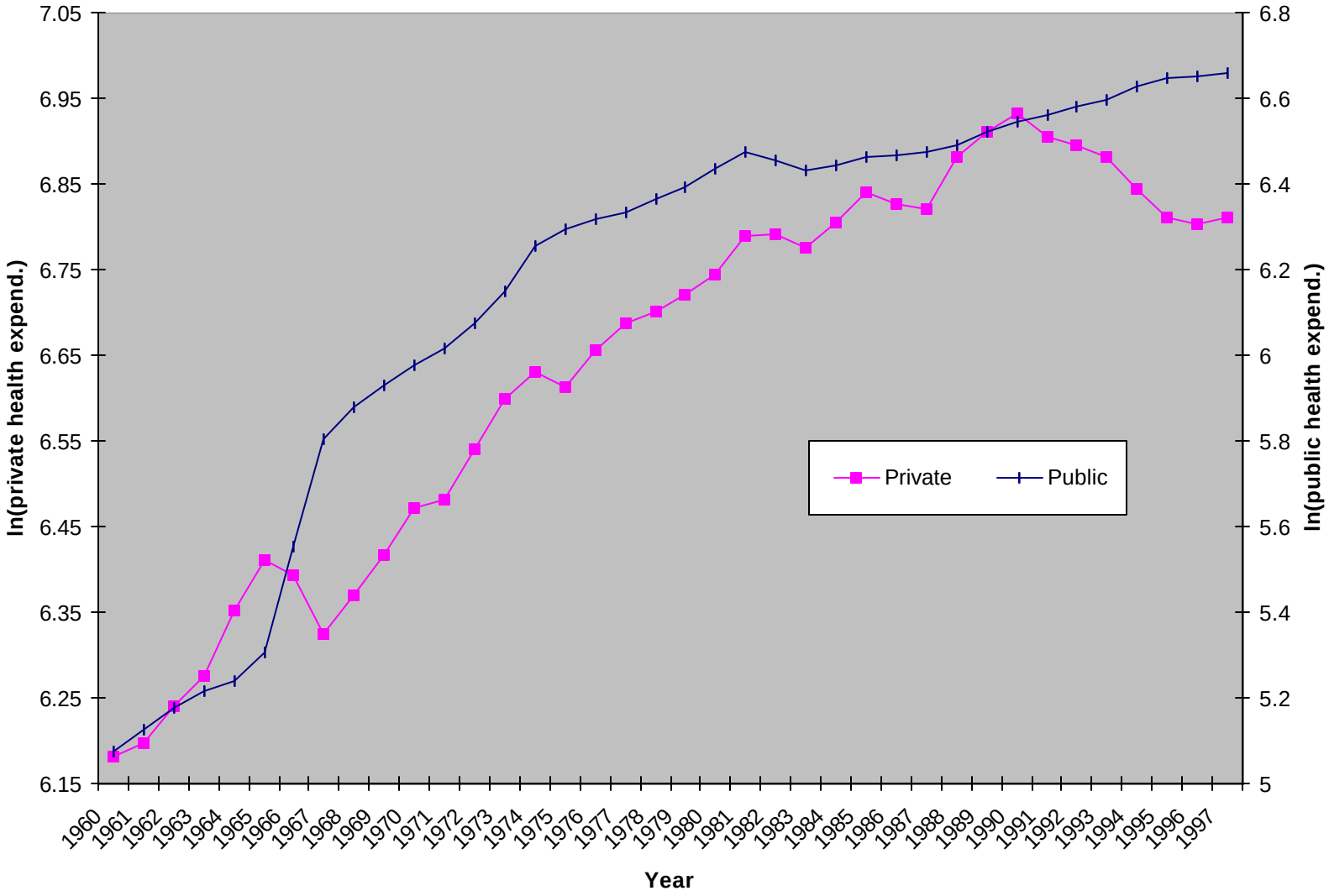


Figure 6
Mean medical expenditure in 1977, by age

