

# The Allocation of Public Funds for Biomedical R&D



# The Allocation of Public Funds for Biomedical R&D

---

Frank R. Lichtenberg

The AEI Press

*Publisher for the American Enterprise Institute*

WASHINGTON, D.C.

*1999*

*The American Enterprise Institute would like to thank the Burroughs Wellcome Fund for its support.*

*The author is grateful to the American Enterprise Institute and to the Center for Economic Studies at the University of Munich for financial support.*

Available in the United States from the AEI Press, c/o Publisher Resources Inc., 1224 Heil Quaker Blvd., P.O. Box 7001, La Vergne, TN 37086-7001. To order, call toll free 1-800-269-6267. Distributed outside the United States by arrangement with Eurospan, 3 Henrietta Street, London WC2E 8LU England.

ISBN 0-8447-7130-9

1 3 5 7 9 10 8 6 4 2

© 1999 by the American Enterprise Institute for Public Policy Research, Washington, D.C. All rights reserved. No part of this publication may be used or reproduced in any manner whatsoever without permission in writing from the American Enterprise Institute except in the case of brief quotations embodied in news articles, critical articles, or reviews. The views expressed in the publications of the American Enterprise Institute are those of the authors and do not necessarily reflect the views of the staff, advisory panels, officers, or trustees of AEI.

THE AEI PRESS

Publisher for the American Enterprise Institute  
1150 17th Street, N.W., Washington, D.C. 20036

*Printed in the United States of America*

# Contents

---

INTRODUCTION	1
<hr/>	
SIMPLE MODEL OF THE DETERMINANTS OF RESEARCH EXPENDITURE AT THE DISEASE LEVEL	5
<hr/>	
DATA SOURCES AND METHODS	9
<hr/>	
PRELIMINARY ESTIMATES	17
<hr/>	
SUMMARY	31
<hr/>	
NOTES	33
<hr/>	
REFERENCES	37
<hr/>	
ABOUT THE AUTHOR	39

---



## Introduction

The explanation of long-run growth in the value of per capita output is a central issue in economics. In recent years economists have increasingly recognized that traditional, official measures of growth in real per capita output significantly understate true economic growth. One reason is that the price indexes that are used to deflate nominal output tend to overestimate inflation by approximately 1–1.5 percentage points per year (Advisory Commission to Study the Consumer Price Index 1995).

A second reason is that the gross domestic product does not fully account for several important, highly valued “commodities” such as leisure, health, and longevity. The average person born in 1995 expects to live 22 years (41 percent) longer than the average person born in 1920. Although the rate of increase of longevity appears to be declining, between 1970 and 1991 mean age at death still increased 5.4 years. Nordhaus (1998) points out that economic growth is substantially underestimated because increased longevity is not taken into account: he estimates that “to a first approximation, the economic value of increases in longevity over the twentieth century is about as large as the value of measured growth in non-health goods and services” (17). In other words economic growth adjusted for longevity increase is twice as large as unadjusted economic growth.

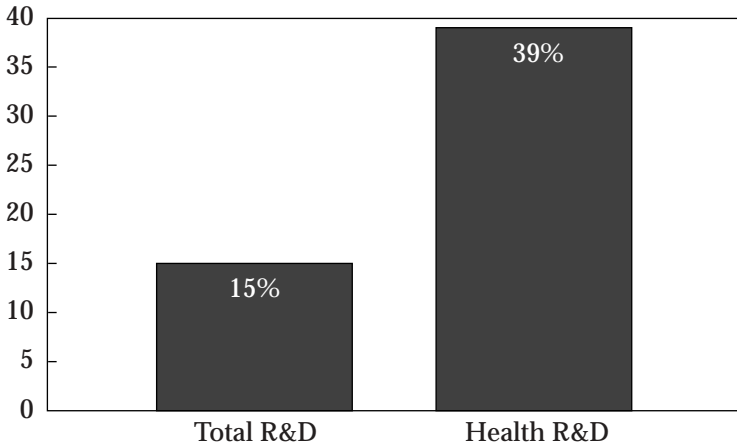


The Solow model,<sup>1</sup> which is perhaps the most widely accepted theory of economic growth, implies that technological progress is the fundamental source of growth in per capita income: “Once the economy is in a steady state, the rate of growth of output per worker depends only on the rate of technological progress” (Mankiw 1992, 102). While early models of growth treated the rate of technological progress as an exogenous variable, more recent (endogenous growth) models recognize that technological progress depends on investment in research and development. Abundant empirical evidence supports the hypothesis that the growth in conventionally defined per capita output (or total-factor productivity) is positively related to previous investments in research and development. Griliches and Lichtenberg (1984), for example, found that the most R&D-intensive manufacturing industries tend to have the highest rates of growth of output per worker.

In 1993, health R&D accounted for 18.8 percent of the total U.S. R&D expenditure (NCHS 1995, table 132; NSF 1996, table C-2). As figure 1 shows, the federal government plays a much larger role in health R&D than in other areas of civilian R&D (NCHS 1994, table 132; NSF 1996, table C-14).<sup>2</sup> The National Institutes of Health (NIH) funded 31.5 percent of all public and private health R&D in 1993 and 81 percent of federal health R&D.

The other (unmeasured) half of economic growth—the increase in longevity—is therefore quite likely, to an important extent, to be due to public and private investments in health R&D. Elsewhere (Lichtenberg 1998), I provide evidence that reductions in mortality are significantly directly related across diseases to the extent of pharmaceutical innovation, which results primarily from privately funded biomedical research. I show that the mean reduction in life-years lost for the nineteen diseases with the highest relative use of new drugs was more than five times as great as the mean reduction in life-years lost for the nineteen diseases with the lowest relative use

FIGURE 1  
FEDERAL GOVERNMENT SHARE OF NATIONAL R&D EXPENDITURE  
(in percent)



SOURCE: NCHS 1994, table 132; NSF 1996, table C-14.

of new drugs. On average, each new drug approved during the period 1970–1991 saved an estimated 11,200 life-years in 1991 (and presumably continued to do so). If, as previous researchers have argued, the value of a life-year is about \$25,000 and the average cost of a new drug approval is \$697 million, then the social rate of return to pharmaceutical innovation, to a first approximation, is about 40 percent.

Establishing the contribution of publicly funded biomedical research to health progress may be much more difficult. As some officials of the National Institutes of Health have argued, much NIH-sponsored research is basic in nature, and although “scientific advances would not have been possible without continuing insight and understanding regarding the fundamental mechanisms of life and disease . . . basic research linkages to health care advances

are complicated, long-term, and impossible to allocate clearly” (NIH 1993, 3). NIH has produced estimates of cost savings from thirty-four advances in health care resulting from NIH support for applied research and clinical trials. Most of these examples focus on a single innovation, such as a new vaccine, a new diagnostic test, or a particular therapy. But because these case studies are not necessarily a random sample of all NIH-sponsored research, they may not reveal the aggregate or average effect of this research on costs.<sup>3</sup> Mushkin (1979) attempted to determine econometrically the contribution of biomedical research to reductions in mortality and morbidity. But most of her analysis was in an aggregate time-series framework and was based on crude measures of biomedical research, such as the number of biomedical doctorates lagged ten years.

Although identifying the ultimate benefits of public biomedical research may be difficult, it is still possible and useful to develop a simple theory of the allocation of public biomedical research expenditure and to present some empirical evidence about the determinants of this allocation. Those are the objectives of this monograph. The implications of the theoretical model developed here are consistent with government officials’ descriptions of the allocation process: the structure of expenditure should depend on research productivity (or scientific opportunity) as well as on public health need, that is, the societal and economic burden of the disease or condition.<sup>4</sup> Although at this point there are no useful indicators of research productivity (that is, the cost of achieving research advances), there are several measures of disease burden (that is, the benefit of achieving these advances). Analysts of technical change typically have data on neither the costs nor the benefits of technical advance. Failure to measure research productivity will not necessarily bias my estimates; if it does, it seems likely to bias them toward zero.

The next section develops the simple model of the allocation of public research expenditure. Estimating the

parameters of the model relies on three types and sources of data: data on research activity derived from the NIH database for the Computerized Retrieval of Information on Scientific Projects (CRISP), premature mortality data from the Vital Statistics–Mortality Detail file, and data on the prevalence and severity of chronic conditions from the National Health Interview Survey. These are discussed next. Preliminary estimates are then presented; a concluding summary follows.

