By Jason D. Buxbaum, Michael E. Chernew, A. Mark Fendrick, and David M. Cutler

DOI: 10.1377/hlthaff.2020.00284
HEALTH AFFAIRS 39, NO. 9 (2020): 1546–1556

Contributions Of Public Health, Pharmaceuticals, And Other Medical Care To US Life Expectancy Changes, 1990-2015

Jason D. Buxbaum (jasonbuxbaum@g.harvard.edu) is a student in the Program in Health Policy at Harvard University, in Cambridge, Massachusetts.

Michael E. Chernew is the Leonard D. Schaeffer Professor of Health Care Policy and director of the Healthcare Markets and Regulation (HMR) Lab in the Department of Health Care Policy, Harvard Medical School, in Boston, Massachusetts.

A. Mark Fendrick is a professor in the Department of Internal Medicine and director of the Center for Value-Based Insurance Design at the University of Michigan, in Ann Arbor, Michigan.

David M. Cutler is the Otto Eckstein Professor of Applied Economics in the Department of Economics at Harvard University and a research associate at the National Bureau of Economic Research, in Cambridge, Massachusetts.

ABSTRACT **Life expectancy in the US increased 3.3 years between 1990 and 2015, but the drivers of this increase are not well understood. We used vital statistics data and cause-deletion analysis to identify the conditions most responsible for changing life expectancy and quantified how public health, pharmaceuticals, other (nonpharmaceutical) medical care, and other/unknown factors contributed to the improvement. We found that twelve conditions most responsible for changing life expectancy explained 2.9 years of net improvement (85 percent of the total). Ischemic heart disease was the largest positive contributor to life expectancy, and accidental poisoning or drug overdose was the largest negative contributor. Forty-four percent of improved life expectancy was attributable to public health, 35 percent was attributable to pharmaceuticals, 13 percent was attributable to other medical care, and −7 percent was attributable to other/unknown factors. Our findings emphasize the crucial role of public health advances, as well as pharmaceutical innovation, in explaining improving life expectancy.**

Growth in medical spending consistently outpaces overall economic growth in the United States, prompting questions about the extent to which health care expenditures deliver value to justify their cost. If medical advances have contributed substantially to improved survival (life expectancy increased 3.3 years between 1990 and 2015), then growing investment in medical spending might be more palatable. The issue is particularly acute for pharmaceuticals, whose spending growth averaged 2.3 percentage points above growth in the rest of the health sector between 1990 and 2015.[1]

A recent synthesis estimated that a lack of modern medical care is directly responsible for 5–15 percent of premature mortality, with most premature mortality attributable to health-related behavior and social circumstances.[2] However, the impact of medical advances in explaining improving life expectancy over time has received somewhat less attention. One study estimated that half of all health improvements between 1960 and 2000 are due to medical care, although that estimation was extrapolated from a small number of conditions.[3] The difficulty is in part because medical care is difficult to evaluate as a whole. The determinants of health may shift within and across conditions over time, and many once-accepted scientific consensuses are later modified.

Even when one looks at a particular type of care, such as pharmaceuticals, the evidence of health impact is mixed. One recent study estimated that the expansion of cardiovascular medications led to a large reduction in heart disease mortality.[4] At the same time, evidence on the cost-effectiveness of anticancer agents varies greatly.[5,6] Further, excessive use of opioids has led to tens of thousands of deaths annually, contributing to flat or declining life expectancy between 2015 and 2017.

Given the salience of health care value in policy discussions, we sought to quantify the importance of medical care in total, and pharmaceutical treatments specifically, for recent changes in US life expectancy. We focused on 1990–2015 because some data sources used were unavailable for more recent years.

We began with vital statistics data to apportion improvements in mortality to various causes. For each of twelve causes responsible for life expectancy changes of 0.1 years or more, we reviewed the literature on the factors explaining mortality changes. We apportioned survival improvements into public health, pharmaceuticals, other (nonpharmaceutical) medical care, and a residual category comprising other or unknown factors. Aggregated results provide insight into the key drivers of increasing life expectancy between 1990 and 2015.