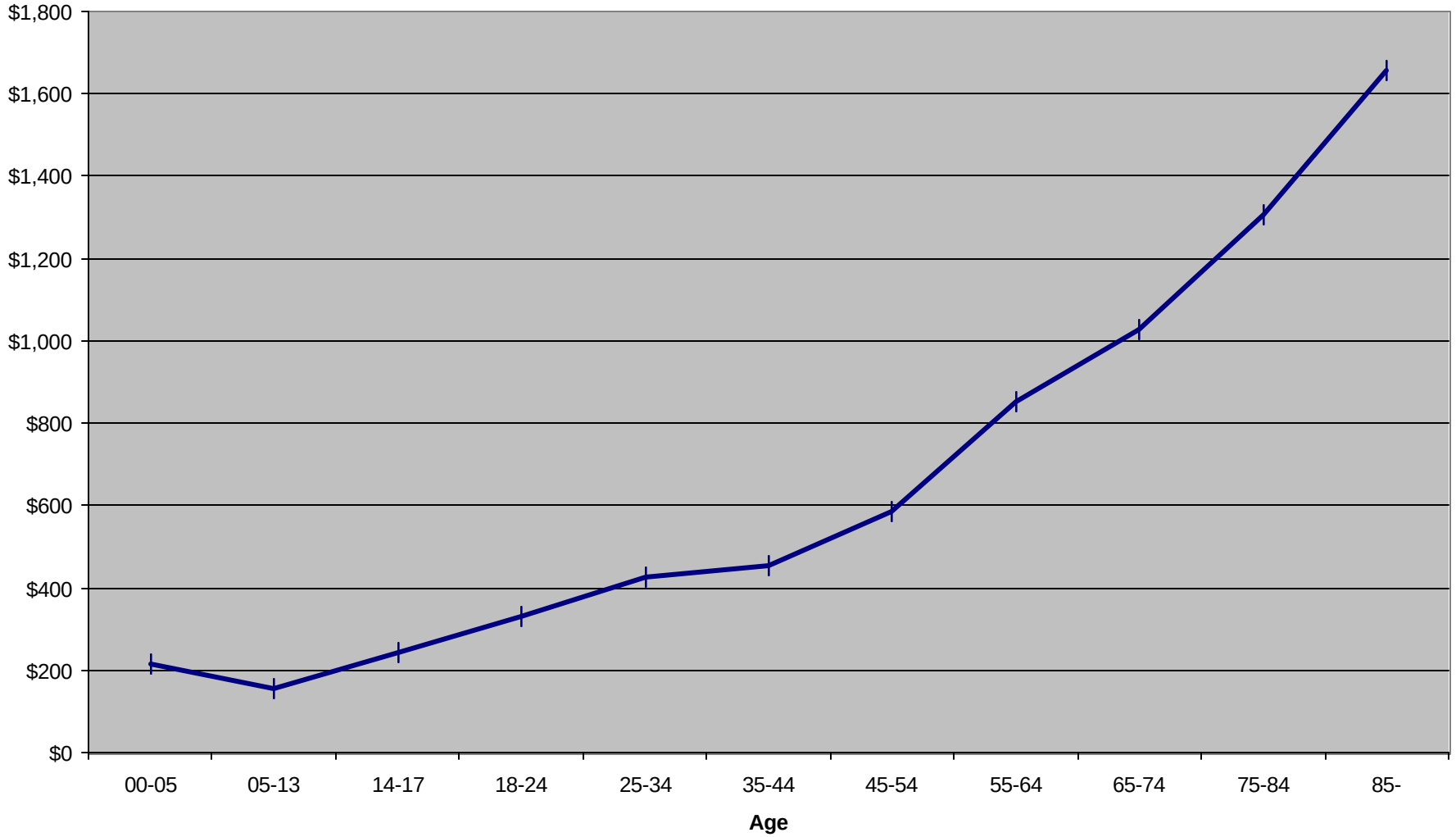


Figure 7
Number of new molecular entities approved by the FDA, 1960-97