### **Simple Model of the Determinants of Research Expenditure at the Disease Level**

To motivate the discussion and develop a few intuitions, we consider the simplest possible model of research funding allocation. This model is based on the following strong assumptions (some are relaxed below): (1) there are only two diseases; (2) the number of people suffering from the two diseases,  $N_1$  and  $N_2$ , is exogenous; (3) the average severity of the two diseases is identical; (4) the probability  $P_i$  of finding a cure for disease  $i$  ( $i = 1, 2$ ) is a concave (deterministic) function of research funding for that disease,  $X_i$ :  $P_i = X_i^\alpha$  where  $0 < \alpha < 1$ ;<sup>5</sup> (5) the effect of funding on the probability of finding a cure is the same across diseases; and (6) the total research budget  $X = X_1 + X_2$  is fixed.

Suppose that policymakers attempt to maximize the (expected) total number of people cured of both diseases subject to the budget constraint;<sup>6</sup> that is, they choose  $X_1$  to maximize

$$\begin{aligned} J^* &= N_1 P_1 + N_2 P_2 = N_1 X_1^\alpha + N_2 X_2^\alpha \\ &= N_1 X_1^\alpha + N_2 (X - X_1)^\alpha. \end{aligned} \quad (1)$$

The first-order condition implies that relative funding of research on the two diseases should satisfy

$$\ln (X_1/X_2) = [1/(1 - \alpha)] \ln (N_1/N_2). \quad (2)$$

Research funding should increase with disease incidence: for example,  $X_1 > X_2$  if  $N_1 > N_2$ . This happens because the benefit of discovering a cure for the disease is proportional to its incidence but the cost is independent of incidence. Moreover, the elasticity of funding with respect to incidence should exceed unity: if disease 1 is twice as prevalent as disease 2, research funding for disease 1 should be more than twice as great as research funding for disease 2.

One could generalize this model to the case of  $I > 2$  diseases, to obtain  $I - 1$  equilibrium conditions of the form

$$\ln X_i = \text{constant} + [1/(1 - \alpha)] \ln N_i \quad (3)$$

where ( $i = 1, 2, \dots, I - 1$ ). Given cross-sectional or panel data on research funding and incidence by disease, one could estimate equation 3 to test the hypothesis of diminishing returns to research funding at the disease level and to estimate the parameter  $\alpha$ . But this simple model can and should be extended in at least two directions: we should allow for multiple indicators of incidence and for differences in research productivity (scientific opportunity) across diseases.

**Multiple Indicators of Incidence.** As the director of NIH says, a given disease imposes different kinds of burdens on society, and “policy makers will need to consider the relative importance or weight to be placed on each criteri[on] when assessing the overall societal burden imposed by each disease” (Varmus 1995). Suppose that policymakers perceive the overall burden of a disease as a function of  $K$  attributes of the disease:  $N_i = f(A1_i, A2_i, \dots, AK_i)$  where, for example,  $A1$  is the number of deaths,  $A2$  is the number of bed-disability days,  $A3$  is the number of hospital stays, and so forth. Further suppose that the functional form of this relationship is

$$\ln N_i = \beta_1 \ln A1_i + \beta_2 \ln A2_i + \dots + \beta_K \ln AK_i \quad (4)$$

where  $\sum_k \beta_k = 1$ .  $\beta_k$  reveals the relative weight assigned by policymakers to attribute  $k$  in the determination of overall disease burden. Substituting equation 4 into equation 3,

$$\ln X_i = \text{constant} + [1/(1 - \alpha)] \{ \beta_1 \ln A1_i + \beta_2 \ln A2_i + \dots + \beta_K \ln AK_i \}. \quad (5)$$

Estimation of equation 5 would provide estimates of these (revealed preference) weights as well as of the technological parameter  $\alpha$ . They would indicate, for example, the relative weight given to mortality and bed-disability days.

Since disease outcome and incidence data are available by demographic group, we can also make inferences about weights associated with different demographic groups.<sup>7</sup> Let us define, for example, “adjusted” bed-disability days  $A2^* = A2\text{YOUNG} + (1 + \theta)A2\text{OLD}$ , where  $A2\text{YOUNG}$  and  $A2\text{OLD}$  denote bed-disability days of young and old people, respectively. If policymakers’ evaluation of the marginal burden of the two groups’ bed-disability days differs,  $\theta$  will differ from zero. This parameter can be estimated by replacing  $A2$  by  $A2^*$  in equation 5.

**Differences in Research Productivity (Scientific Opportunity) across Diseases.** The preceding model is based on the assumption that the effect of funding on the probability of finding a cure is the same across diseases. This assumption is clearly unrealistic, and it is desirable to relax it.<sup>8</sup> We can modify the cure-probability equation to include a disease-specific research productivity parameter  $\pi_i$ :  $P_i = \pi_i X_i^\alpha$ . The objective function policymakers seek to maximize is now  $J^* = N_1 P_1 + N_2 P_2 = N_1 \pi_1 X_1^\alpha + N_2 \pi_2 X_2^\alpha$ , and the optimal expenditure on research on disease  $i$  is now

$$\ln X_i = \text{constant} + [1/(1 - \alpha)] \ln N_i + [1/(1 - \alpha)] \ln \pi_i. \quad (6)$$



The research-productivity parameters  $i$  enter the objective function and the optimal expenditure equation in the same way as the disease incidence measures  $N_i$ . Research expenditure should be an increasing function of scientific opportunity as well as of disease burden. This implication is consistent with the views expressed by government officials: "It is vital that the allocation of medical research dollars takes into account several factors, including scientific opportunity, public health need, gaps in knowledge, as well as societal and economic burden of the disease/condition."<sup>9</sup>

The CRISP data can eventually be exploited to obtain indicators of (changes in) the relative productivity of research on different diseases. The data will enable us to determine, for example, the extent to which research related to a given disease tends to be concentrated in rapidly growing and advancing scientific fields (for example, molecular genetics) as compared with mature fields. The data will also allow us to quantify the extent to which research on a disease uses innovative research techniques (for example, protein engineering) and how much the distribution of techniques has changed over time.

At present, however, we must treat  $\pi_i$  as unobservable. If research productivity is uncorrelated across diseases with disease burden, that is, if differences in supply (or cost of achieving progress) are uncorrelated with differences in demand (or benefits of achieving progress), estimation of equation 5 will yield an unbiased estimate of the relationship between research expenditure and disease burden.  $N$  and  $\pi$ , however, may be negatively correlated: the diseases that impose the heaviest burden do so, in part, because of the low productivity of past research on those diseases (which should also have resulted in relatively low research funding for them). If this is the case, then the omission of  $\pi_i$  from the research expenditure equation would bias the estimated coefficient for  $\ln N_i$  toward zero. In particular, although the theory implies that the coefficient for  $\ln N_i$

should be greater than one, we should not be surprised if we obtain estimated coefficients smaller than one, that is, if we fail to observe increasing returns of this kind.

Future research may directly estimate the contribution of public biomedical research expenditure to subsequent progress against disease by analyzing the correlation across diseases between research investment and indicators of progress, such as reductions in potential life-years lost. Heterogeneous, unobserved research productivity, however, is likely to lead to overestimates of the average return to research expenditure. Diseases with the greatest research funding are presumably those with the highest research productivity. The slope of the relationship across diseases between research funding and progress exceeds the mean of the slopes of the disease-specific relationships.<sup>10</sup>

### **Data Sources and Methods**

**Data on Government Research Funding by Disease.** This study has calculated distributions of government-funded biomedical research expenditure by disease from records of research grants contained in NIH's CRISP system. This database includes records of all research ventures supported by the Public Health Service since 1972. In fiscal year 1995 there were records of 63,289 grants, with a total value of \$10.1 billion. Most research falls within the broad category of extramural projects: grants, contracts, and cooperative agreements conducted primarily by investigators at universities, hospitals, and other research institutions. The projects were funded by NIH and the Substance Abuse and Mental Health Services Administration. A few of these research grants were funded by the Centers for Disease Control, the Food and Drug Administration, the Health Resources and Services Administration, and the Agency for Health Care Policy and Research. CRISP also contains information on intramural research programs conducted by

scientists employed by the FDA and the various components of NIH.

Each record reports the name of the investigator, the name and address of the organization (for example, university and department), the title (and in many cases an abstract) of the project, the administering organization (for example, the National Cancer Institute), the award amount (including both direct and indirect costs), the type of award, and a number of indexing terms (generally about fifteen) assigned by technical information specialists in the Research Documentation Section, Information Systems Branch, of the Division of Research Grants at NIH. The indexing process is governed by the CRISP Thesaurus, which is the “controlled vocabulary used to assign indexing terms for the CRISP System, and to retrieve subject-related information from it.”