Study Data And Methods

CALCULATING CHANGES IN LIFE EXPECTANCY

▶ **DATA SOURCE:** Data on mortality by age and cause were obtained from the Centers for Disease Control and Prevention Wide-ranging Online Data for Epidemiologic Research (CDC WONDER) resource for 1990 through 2015. We identified causes using a hybrid of the National Center for Health Statistics' 113-cause-of-death and 39-cause-of-death classification systems. Details on our approach are in online appendix exhibits A1–A3.[7]

Although the 39-cause list is common in analyzing mortality, the level of aggregation is problematic with respect to some key conditions. For example, all accidents not caused by motor vehicles are grouped together. Accordingly, we conducted the analysis described here on both the 39- and 113-cause lists. We used the 39-cause list as our starting point, but we turned to the 113-cause list to avoid obscuring meaningful differences in trends among causes of death in overly broad categories, reduce the size of the "all other deaths" category, and group infant deaths together. Appendix exhibit A2 lists relevant *International Statistical Classification of Diseases and Related Health Problems*, Tenth Revision (ICD-10), codes.[7]

Data were available in *International Classification of Diseases*, Ninth Revision (ICD-9), for 1990–98 and ICD-10 for 1999–2015. ICD-9-to-ICD-10 comparability ratios were used to adjust pre-1999 death rates to form a consistent series and minimize potential bias associated with changes in coding practices (appendix exhibit A3).[7]

▶ **ESTIMATION:** Cause-deletion methods were employed to estimate the relative contribution of each condition to overall improvement in life expectancy at birth. This entailed starting with 1990 mortality rates and estimating sequentially the impact of changes in mortality for each condition on life expectancy at birth, holding other conditions at their 1990 mortality rates. The difference between life expectancy with and without the change for each condition is the life expectancy gain or loss attributable to that cause. For each cause studied in detail, we also examined mortality change in successive five-year intervals.

EXPLAINING LIFE EXPECTANCY CHANGES We sought to apportion mortality changes for each cause of death associated with an increase or decrease in life expectancy of 0.1 years or more into the change attributable to public health, pharmaceuticals, and other medical care. We used multiple approaches because the most preferred method was not always possible (appendix exhibit A1, flowchart B).[7]

Whenever available, statistical models appearing in the peer-reviewed literature were used to explain changing mortality. Literature reviews were conducted to identify models using the union of the following Medical Subject Headings (MeSH) terms: "United States/epidemiology," "mortality/trends," and the condition name. On finding a relevant article, we consulted citations (a technique known as "snowballing") and used reverse citation look-up to identify related studies. We contacted subject-matter experts for guidance when we were unable to identify suitable sources through literature reviews. Some models were identified that did not cover the full period of interest. In these instances, we updated the model if the condition represented a large share of life expectancy change. Otherwise, we used the results available.

In some instances, we were unable to identify suitable existing condition-specific models. In those cases, we relied on alternative approaches to apportion mortality change among factors. These included the creation of new models, published surveys of physician opinion, and literature-informed judgements as to plausibility. Appendix exhibit A1 (flowchart C) depicts our approach in these cases.[7]

Public health was broadly defined as reductions in identifiable risk factors for injury or disease not classified in the three following categories: pharmaceuticals, which included opioids, biologics, and oncology agents among other agents; other (nonpharmaceutical) medical care, which included physician/hospital services such as cancer screenings, diagnostic testing, radiotherapy, and surgery; and a residual category for other/unknown factors. There may be overlaps among factors. For example, public health ef-

forts could lead to more cancer screenings, which could reduce mortality. Our primary analysis looked at the most proximal cause, so benefits would be attributed to medical care, not public health, in the previous example. We performed a sensitivity analysis to vary characterizations in areas with the greatest overlap of potential characterizations: overdoses, cancer screenings, and pregnancy terminations.