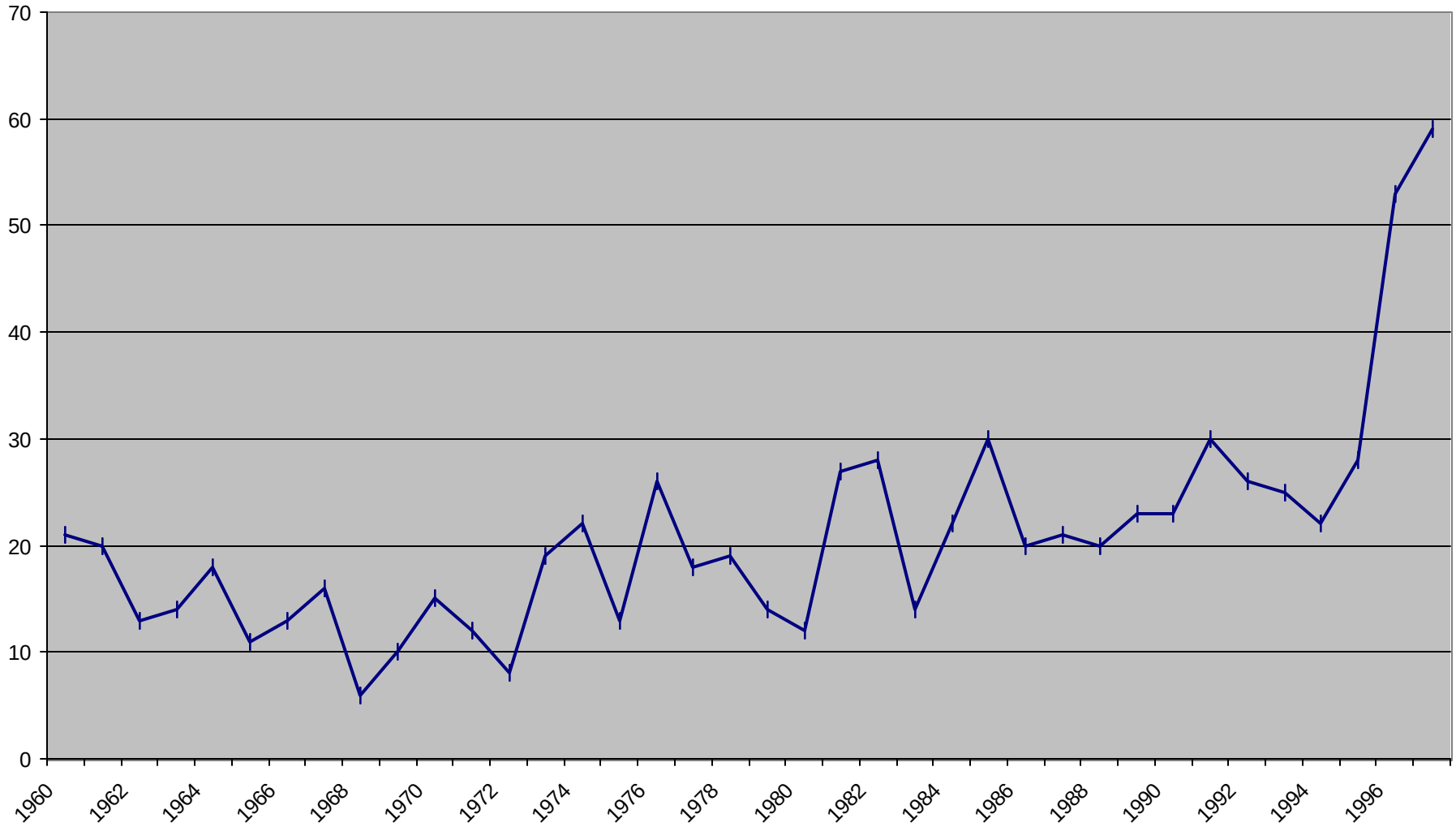
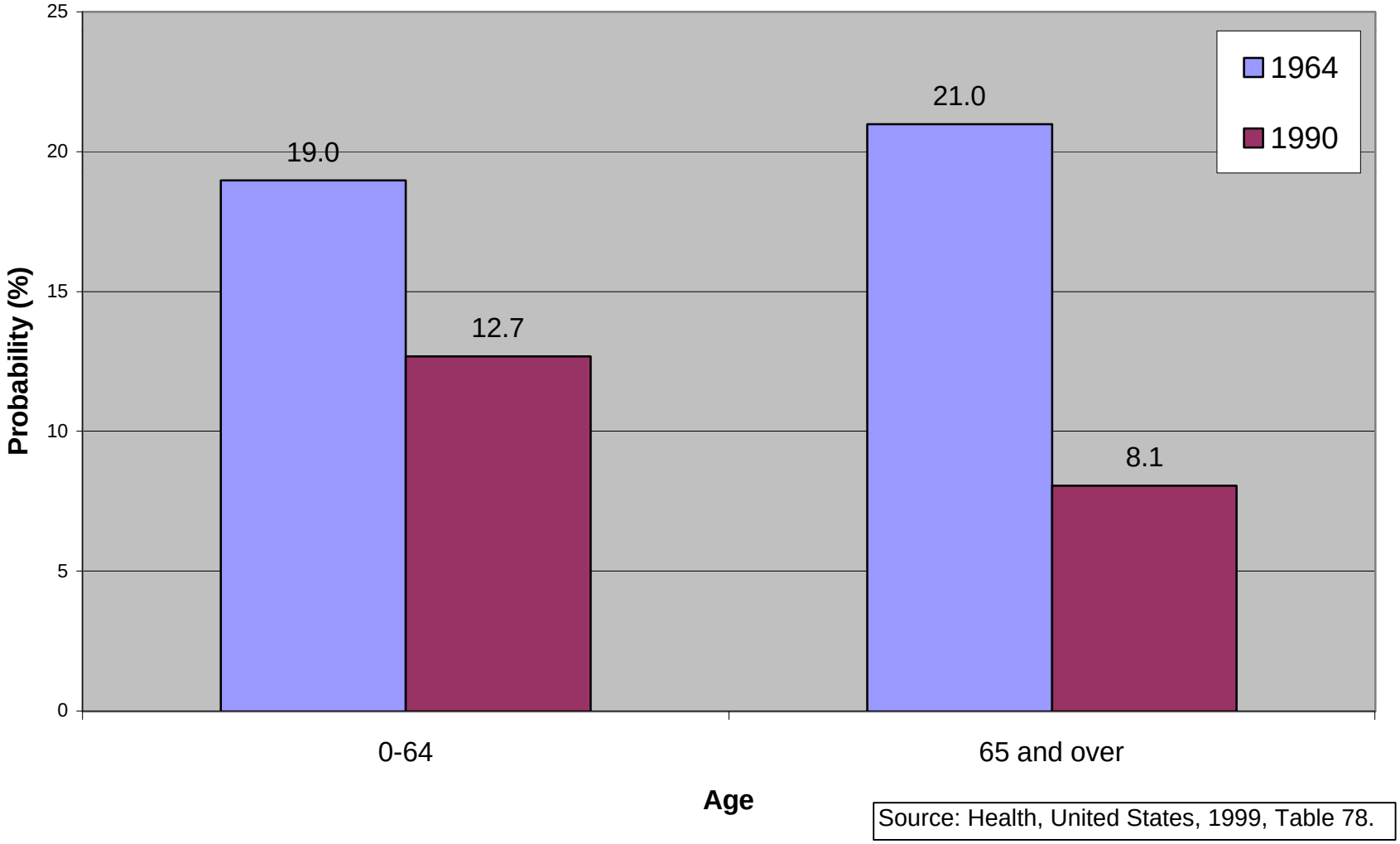