The number of distinct indexing terms in the CRISP Thesaurus is quite large (about 9,000), but most of these terms are organized into a small number of hierarchical classification schemes, including one for diseases. Table 1 illustrates the disease classification; it is similar to the International Classification of Diseases, the system used for reporting diagnoses in most health-related data. There are thirty-five disease categories at the highest level of aggregation. Within each of these is a series of more specific disease categories. Space limitations prevent a display of the entire tree structure of diseases (which includes about 2,900 items), but the second-level classification of nervous disorders and a branch leading to a fifth-level disease (with no further subcategories), lymphocytic choriomeningitis, illustrate the classification system.

This disease classification scheme enables us to compute distributions of research grants and dollars by disease at various levels of aggregation<sup>11</sup> (see figure 2). How accurate are these distributions? Recently the Office of the Director of NIH reported estimates of NIH FY1994 research support by disease. These figures, based on data provided

TABLE 1  
CLASSIFICATION SYSTEMS FOR DISEASES  
USED IN CRISP DATABASE

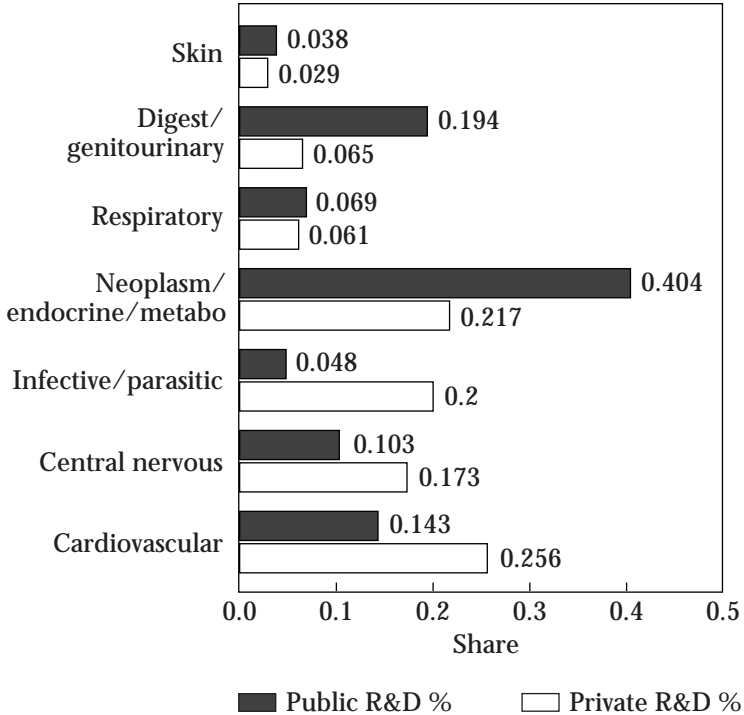
---

Blood disorder 04273600
Calcium disorder 05316510
Cardiovascular disorder 05710209
Communicable disease 07156766
Communication disorder 15792995
Congenital disorder 07231051
Connective tissue disorder 07297208
Digestive disorder 40000163
Ear disorder 09775187
Endocrine disorder 10255693
Enzyme deficiency 40010049
Eye disorder 11148096
Genetic disorder 12547727
Hernia 09445779
Immunopathology 15604280
Infection 40000216
Injury 15823104
Lymphatic disorder 04277757
Mental disorder 24836609
Metabolism disorder 18462030
Musculoskeletal disorder 40000257
Neoplasm/cancer 20000173
Nervous disorder 20422001
autonomic disorder 20423808
central nervous system disorder 20424612
brain disorder 04850499
cataplexy 20573270
central nervous system neoplasm 20125421
degenerative motor system disease 20573603
encephalomyelitis 20424989
gliosis 20422145
hemiplegia 20573642
meningitis 20425301
infectious meningitis
bacterial meningitis 20425411
viral meningitis 20425450
lymphocytic choriomeningitis 20425332
[other disorders]

---

SOURCE: CRISP Thesaurus computer file provided to the author by NIH.

**FIGURE 2**  
**SHARES OF PUBLIC AND PRIVATE HEALTH R&D ALLOCATED TO MAJOR DISEASE CATEGORIES IN 1982**



SOURCE: Public R&D-NIH FY1982 CRISP file. Private R&D-Unpublished data based on Pharmaceutical Manufacturers and Research Association Annual Survey (see <http://www.phrma.org>).

by NIH institutes, centers, and divisions (ICDs), “reflect NIH-wide resources devoted to research on the listed diseases . . . [and] generally do not correspond to budget figures for the ICD identifying the cost data” (NIH 1995). For sixteen randomly selected diseases, this study compared FY1994 funding as reported there with the number of

TABLE 2  
COMPARISON OF FY1994 FUNDING AND FY1995 GRANTS,  
FOR SIXTEEN DISEASES

<i>Disease or Disorder</i>	<i>FY1995 Grants</i>	<i>FY1994 Funds</i>
Arthritis	476	191
Asthma	345	66
Atherosclerosis	650	116
Diabetes	1,390	292
Epilepsy	338	52
MS	123	78
Obesity	474	83
Osteoporosis	288	92
Parkinson's	253	68
Pneumonia and influenza	230	60
Psoriasis	53	3
Schizophrenia	458	111
Sickle cell anemia	278	54
Suicide	94	17
Tuberculosis	248	50

SOURCE: NIH 1995 and author's calculations based on FY1995 CRISP file.

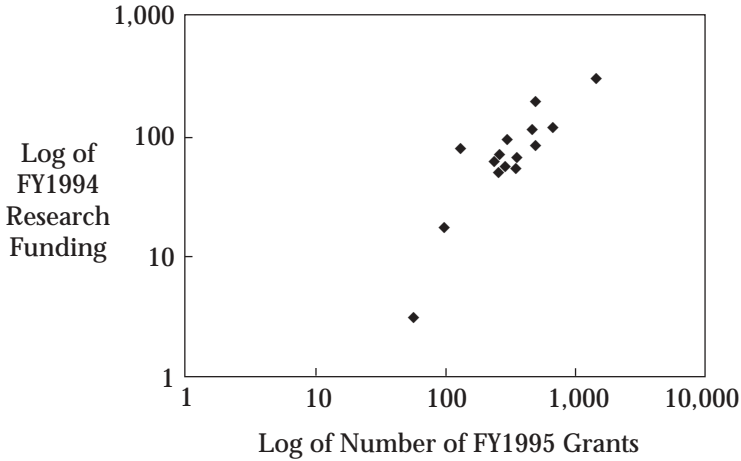
FY1995 grants citing the disease contained in the FY1995 CRISP database. The raw data are reported in table 2.

A scatter plot of the logarithms of these two variables is shown in figure 3; their correlation coefficient is 0.91. Despite differences in timing and the unit of measurement, the two estimates of relative research support by disease are quite similar; this similarity suggests that the CRISP data provide accurate statistics.

As noted, NIH officials have observed the basic nature of much of the agency's research. Linking research exactly to specific advances in health is complex (NIH 1993).

Many research grants do not refer to any disease (even though the research may ultimately lead to breakthroughs in the treatment of that disease). The grants fall into two categories: those that have been assigned to at least one

FIGURE 3  
RELATIONSHIP BETWEEN ESTIMATED NIH RESEARCH FUNDING,  
BY DISEASE, AND NUMBER OF NIH GRANTS CITING DISEASE



SOURCE: Author's calculations based on NCHS 1980 Vital Statistics-Mortality Detail file and NIH FY1982 CRISP file.

disease and those that have not.<sup>12</sup> These estimates of research activity by disease are based only on grants that have been assigned.<sup>13</sup> Because of the logarithmic specification of equation 6, the validity of these parameter estimates does not require us to measure the absolute level of research funding reliably by disease; their validity is predicated only on the reliable measurement of relative research funding or activity. If the disease distribution of unassigned grants is similar to that of assigned grants, these estimates could be regarded as applicable to all public biomedical research.

**Data on Disease Burden, Prevalence, and Incidence.** As indicated in equation 4, rather than treating disease burden  $N$  as a scalar, we regard it as an index of a number of disease attributes. Data on these attributes are obtained

from two sources: the Vital Statistics–Mortality Detail file, a virtually complete census of deaths in the United States, and the National Health Interview Survey (NHIS), a continuing nationwide survey of households for which a probability sample of the civilian noninstitutionalized<sup>14</sup> population of the United States is interviewed by the Bureau of the Census regarding health and other characteristics of each member of the household. (The sample for the years 1990–1992 covered 142,638 households containing 368,075 persons.)

This use of these two data sources reflects the belief that obtaining a reasonably complete accounting for disease burden must consider data on both the dying and the living. Analysis based on only one source will almost surely be subject to considerable sample selection bias. Because the disease classifications used for the mortality and morbidity data are quite different, these two dimensions of disease burden require separate analyses.

**Premature Mortality Data.** The measure of disease burden computed from the mortality file is potential life-years lost before age sixty-five because of disease.<sup>15</sup> (Potential life-years lost is sixty-five minus the age at death of decedents younger than age sixty-five.) This is a standard measure of disease burden, or progress against disease, in health statistics. The drawback is that it ignores the deaths of people aged sixty-five and older.

**Data on Prevalence of Selected Chronic Conditions.** Collins (1997) presents statistics on the prevalence of selected chronic conditions in the United States during 1990–1992 by age, sex, race, family income, and geographic region, as derived from data collected in the NHIS. He also reports the percentages of selected conditions that limit activity, the percentages for which a physician was consulted, and the percentages that caused hospitalization.

The survey collects information from responsible family members residing in the household. Methodological studies have shown that chronic conditions are generally underreported in interview surveys. Respondents in health interviews tend to report conditions that they are aware of and are willing to report to the interviewer. Reporting is more reliable for conditions that have significantly affected individuals and their families. Conditions that are severe or costly, or are being treated, tend to be better reported than others. Methodological studies have also indicated that the inclusion of a checklist of descriptive titles of conditions as part of the interview questionnaire increases the probability that a respondent will recognize the terms and report those conditions.

The current procedure for collecting information on chronic conditions was established in 1978. Six categorical lists of selected chronic conditions are included in the NHIS questionnaire: circulatory conditions; respiratory conditions; digestive conditions; impairments and conditions of the nervous system and sense organs; conditions of the skin and subcutaneous tissue and of the musculoskeletal system and connective tissue; and endocrine, nutritional, and metabolic diseases and immunity disorders, diseases of the blood and blood-forming organs, and conditions of the genitourinary system. Each family in the NHIS is questioned for only one of these six lists, selected on a predetermined basis: each list is thus administered to only one-sixth of the total NHIS sample each year. For some items, responses are based on the following question: "During the past twelve months did anyone in the family (read names) have . . .?" For other items, responses are based on the question "Does anyone in the family (read names) now have . . .?" For the rest, responses are based on the question "Has anyone in the family (read names) ever had . . .?" Estimates for days of disability caused by chronic conditions are based on the number of disability days reported for the two weeks before the interview.

The survey includes data only on persons living in the household at the time of the interview. The data do not report any conditions of persons who died before the interview. Also excluded is the experience of persons who were institutionalized or who were members of the armed forces at the time of the household interview.

In these data, *prevalence* is defined as the average number of some condition existing during a specified interval—usually referred to as *period prevalence*—rather than the number of some condition existing at a given point in time—usually referred to as *point prevalence*. *Chronic conditions* are defined as conditions that either were first noticed three months or more before the date of the interview or belong to a group of conditions considered chronic regardless of onset.

The data represent the prevalence of conditions, not the prevalence of persons with a chronic condition. For most conditions, however, the condition prevalence and the person prevalence are almost identical.<sup>16</sup>

### **Preliminary Estimates**

**Premature Mortality.** The first measure of disease burden analyzed is potential life-years lost before age sixty-five (PLYL). Data on PLYL in 1980 and on government research funding in 1982 for fourteen major disease categories are shown, in descending PLYL order, in table 3. Diseases of the circulatory system and neoplasms record, by far, the largest tolls in terms of premature death. While the research funding for these two diseases is among the highest for all diseases, R&D funding for two other diseases with much smaller burdens exceeds that funding, in one case by a large amount. Nevertheless, as the scatter plot in figure 4 and the following regression indicate, there is a strong positive relationship across the entire sample between life-years lost and public R&D expenditure ( $t$ -statistics in parentheses):

TABLE 3  
 LIFE-YEARS LOST BEFORE AGE SIXTY-FIVE IN 1980,  
 AND PUBLIC R&D EXPENDITURE IN 1982, FOR  
 FOURTEEN MAJOR DISEASE CATEGORIES

<i>Disease or Disorder (ICD9 codes)</i>	<i>Life-Years Lost before Age 65 in 1980</i>	<i>Public R&amp;D Expenditure in 1982 (millions of \$)</i>
Circulatory system (390-459)	2,043,559	117
Neoplasms (140-239)	1,860,531	113
Congenital anomalies (740-759)	760,820	37
Digestive system (520-579)	503,531	27
Respiratory system (460-519)	434,770	47
Nervous system and sense organs (320-389)	294,239	118
Endocrine, nutritional, and metabolic diseases and immunity disorders	223,015	168
Infectious and parasitic diseases (001-139)	162,568	33
Mental disorders (290-319)	124,407	70
Genitourinary system (580-629)	86,015	73
Diseases of the blood and blood-forming organs (280-289)	49,814	16
Musculoskeletal system and connective tissue	37,403	25
Complications of pregnancy, childbirth, and the puerperium	12,536	8
Skin and subcutaneous tissue (680-709)	7,848	21

SOURCE: Author's calculations based on NCHS 1980 Vital Statistics–Mortality Detail file and NIH FY1982 CRISP file.

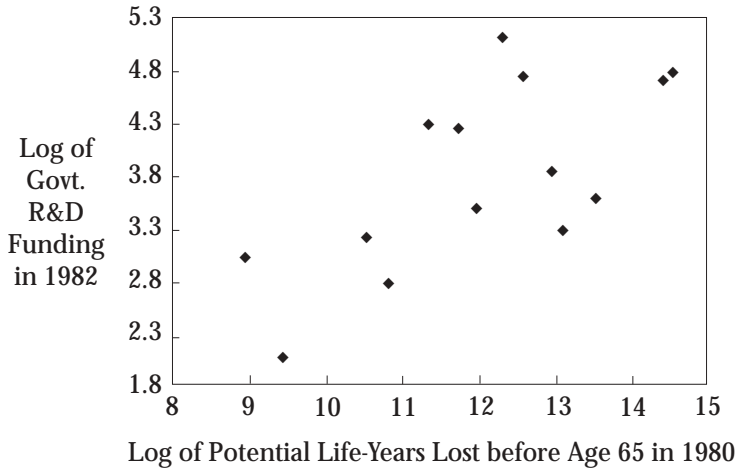
$$\ln(\text{RD82}) = -0.464 + 0.355 \ln(\text{LYL80}) + e.$$

(0.34)    (3.19)

$$R^2 = .459 \quad N = 14$$

The total life-years lost in 1980 explains almost half the variation across diseases in 1982 research expenditure. Con-

FIGURE 4  
RELATIONSHIP BETWEEN GOVERNMENT RESEARCH FUNDING,  
BY DISEASE, IN 1982, AND LIFE-YEARS LOST BEFORE AGE SIXTY-FIVE,  
BY DISEASE, IN 1980



SOURCE: Author's calculations based on NCHS 1980 Vital Statistics-Mortality Detail file and NIH FY1982 CRISP file.

trary to the implication of our simple theoretical model of research allocation, however, the coefficient for  $\ln(\text{LYL80})$  is significantly less than one. As argued, this may result from a negative correlation between the regressor and the omitted variable for research productivity.

Life-years lost can be classified by sex, race, educational attainment, and other characteristics; thus, we can investigate whether premature mortality among certain demographic groups tends to be associated with especially high government research funding. Sixty percent of life-years lost before age sixty-five are lost by males, and 25 percent are lost by nonwhites, who are about 12 percent of the population (NCHS 1995, table 1); these figures reflect the lower life expectancy of these two groups. The proportion of life-years lost by men and by nonwhites varies consider-

TABLE 4  
MATRIX OF CORRELATION COEFFICIENTS

	<i>R&amp;D 1982</i>	<i>Life-Years Lost, 1980</i>	<i>Male (%)</i>
Life-years lost, 1980	0.67714 0.0078		
Male (%)	0.55375 0.0399	0.56093 0.0369	
White (%)	0.75643 0.0017	0.88477 0.0001	0.50235 0.0672

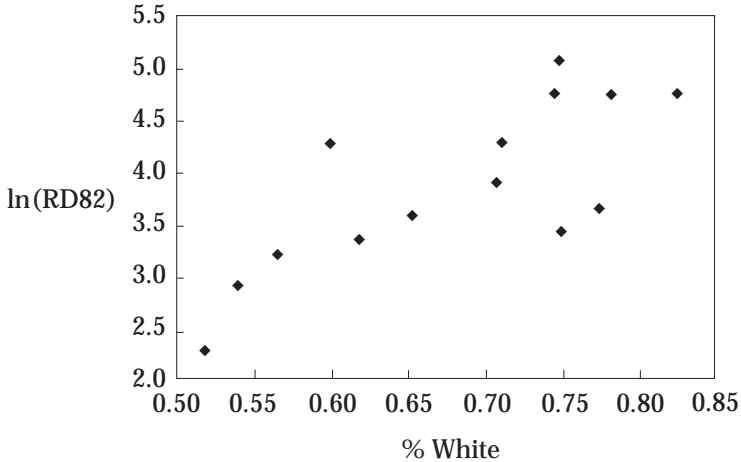
NOTE: *P*-values are below coefficients.