Figure 8
Probability of no physician contact within last 2 years, by age and year



Source: Health, United States, 1999, Table 78.

Figure 9
Percentage of drugs no longer marketed in 1999, by FDA approval year

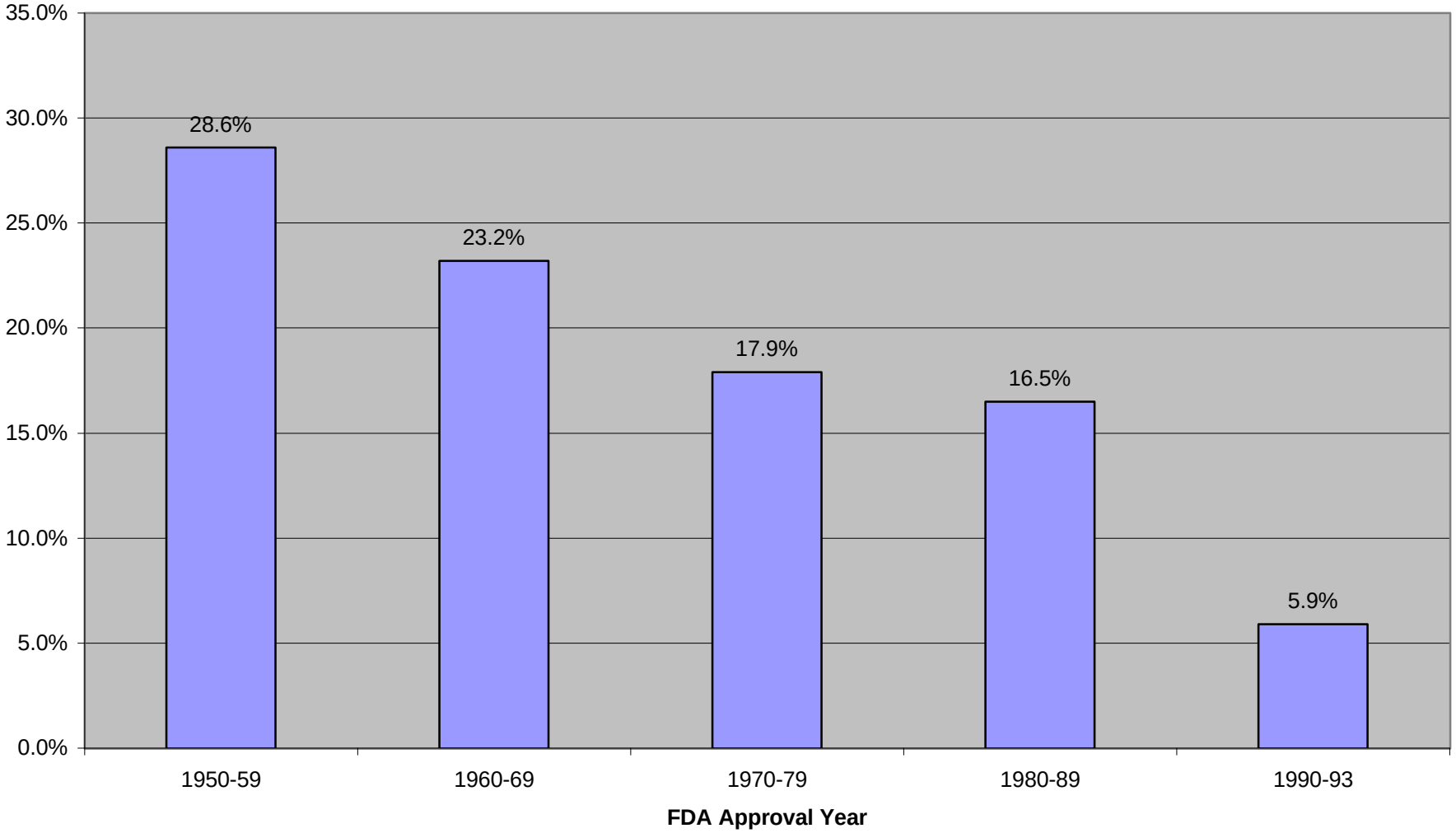


Table 1
Summary statistics for 1960-1997 sample period

	mean	standard deviation	minimum	maximum	first-order serial correl. coeff.
	Levels				
Life expectancy at birth (years)	73.1	2.247534	69.7	76.5	0.995
Number of new molecular entities approved	20.8	10.45675	6	59	0.561
Real health expenditure per capita	1305.9	361.2648	643.8	1721.5	0.998
Real public health expenditure per capita	521.9	198.1603	159.8	778.1	0.995
Real private health expenditure per capita	784.0	167.293	484	1024.3	0.990
Real GDP per capita	21286.1	4959.36	13155	30470	0.996
	Growth rates				
Life expectancy at birth (years)	0.003	0.003	-0.004	0.008	-0.018
Number of new molecular entities approved	0.028	0.413	-0.981	0.865	-0.307
Real health expenditure per capita	0.026	0.025	-0.019	0.064	0.595
Real public health expenditure per capita	0.043	0.056	-0.022	0.253	0.643
Real private health expenditure per capita	0.017	0.033	-0.068	0.077	0.333
Real GDP per capita	0.023	0.021	-0.030	0.061	0.193

Table 2
Maximum likelihood estimates of longevity equations
(t-statistics in parentheses)