SOURCE: Author's calculations based on NCHS 1980 Vital Statistics—Mortality Detail file and NIH FY1982 CRISP file.

ably across diseases. Whites account for 81 percent of life-years lost to neoplasms but for only 53 percent of those lost to diseases of the blood and blood-forming organs. Men account for 81 percent of life-years lost to infectious and parasitic diseases but for only 28 percent of life-years lost to musculoskeletal and connective-tissue diseases.

The matrix of correlation coefficients for four variables— $\ln(\text{RD82})$ ,  $\ln(\text{LYL80})$ , and the fractions of life-years lost to men (%MALE) and to whites (%WHITE)—is reported in table 4 (*p*-values are shown below the correlation coefficients). Public R&D investment is significantly positively correlated with the fractions of life-years lost to men, especially to whites, as well as with the total number of life-years lost. Indeed, %WHITE is more strongly correlated with R&D than is total life-years lost. A scatter plot of  $\ln(\text{RD82})$  against %WHITE is shown in figure 5. But as the second column of coefficients reveals, both %MALE and %WHITE are significantly positively correlated with total life-years lost: the diseases associated with the greatest number of premature deaths are those for which men and whites account for the greatest fractions of life-years lost. We therefore need to determine whether %MALE and %WHITE

FIGURE 5  
CORRELATION OF R&D WITH PERCENTAGE OF  
LIFE-YEARS LOST BY WHITE MEN



SOURCE: Author's calculations based on NCHS 1980 Vital Statistics-Mortality Detail file and NIH FY1982 CRISP file.

have significant effects on public R&D, controlling for total life-years lost (although our ability to determine this will be hampered by multicollinearity). The appropriate regressions are

$$\ln(\text{RD82}) = -0.164 + 0.280 \ln(\text{LYL80}) + 1.11 \% \text{MALE} + e$$

(0.12)      (2.08)                      (0.99)

$$R^2 = .503 \quad N = 14$$

$$\ln(\text{RD82}) = -0.813 + 0.019 \ln(\text{LYL80}) + 6.56 \% \text{WHITE} + e$$

(0.64)      (0.09)                      (1.71)

$$R^2 = .573 \quad N = 14$$

The coefficient for %MALE is insignificant, and the inclusion of this variable reduces only slightly the coefficient for

$\ln(\text{LYL80})$ . In contrast, the coefficient for %WHITE is marginally significant, even in the presence of the other regressor, which becomes completely insignificant when %WHITE is included. We also estimate an alternative functional form of the relationship  $\text{RD82} = f(\text{LYL80}, \% \text{WHITE})$ :

$$\begin{aligned} \ln(\text{RD82}) &= 2.29 + 1.35 \ln(\text{LYL80} * \% \text{WHITE}) \\ &\quad (1.14) \quad (1.90) \\ &\quad - 1.30 \ln(\text{LYL80} * (1 - \% \text{WHITE})) + e \\ &\quad (1.44) \\ R^2 &= .560 \quad N = 14 \end{aligned}$$

These estimates indicate that research expenditure is positively correlated with life-years lost by whites but not with life-years lost by nonwhites; the coefficient on the latter is negative, but its *p*-value is only 0.18. The two coefficients are virtually equal in magnitude and opposite in sign; if one imposes that restriction (which is not nearly rejected by the data), the estimates are

$$\begin{aligned} \ln(\text{RD82}) &= 2.72 + 1.47 \ln(\% \text{WHITE} / (1 - \% \text{WHITE})) + e \\ &\quad (8.40) \quad (3.89) \\ R^2 &= .558 \quad N = 14 \end{aligned}$$

The data are highly consistent with the hypothesis that the amount of publicly funded research on a disease decreases with the share of life-years before age sixty-five that are lost to the disease by nonwhites. A possible explanation for this finding is that the lack of scientific knowledge is a less important cause of premature mortality among nonwhites than it is among whites. Nonwhite premature mortality may be due, to a greater extent, to poor diet, less use of medical care, or other factors. In other words, the health status of nonwhites may be well below the frontier of medical knowledge, whereas the health

status of whites tends to be on or closer to the frontier. The purpose of biomedical research is to shift the frontier outward; the allocation or direction of research should depend more on the distribution of the disease burden of those on or close to the frontier. If cures for diseases that impose a heavy toll on minorities have already been found, then the productivity of further research on those diseases may be quite low.

The relative lack of research on diseases borne disproportionately by minorities may also be due to other reasons. The lack may reflect the relatively low representation of minorities among biomedical scientists. The National Science Foundation monitors the participation of women and minorities in science and engineering and has adopted some policies to increase their participation.

**Prevalence and Severity of Chronic Conditions.** Table 5 presents data on the number of FY1995 research grants mentioning chronic conditions surveyed in the National Health Interview Survey and the number of people having, and limited in activity by, these conditions.<sup>17</sup> The condition mentioned in the most research grants (1,807) is diabetes. About 7 million Americans suffer from diabetes, according to this household survey; the condition limits the activities of about one-third of them. Although arthritis, afflicting 32 million Americans, is far more prevalent, fewer research grants (609) are involved.

Table 6 presents correlation coefficients for the logarithms of these variables and related measures of condition severity. This table indicates that the number of research grants mentioning a chronic condition is completely uncorrelated with the number of people with the condition and with the number who have seen a physician about that condition. Research activity is weakly positively related ( $p$ -value = 0.08) to the number of people who have been hospitalized for a condition. It is strongly positively related ( $p$ -value = 0.0003) to the number of



TABLE 5  
 FY1995 RESEARCH GRANTS AND NUMBER OF  
 PEOPLE REPORTING AND LIMITED IN ACTIVITY,  
 FOR MAJOR CHRONIC CONDITIONS, 1990-92

<i>Chronic Condition</i>	<i>Number of Grants</i>	<i>People with Condition (000s)</i>	<i>People with Limited Activity (000s)</i>
Diabetes	1,807	6,962	2,415.81
High blood pressure (hypertension)	1,540	27,600	2,925.60
Diseases of retina	671	1,293	373.68
Arthritis	609	31,788	6,739.06
Diseases of prostate	593	1,513	78.68
Anemias	573	3,739	157.04
Asthma	493	11,482	2,503.08
Epilepsy	425	1,243	551.89
Mental retardation	402	1,562	1,366.75
Blindness and other visual impairments	315	8,169	1,290.70
Liver diseases including cirrhosis	293	766	130.22
Cerebrovascular disease	288	3,002	1,077.72
Deafness and other hearing impairments	282	23,266	1,279.63
Ischemic heart disease	258	7,732	2,435.58
Multiple sclerosis	241	180	124.92
Speech impairments	241	2,725	555.90
Malignant neoplasm of breast	216	802	190.07
Cataracts	203	6,416	391.38
Glaucoma	195	2,433	326.02
Enteritis and colitis	119	2,333	160.98
Congenital heart disease	118	741	132.64
Disease of the esophagus	118	834	45.04
Malignant neoplasms of stomach, intestines, colon, and rectum	103	322	199.96
Malignant neoplasms of lung bronchus and other respiratory	99	218	132.11
Menstrual disorders	90	1,984	17.86
Psoriasis	85	2,378	49.94
Emphysema	84	1,861	820.70
Kidney infections	84	1,325	46.38
Tachycardia or rapid heart	84	1,911	133.77
Heart rhythm disorders	83	7,868	503.55

<i>Chronic Condition</i>	<i>Number of Grants</i>	<i>People with Condition (000s)</i>	<i>People with Limited Activity (000s)</i>
Malignant neoplasm of prostate	83	344	76.02
Ulcer, gastric, duodenal, and/or peptic	80	4,201	327.68
Malignant neoplasms of the skin	74	2,269	88.49
Hardening of arteries	61	2,074	199.10
Gastric ulcer	59	3,121	240.32
Benign neoplasm of breast	54	73	1.97
Cleft palate	53	217	6.94
Gastritis and duodenitis	50	3,003	30.03
Peptic ulcer	49	468	24.80
Neuritis or neuralgia unspecified	47	518	23.83
Cerebral palsy	46	258	191.95
Dermatitis	44	9,273	139.10
Kidney stones	42	1,009	34.31
Chronic ulcer of skin	35	332	80.01
Aneurysm	33	226	35.03
Bladder infections	32	1,616	40.40
Duodenal ulcer	31	613	63.75
Inflammatory disease of female genital organs	31	184	4.05
Malignant neoplasm of female genital organs	31	221	36.02
Constipation	25	4,302	30.11
Benign neoplasms of the skin	24	862	18.10
Chronic bronchitis	22	12,884	257.68
Pneumoconiosis and asbestosis	21	324	84.89
Goiter	20	478	26.77
Migraine headache	20	9,992	339.73
Rheumatic fever with or without heart disease	16	2,029	306.38
Acne	15	4,904	.
Gout	14	2,167	208.03
Rheumatism unspecified	14	454	77.18
Benign neoplasm of female genital organs	13	746	37.30

*(continues)*

TABLE 5 (*continued*)

<i>Chronic Condition</i>	<i>Number of Grants</i>	<i>People with Condition (000s)</i>	<i>People with Limited Activity (000s)</i>
Chronic sinusitis	13	33,736	168.68
Tinnitus	13	7,144	57.15
Color blindness	11	2,697	5.39
Hay fever or allergic rhinitis without asthma	10	24,060	336.84
Gallbladder stones	9	1,068	117.48
Intervertebral disc disorders	7	4,976	2249.15
Sciatica (including lumbago)	5	2,058	133.77
Hernia of abdominal cavity	4	4,768	467.26
Bone spur or tendinitis not otherwise specified	3	2,633	252.77
Varicose veins of lower extremities	3	7,403	162.87
Deformities or orthopedic impairments of back	2	18,144	4064.26
Phlebitis thrombophlebitis	2	727	125.04
Pleurisy	2	690	11.04
Chronic laryngitis	1	1,508	.
Heart murmurs	1	4,276	94.07
Hemorrhoids	1	9,441	37.76
Nasal polyps	1	805	3.22
Spastic colon	1	1,686	45.52
Bunions	0	2,907	26.16
Bursitis not elsewhere classified	0	4,674	247.72
Chronic disease of tonsils and adenoids	0	2,836	11.34
Corns and calluses	0	4,731	23.66
Curvature or other deformity of back or spine	0	5,078	619.52
Deviated nasal septum	0	1,646	3.29
Diverticula of intestines	0	1,999	85.96
Flat feet	0	3,698	44.38
Indigestion and other functional disorders of stomach	0	6,437	70.81
Ingrown nails	0	6,078	18.23
Noninflammatory disease of female genital organs	0	1,219	0.00
Sebaceous skin cyst	0	1,249	2.50

SOURCE: Author's calculations based on NIH FY1995 CRISP file and Collins 1997.

people whose activities are limited by that condition. Somewhat surprisingly, research activity is significantly positively correlated with the proportion of people who have seen a doctor or been hospitalized, as well as those whose activities are limited.<sup>18</sup>

The determinants of the number of research grants citing chronic conditions are further analyzed in table 7. The first column presents the regression of  $\ln(NGRANTS95)$  on a measure of condition prevalence ( $\ln N$ ) and severity ( $\%LA$ ). As one might expect given the simple correlations in table 6, only the severity measure has a significant positive effect on research activity. In the second column we estimate an alternative functional form of the relationship; the regressors are the logarithms of the number of people with the condition whose activities are ( $N * \%LA$ ) and are not ( $N * (1 - \%LA)$ ) limited by the condition. The coefficient for the former is positive and highly significant; this indicates that the amount of public research about a chronic condition increases with the number of people whose activities are limited by that condition.<sup>19</sup> Moreover, the amount of public research is significantly inversely related to the number of people who have a condition but whose activities are not limited by it. Conceivably, the greater the number of people who have a condition but are not seriously affected by it, the greater may be the odds that an adequate treatment for the condition already exists, and the less worthy that condition is of further research.

This inverse relation becomes insignificant, however, when we include (in column 3) measures of the income- and age-distribution of persons reporting the condition. This regression indicates that there tends to be more research about chronic conditions that are prevalent among people living in low-income (below \$10,000) households and that are prevalent among the young (under age eighteen) and the old (older than seventy-five). The poor, the young, and the very old may derive disproportionately large benefits from government-sponsored biomedical research.

TABLE 6  
CORRELATIONS BETWEEN RESEARCH ACTIVITY AND PREVALENCE/SEVERITY OF CHRONIC CONDITIONS

	LGRANTS	LN	LNLA	LNHOSP
LGRANTS	1.0000	0.03858	0.39969	0.19966
log(number of research grants)	0.0	0.7374	0.0003	0.0817
	78	78	76	77
LN	0.03858	1.00000	0.54194	0.61135
log(number of people with condition)	0.7374	0.0	0.0001	0.0001
	78	126	123	125
LNLA	0.39969	0.54194	1.00000	0.73564
log(number with limited activity)	0.0003	0.0001	0.0	0.0001
	76	123	123	122
LNHOSP	0.19966	0.61135	0.73564	1.00000
log(number hospitalized)	0.0817	0.0001	0.0001	0.0
	77	125	122	125

LNPHYS						
log (number seeing physician)	0.07243	0.99309	0.59053	0.66237		
	0.5286	0.0001	0.0001	0.0001		
LA						
% with limited activity	78	126	123	125		
	0.34507	-0.26328	0.49119	0.11785		
	0.0023	0.0031	0.0001	0.1942		
HOSP						
% hospitalized	76	124	123	123		
	0.21568	-0.45766	0.09308	0.28156		
	0.0596	0.0001	0.3079	0.0015		
PHYS						
% seeing physician	77	125	122	125		
	0.32167	-0.42512	0.17895	0.18110		
	0.0041	0.0001	0.0477	0.0433		
	78	126	123	125		

---

NOTE: Pearson Correlation Coefficients/Prob > |R| under Ho: Rho = 0/Number of Observations.  
SOURCE: Author's calculations based on NIH FY1995 CRISP file and Collins 1997.

TABLE 7  
 DETERMINANTS OF NUMBER OF FY1995 RESEARCH GRANTS  
 MENTIONING CHRONIC CONDITIONS  
 ( $N = 54$ )

<i>Variable</i>	<i>Equation 1</i>	<i>Equation 2</i>	<i>Equation 3</i>
$\ln(N)$	0.142 (0.73)		
%LA	4.45 (2.77)		
$\ln(N * \%LA)$		0.651 (4.12)	0.369 (2.39)
$\ln(N * (1 - \%LA))$		-0.436 (2.17)	-0.167 (0.88)
%INCOME < \$10K			8.61 (2.17)
%AGE < 18			5.67 (2.63)
%AGE > 75			7.30 (2.73)
Intercept	2.09 (1.31)	3.82 (2.81)	0.267 (0.17)
$R^2$	0.1317	0.2460	0.4470

NOTE: The dependent variable is the log of the number of FY1995 grants. Figures in parentheses are *t*-statistics.

$N$ : Average number of people (in thousands) in 1990–92 reporting that they have the condition.

%LA: Fraction of people reporting their activities limited by the condition.

%INCOME < \$10K: Fraction of people with household income < \$10K.

%AGE < 18: Fraction of people under 18 years of age.

%AGE > 75: Fraction of people over 75 years of age.

SOURCE: Author.

The previous section reported that the amount of publicly funded research on a disease decreases with the share of life-years before age sixty-five lost to the disease by nonwhites. Since nonwhites are more likely to be poor than whites, chronic conditions prevalent among the poor surprisingly tend to be more intensively researched.

## Summary

In this study I have developed a simple theoretical model of the allocation of public biomedical research expenditure and have presented some empirical evidence about the determinants of this allocation. The implications of the theoretical model are consistent with descriptions of the allocation process by government officials: the structure of expenditure should depend on research productivity (or scientific opportunity) as well as on public health need, that is, the societal and economic burden of the disease or condition.

Although at this point we lack useful indicators of research productivity (that is, of the cost of achieving research advances), we have several measures of disease burden (that is, of the benefit of achieving these advances). Analysts of technological change typically have data on neither the costs nor the benefits of technical advance. Failure to measure research productivity will not necessarily bias these estimates; if it does, it seems likely to bias them toward zero.

This study has calculated distributions of government-funded biomedical research expenditure by disease from records of all research projects supported by the Public Health Service; in fiscal year 1995, there were records of 63,289 projects, with a total value of \$10.1 billion. Some research expenditure cannot be assigned to specific diseases, in some cases because the research is basic in nature. The distribution of research expenditure by disease constructed here is quite similar to one calculated by NIH based on data provided by NIH institutes, centers, and divisions (ICDs) and designed to “reflect NIH-wide resources devoted to research on the listed diseases” (as opposed to budget figures for the ICD identifying the cost data).

The study performed an empirical examination of the relationship of public research expenditure to several measures of disease burden. To avoid sample-selection bias and to obtain a reasonably complete accounting of disease bur-



den, data on both the dying (from the Vital Statistics–Mortality Detail file) and the living (from the National Health Interview Survey) were used.