Column	1	2	3	4	5	6	7	8
race	both	both	both	both	both	both	white	black
le_{-1}	0.7886	0.7612	0.7748	0.8162	0.7614	0.7611	0.794	0.8219
	(23.36)	(21.86)	(13.06)	(15.45)	(11.73)	(11.74)	(14.13)	(13.77)
nme_{+1}						0.001262		
						(1.02)		
nme	0.005673	0.00581	0.005962	0.00623	0.005435	0.005104	0.005411	0.0137
	(5.18)	(5.67)	(5.43)	(5.75)	(5.05)	(4.11)	(5.48)	(5.55)
nme_{-1}					0.003043	0.003721		
					(2.45)	(2.56)		
exp	0.016							
	(5.47)							
exp_{-1}		0.0176	0.0204					
		(6.25)	(4.62)					
$public_{-1}$				0.006855	0.007901	0.007792	0.005184	0.0114
				(3.93)	(4.33)	(4.19)	(3.25)	(2.82)
$private_{-1}$				0.008674	0.01	0.009707	0.0108	0.0121
				(1.48)	(1.62)	(1.34)	(2.02)	(0.95)
gdp_{-1}			-0.007233		0.000754	0.007399		
			(0.51)		(0.05)	(0.48)		
year			0.0000324	-0.000141	-0.000127	-0.000303	-0.000047	-0.000572
			(0.09)	(1.09)	(0.37)	(0.81)	(0.36)	(2.44)
first-order serial correl. coeff.	0.3555	0.3831	0.4177	0.4253	0.44	0.4792	0.456	0.3486
	(2.09)	(2.27)	(2.43)	(2.48)	(2.54)	(2.73)	(2.68)	(1.91)
DW	1.89	1.86	1.92	1.97	1.97	2.02	1.97	1.95
long-run elasticities:								
nme	0.0268354	0.02433	0.0264742	0.0338955	0.0355323		0.026267	0.0769231
total	0.0756859	0.0737018	0.0905861					
private				0.0471926				0.0679394
public				0.037296				0.064009

Figure 10
Comparison of relative (public/private) longevity coefficients to relative expenditure, by race