The mortality-related measure of disease burden used is life-years lost before age sixty-five. There is a strong positive relationship across diseases between total life-years lost and public R&D expenditure (although the slope of this relationship was smaller than that implied by the theory, perhaps because of a failure to measure research productivity). Further analysis indicated that research expenditure is positively correlated with life-years lost by whites but not with life-years lost by nonwhites. The amount of publicly funded research on a disease decreases with the share of life-years before age sixty-five lost to the disease by nonwhites. A possible explanation for this finding is that lack of scientific knowledge is a less important cause of premature mortality among nonwhites than among whites.

Disease prevalence and severity data for the living population provide additional indicators of disease burden. The number of research grants mentioning a chronic condition is completely uncorrelated with the number of people having the condition and with the number who have seen a physician about that condition. Research activity is weakly positively related to the number of people who have been hospitalized for a condition and strongly positively related to the number of people whose activities are limited by that condition. Moreover, there tends to be more research about chronic conditions that are prevalent among people living in low-income households, and that are prevalent among the young (younger than eighteen) and the old (older than seventy-five).

# Notes

1. See chapter 2 of Jones 1997 for an excellent discussion of the Solow model.

2. This calculation assumes that all nonfederal R&D is civilian (that is, not defense- or space-related).

3. The distribution of cost savings, for example, could be highly skewed to the right—a few programs confer large cost savings, but the majority confer scant savings—and the specific examples chosen could tend to be concentrated in the upper tail of the distribution.

4. See NIH 1997 for NIH's own description of how it makes "choices about where and how it spends its money, approximately \$13 billion in fiscal year 1997."

5. Viscusi (1995, 3) notes that "in the case of biomedical research, the typical outcome will be a change in societal risk levels induced by the biomedical research outcomes."

6. Assume, for simplicity, that policymakers do not pay attention to *privately* funded biomedical R&D; that is, they are not merely trying to fill gaps in private research, nor do they consider the potential impact of public R&D on future private research activity. Toole (1997), however, presents evidence that suggests that public biomedical research may have a significant, albeit delayed, impact on private drug discovery.

7. NIH officials acknowledge that "research funding decisions will also reflect concerns about equity among groups of potential beneficiaries of the research as defined in terms of age, sex, and ethnic origin. Certain criteria favor one group over another. For example, mortality rates and measures of the impact on functioning may favor the elderly whereas measures of economic impact, such as lost productivity, would favor younger citizens" (Varmus 1995).

8. Henderson and Cockburn (1996) have studied the determinants of research productivity of pharmaceutical firms, with patents and scientific papers as measures of research output.

9. Garber and Romer (1995) also argue that “federal policy toward research and development should respond to scientific advances, technology trends, and changes in the political and social environment.”

10. The reasoning underlying this is the same as that underlying Gary Chamberlin’s argument that estimation of production functions using data for a cross-section of firms will result in overestimates of the returns to factors of production, for example, labor. Firms with exogenously higher productivity (due to, for example, greater managerial ability) will employ more workers.

11. Data on the disease distribution of *private* R&D sponsored by pharmaceutical firms are available from the Pharmaceutical Research and Manufacturers Association’s Annual Survey of companies. Unfortunately, the private R&D data are disaggregated into only about seven broad categories. Figure 2 shows the percentage distributions of both private and government R&D, by these categories. Public R&D seems to be more concentrated on digestive/genitourinary and neoplasm/endocrine/metabolic diseases and less concentrated on infective/parasitic, nervous system, and cardiovascular diseases than is private R&D.

12. This distinction resembles the distinction made in industrial R&D between basic and applied research.

13. When two or more diseases are cited by a grant, I assigned the *entire* amount of funding for the grant to *each* of the diseases cited.

14. The restriction of the NHIS to the civilian population not confined to institutions affects the estimated prevalence of chronic conditions. Omission of the institutionalized population reduces the prevalence estimates, especially for the elderly, because the proportion of persons in institutions who have chronic conditions is high. These estimates do not indicate the prevalence in the total population.

15. Demographic information on the death certificate is provided by the funeral director based on information supplied by an informant. Medical certification of the cause of death is provided by a physician, medical examiner, or coroner.

16. Great variances occur for two different reasons. First, a

prevalence estimate of a condition may include more than one of the specified checklist items or a checklist item and a specified “other condition” item that falls into the same ICD category as the checklist item. Second, some prevalence categories shown are a combination of other categories; as a result, a person may have more than one of the conditions that are added to form the combined category. The concept of condition prevalence is generally used in NHIS, because specific health indexes such as limitation of activity and disability days can be ascribed to specific conditions. In addition, prosthetic and pharmaceutical treatment modes are more condition specific than person specific.

17. This section uses as the measure of public research activity the number of grants rather than the dollar value of those grants. For technical reasons the former is much easier to compute. Substitution of the former for the latter will not affect results if the average size of grants is uncorrelated across conditions with the number of grants. Future study will compute the distribution of dollars by condition and integrate the premature mortality and chronic-condition prevalence analyses.

18. This is particularly surprising since, as the second column of the table indicates, these proportions are significantly inversely related to prevalence per se: conditions that are more prevalent tend to be less severe (that is, associated with lower probabilities of hospitalization, limitation of activity, and physician consultation).

19. As in the analysis of premature mortality, however, the elasticity is significantly less than unity.



## References

- Advisory Commission to Study the Consumer Price Index. 1995. *Toward a More Accurate Measure of the Cost of Living*. Interim Report to the Senate Finance Committee, September 15. Washington, D.C.
- Collins, J. G. 1997. "Prevalence of Selected Chronic Conditions: United States, 1990–1992." *Vital Health Statistics*, series 10, vol. 194.
- Garber, Alan, and Paul Romer. 1995. "Evaluating the Federal Role in Financing Health-Related Research." Paper prepared for October 19 Roundtable on Economics, National Institutes of Health.
- Griliches, Zvi, and Frank Lichtenberg. 1984. "R&D and Productivity at the Industry Level: Is There Still a Relationship?" In *R&D, Patents, and Productivity*, edited by Zvi Griliches. Chicago: University of Chicago Press.
- Henderson, Rebecca, and Iain Cockburn. 1996. "Scale, Scope, and Spillovers: The Determinants of Research Productivity in Drug Discovery." *RAND Journal of Economics* 27 (1) (spring): 32–59.
- Jones, Charles, 1997. *Introduction to Economic Growth*. New York: W.W. Norton.
- Lichtenberg, Frank. 1998. "Pharmaceutical Innovation, Mortality Reduction, and Economic Growth." Working Paper W6569, May. Cambridge, Mass.: National Bureau of Economic Research.
- Mankiw, N. Gregory. 1992. *Macroeconomics*. New York: Worth Publishers.
- Mushkin, Selma. 1979. *Biomedical Research: Costs and Benefits*. Cambridge, Mass.: Ballinger.

- National Center for Health Statistics. 1995. *Health, United States, 1994*. Hyattsville, Md.: Public Health Service.
- National Institutes of Health. 1993. *Cost Savings Resulting from NIH Research Support*. 2d ed. NIH Publication 93-3109. Rockville, Md.: NIH.
- . 1995. "Disease-Specific Estimates of Direct and Indirect Costs of Illness and NIH Support."
- . 1997. "Setting Research Priorities at the National Institutes of Health." [Updated as needed at <http://www.nih.gov/news/ResPriority/priority.htm>.]
- National Science Foundation. 1996. *National Patterns of R&D Resources: 1996*. NSF 96-333. Arlington, Va.: NSF.
- Nordhaus, William. 1998. "The Health of Nations: Irving Fisher and the Contribution of Improved Longevity to Living Standards." Paper presented at Fisher Conference, Yale University, May.
- Toole, Andrew. 1997. "The Impact of Federally Funded Basic Research on Industrial Innovation: Evidence from the Pharmaceutical Industry." Madison, Wisc.: Laurits R. Christensen Associates.
- Varmus, Harold. 1995. "Responses to Questions from Senator Slade Gordon." Labor, HHS, Education Subcommittee Hearing, NIH appropriations for FY1996, May 18.
- Viscusi, W. Kip. 1995. "Valuing the Health Consequences of Biomedical Research." Paper prepared for October 19 Roundtable on Economics, National Institutes of Health.

## About the Author

FRANK R. LICHTENBERG is Courtney C. Brown Professor of Business at the Columbia University Graduate School of Business and a research associate of the National Bureau of Economic Research. He has taught at Harvard University and the University of Pennsylvania and has been a visiting scholar at the Wissenschaftszentrum Berlin and the University of Munich.

Mr. Lichtenberg has worked for several U.S. agencies, including the Department of Justice, Congressional Budget Office, and Census Bureau, and has been an econometrics expert for the Federal Trade Commission.