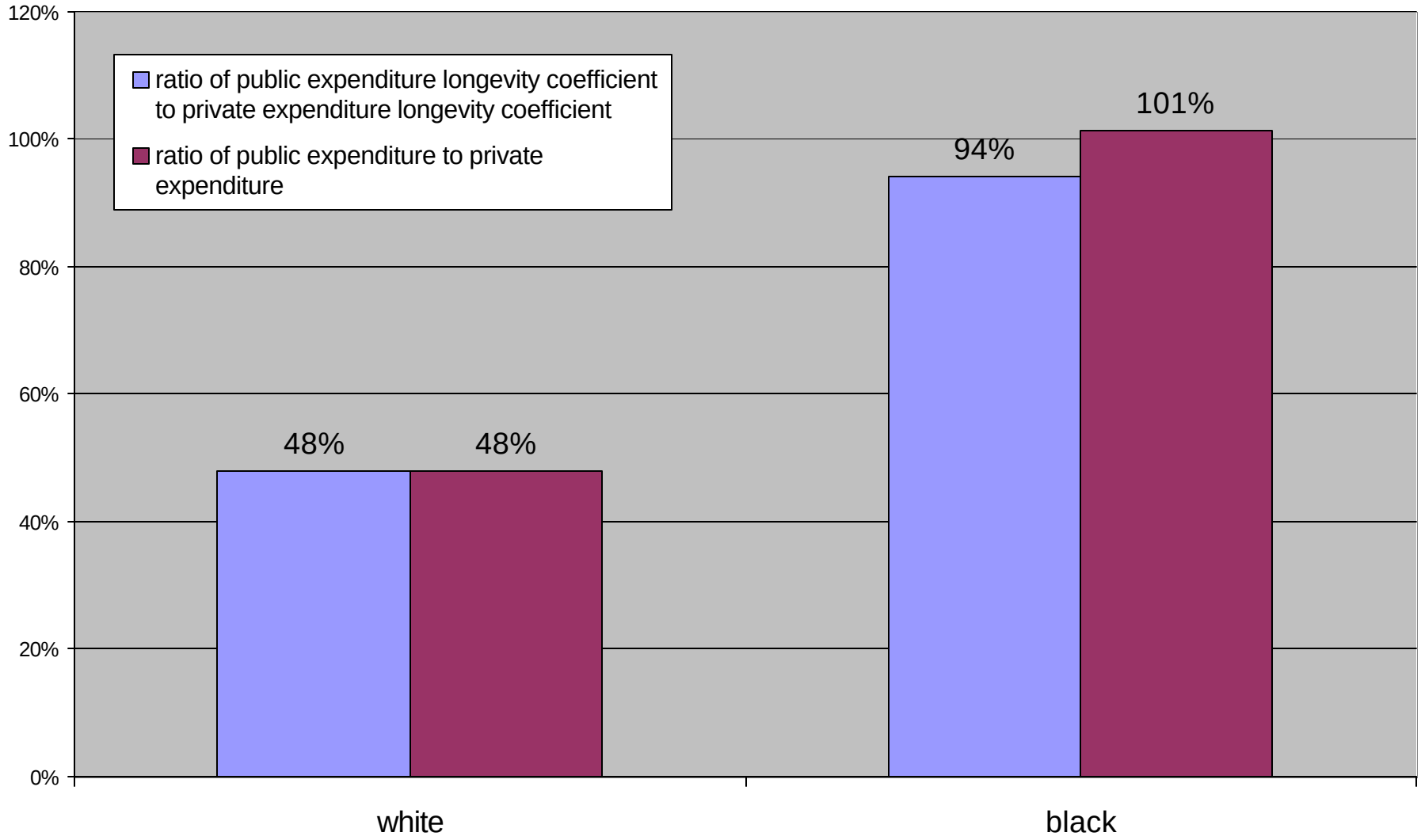


Table 3
Calculation of medical care expenditure and drug R&D cost per life-year gained

			Exogenous variable	
Line			per capita health expenditure	number of new molecular entities
1	long-run longevity elasticity		0.0906	0.0265
2	mean(longevity)		73.1	73.1
3	mean(exogenous variable)		1305.91	20.82
4	marginal long-run effect	= (1) * (2) / (3)	0.0051	0.0929
5	average number of births per year		4,000,000	4,000,000
6	no. of life-years gained per year from permanent unit increase in exog. var.	= (4) * (5)	20,274	371,724
7	annual cost of permanent unit increase in exog. var.		$224,090,962	$500,000,000
8	Cost per life-year gained	= (7) / (6)	$11,053	$1,345

Appendix Table 1
Basic data

Year	Life expectancy at birth (years)	Number of new molecular entities approved	Real health expend. per capita	Real public health expend. per capita	Real private health expend. per capita	Real GDP per capita
1960	69.7	21	643.8	159.8	484.0	13,155
1961	70.2	20	659.3	168.1	491.2	13,240
1962	70.1	13	689.7	176.7	512.9	13,825
1963	69.9	14	715.5	184.1	531.4	14,217
1964	70.2	18	762.3	188.5	573.8	14,834
1965	70.2	11	810.5	201.6	608.9	15,586
1966	70.2	13	855.5	257.8	597.7	16,420
1967	70.5	16	890.5	332.1	558.4	16,649
1968	70.2	6	941.6	357.4	584.2	17,270
1969	70.5	10	987.1	375.4	611.7	17,621
1970	70.8	15	1039.6	393.3	646.3	17,449
1971	71.1	12	1062.5	409.1	653.4	17,806
1972	71.2	8	1127.7	434.8	692.9	18,573
1973	71.4	19	1202.6	468.4	734.2	19,458
1974	72.0	22	1279.3	521.2	758.1	19,167
1975	72.6	13	1284.8	540.8	743.9	18,912
1976	72.9	26	1332.0	555.3	776.7	19,775
1977	73.3	18	1364.5	562.3	802.2	20,486
1978	73.5	19	1394.6	581.8	812.8	21,388
1979	73.9	14	1425.9	597.2	828.7	21,826
1980	73.7	12	1473.4	624.6	848.7	21,569
1981	74.1	27	1536.9	648.9	888.0	21,881
1982	74.5	28	1526.1	636.1	890.0	21,235
1983	74.6	14	1497.4	622.1	875.4	21,952
1984	74.7	22	1531.7	628.8	902.9	23,344
1985	74.7	30	1574.4	639.7	934.7	24,029
1986	74.7	20	1565.3	644.1	921.2	24,621
1987	74.9	21	1565.5	648.3	917.2	25,231
1988	74.9	20	1634.5	659.7	974.7	26,047
1989	75.1	23	1681.9	679.9	1002.1	26,707
1990	75.4	23	1721.5	697.2	1024.3	26,889
1991	75.5	30	1704.4	706.8	997.7	26,478
1992	75.8	26	1707.3	720.3	987.0	26,977
1993	75.5	25	1704.5	730.9	973.6	27,398
1994	75.7	22	1694.6	756.5	938.0	28,225
1995	75.8	28	1677.1	769.8	907.3	28,705
1996	76.1	53	1673.0	773.3	899.7	29,458
1997	76.5	59	1685.9	778.1	907.8	30,470