The author's research has examined how the introduction of new technology arising from research and development affects the productivity of companies, industries, and nations. His book *Corporate Takeovers and Productivity* was published by MIT Press.

He has received research fellowships, grants, and contracts from the National Science Foundation, National Institute of Standards and Technology, Fulbright Commission, Brookings Institution, Alfred P. Sloan Foundation, German Marshall Fund, and American Enterprise Institute and from Merck and Co. He was awarded the 1998 Schumpeter Prize for his paper "Pharmaceutical Innovation as a Process of Creative Destruction."

The author holds a B.A. in history from the University of Chicago and an M.A. and Ph.D. in economics from the University of Pennsylvania.



---

---

## Board of Trustees

Edward B. Rust, Jr., *Chairman*  
Chairman and CEO  
State Farm Insurance Companies

Tully M. Friedman, *Treasurer*  
Chairman  
Friedman Fleischer & Lowe, LLC

Joseph A. Cannon  
Chairman and CEO  
Geneva Steel Company

Dick Cheney  
CEO  
Halliburton Company

Harlan Crow  
Chief Executive Officer  
Crow Holdings

Christopher C. DeMuth  
President  
American Enterprise Institute

Steve Forbes  
President and CEO  
Forbes Inc.

Christopher B. Galvin  
CEO  
Motorola, Inc.

Harvey Golub  
Chairman and CEO  
American Express Company

Robert F. Greenhill  
Chairman  
Greenhill & Co., LLC

Roger Hertog  
President and COO  
Sanford C. Bernstein and Company

M. Douglas Ivester  
Chairman and CEO  
The Coca-Cola Company

Martin M. Koffel  
Chairman and CEO  
URS Corporation

Bruce Kovner  
Chairman  
Caxton Corporation

Kenneth L. Lay  
Chairman and CEO  
Enron Corp.

John A. Luke, Jr.  
Chairman, President, and CEO  
Westvaco Corporation

Alex J. Mandl  
Chairman and CEO  
Teligent, Inc.

Craig O. McCaw  
Chairman and CEO  
Eagle River, Inc.

## The American Enterprise Institute for Public Policy Research

Founded in 1943, AEI is a nonpartisan, nonprofit research and educational organization based in Washington, D. C. The Institute sponsors research, conducts seminars and conferences, and publishes books and periodicals.

AEI's research is carried out under three major programs: Economic Policy Studies; Foreign Policy and Defense Studies; and Social and Political Studies. The resident scholars and fellows listed in these pages are part of a network that also includes ninety adjunct scholars at leading universities throughout the United States and in several foreign countries.

The views expressed in AEI publications are those of the authors and do not necessarily reflect the views of the staff, advisory panels, officers, or trustees.

Paul H. O'Neill  
Chairman and CEO  
Alcoa

John E. Pepper  
Chairman  
The Procter & Gamble Company

George R. Roberts  
Kohlberg Kravis Roberts & Co.

John W. Rowe  
Chairman, President, and CEO  
Unicom Corporation

James P. Schadt  
Chairman  
Dailey Capital Management

John W. Snow  
Chairman, President, and CEO  
CSX Corporation

William S. Stavropoulos  
Chairman and CEO  
The Dow Chemical Company

Wilson H. Taylor  
Chairman and CEO  
CIGNA Corporation

Marilyn Ware  
Chairman  
American Water Works Co., Inc.

James Q. Wilson  
James A. Collins Professor of  
Management Emeritus  
University of California at Los  
Angeles

## Officers

Christopher C. DeMuth  
President

David Gerson  
Executive Vice President

John R. Bolton  
Senior Vice President

## Council of Academic Advisers

James Q. Wilson, *Chairman*  
James A. Collins Professor of  
Management Emeritus  
University of California at Los  
Angeles

Gertrude Himmelfarb  
Distinguished Professor of History  
Emeritus  
City University of New York

Samuel P. Huntington  
Albert J. Weatherhead III University  
Professor of Government  
Harvard University

D. Gale Johnson  
Eliakim Hastings Moore  
Distinguished Service Professor of  
Economics Emeritus  
University of Chicago

William M. Landes  
Clifton R. Musser Professor of  
Economics  
University of Chicago Law School

---

**Sam Peltzman**  
Sears Roebuck Professor of  
Economics and Financial Services  
University of Chicago Graduate  
School of Business

**Nelson W. Polsby**  
Professor of Political Science  
University of California at Berkeley

**George L. Priest**  
John M. Olin Professor of Law and  
Economics  
Yale Law School

**Thomas Sowell**  
Senior Fellow  
Hoover Institution  
Stanford University

**Murray L. Weidenbaum**  
Mallinckrodt Distinguished  
University Professor  
Washington University

**Paul Wolfowitz**  
Dean, Paul H. Nitze School of  
Advanced International Studies  
Johns Hopkins University

**Richard J. Zeckhauser**  
Frank Ramsey Professor of Political  
Economy  
Kennedy School of Government  
Harvard University

## Research Staff

**Leon Aron**  
Resident Scholar

**Claude E. Barfield**  
Resident Scholar; Director, Science  
and Technology Policy Studies

**Walter Berns**  
Resident Scholar

**Douglas J. Besharov**  
Resident Scholar

**Robert H. Bork**  
John M. Olin Scholar in Legal  
Studies

**Karlyn Bowman**  
Resident Fellow

**Ricardo Caballero**  
Visiting Scholar

**John E. Calfee**  
Resident Scholar

**Charles Calomiris**  
Visiting Scholar

**Lynne V. Cheney**  
Senior Fellow

**Dinesh D'Souza**  
John M. Olin Research Fellow

**Nicholas N. Eberstadt**  
Visiting Scholar

**Mark Falcoff**  
Resident Scholar

**Gerald R. Ford**  
Distinguished Fellow

**Murray F. Foss**  
Visiting Scholar

**Hillel Fradkin**  
Resident Fellow

**Diana Furchtgott-Roth**  
Assistant to the President and  
Resident Fellow

**Suzanne Garment**  
Visiting Scholar

**Jeffrey Gedmin**  
Resident Scholar; Executive Director,  
New Atlantic Initiative

**Newton Gingrich**  
Senior Fellow

**James K. Glassman**  
DeWitt Wallace-Reader's Digest  
Fellow

**Robert A. Goldwin**  
Resident Scholar

**Robert W. Hahn**  
Resident Scholar; Director,  
AEI-Brookings Joint Center for  
Regulatory Studies

**Kevin Hassett**  
Resident Scholar

**Tom Hazlett**  
Resident Scholar

**Robert B. Helms**  
Resident Scholar; Director, Health  
Policy Studies

**R. Glenn Hubbard**  
Visiting Scholar

**James D. Johnston**  
Resident Fellow

**Leon Kass**  
W. H. Brady, Jr., Scholar

**Jeane J. Kirkpatrick**  
Senior Fellow; Director, Foreign and  
Defense Policy Studies

**Marvin H. Kosters**  
Resident Scholar; Director, Economic  
Policy Studies

**Irving Kristol**  
John M. Olin Distinguished Fellow

**Michael A. Ledeen**  
Freedom Scholar

**James Lilley**  
Resident Fellow

**Lawrence Lindsey**  
Arthur F. Burns Scholar in  
Economics

**Clarisa Long**  
Abramson Fellow

**Randall Lutter**  
Resident Scholar

**John H. Makin**  
Visiting Scholar; Director, Fiscal  
Policy Studies

**Allan H. Meltzer**  
Visiting Scholar

**James M. Morris**  
Director of Publications

**Joshua Muravchik**  
Resident Scholar

**Charles Murray**  
Bradley Fellow

**Michael Novak**  
George F. Jewett Scholar in Religion,  
Philosophy, and Public Policy;  
Director, Social and Political Studies

**Norman J. Ornstein**  
Resident Scholar

**Richard N. Perle**  
Resident Fellow

**Sarath Rajapatirana**  
Visiting Scholar

**William Schneider**  
Resident Scholar

**J. Gregory Sidak**  
F. K. Weyerhaeuser Fellow

**Christina Hoff Sommers**  
W. H. Brady, Jr., Fellow

**Herbert Stein**  
Senior Fellow

**Daniel Troy**  
Associate Scholar

**Arthur Waldron**  
Visiting Scholar; Director, Asian  
Studies

**Graham Walker**  
Visiting Scholar

**Peter Wallison**  
Resident Fellow

**Ben J. Wattenberg**  
Senior Fellow

**Carolyn L. Weaver**  
Resident Scholar; Director, Social  
Security and Pension Studies

**David Wurmser**  
Research Fellow

**Karl Zinsmeister**  
J. B. Fuqua Fellow; Editor,  
*The American Enterprise*