LIMITATIONS This approach had limitations. First, the attribution of responsibility for improved survival to public health, pharmaceuticals, and other medical care was constrained by the availability of literature and the need to impose subjective distinctions. It was also limited in that current knowledge may change over time. Second, the use of cause deletion required assuming that competing causes of death are independent from one another. This implies that the impact from any given cause may be misstated if the cause is strongly correlated with other causes. Third, life expectancy trends vary over time by race, ethnicity, education, geography, and other key dimensions, whereas this analysis is limited to overall trends. Fourth, coding changes over time, including the switch from ICD-9 to ICD-10, could affect findings (appendix exhibit A3).[7] Fifth, we examined life expectancy at birth. Results could differ with related metrics, such as average populationwide mortality rates. Sixth, morbidity changes may be equally important but were not included in our primary analysis. Seventh, the time period of the study predated the coronavirus disease 2019 (COVID-19) pandemic. Accordingly, COVID-19 was not analyzed as a unique cause of death.

Study Results

AGGREGATE CHANGES IN LIFE EXPECTANCY The cause-deletion methodology explained 3.3 years of improvement in life expectancy at birth for the modified list of 113 causes of death (appendix exhibit A4).[7] This corresponds with the observed change in life expectancy between 1990 and 2015. Exhibit 1 shows the contribution of each cause of death to changing life expectancy for the modified list of thirty-nine causes of death. Ischemic heart disease (1.76 years), lung cancer (0.34 years), and stroke (0.33 years) accounted for the greatest shares of improvement. Accidental poisoning or overdose (−0.32 years), dementia excluding Alzheimer disease (−0.19 years), and Alzheimer disease (−0.14 years) accounted for the greatest decrements in life expectancy.

Exhibit 2 shows causes of death that accounted for an increase or decrease in life expectancy of at least 0.1 years. Diseases of the circulatory system, cancers, and trauma (excluding suicide) accounted for 62 percent, 18 percent, and 9 percent of life expectancy improvement, respectively.

Public health improvements accounted for the largest part of mortality improvement overall.

Changes in life expectancy were generally continuous across five-year intervals (appendix exhibit A5).[7] Exceptions included ischemic heart disease, for which life expectancy increases slowed after 2005, and HIV, for which gains were concentrated in the 1995–2000 period. The negative impact of accidental poisoning or overdose on life expectancy also increased over time.

EXPLANATIONS FOR CHANGES IN LIFE EXPECTANCY Exhibit 3 attributes life expectancy increases to the impact of public health, pharmaceuticals, other medical care, and other/unexplained factors.

▶ **ISCHEMIC HEART DISEASE:** IMPACT is a validated statistical model that has been used in more than twenty countries to explain changes in death from heart disease over time.[3,8] We updated a 2019 version of the IMPACT model with 1990–2015 US-specific data to explain reductions in ischemic heart disease mortality.[8] Appendix exhibit A6 contains additional details.[7] The IMPACT model estimated that 52 percent of the decrease in mortality was attributable to pharmaceuticals and 7 percent was attributable to other medical care (exhibit 3).

The most important pharmaceutical treatments were care for hypertension and high cholesterol and, to a lesser extent, medications for secondary prevention after myocardial infarction and angina. With respect to other (nonpharmaceutical) medical care, rehabilitation and revascularization were of approximately comparable importance. Another 39 percent of mortality decline was attributed to improved public health not resulting from medications, principally reduced cholesterol and blood pressure. Although smoking decreased, benefits were partially offset by increases in body mass index and diabetes; we characterized these changes as public health. Approximately 2 percent of

EXHIBIT 1

Changes to US life expectancy, by cause of death, 1990–2015

SOURCE Authors' analysis of data from the National Vital Statistics System. **NOTES** Causes of death other than infant mortality exclude deaths for people younger than age one. See online appendix exhibits A1–A4 for further detail (see note 7 in text).

mortality improvement was not explained by the IMPACT model.

▶ **CEREBROVASCULAR DISEASE:** We updated a previously published cerebrovascular disease–specific extension of the IMPACT model to determine the causes of reduced cerebrovascular disease (appendix exhibit A7).[4,7] Sixty percent of reduced mortality was attributable to pharmaceuticals (including antihypertensives, statins, and anticoagulants), and 8 percent of reduced mortality was attributable to other medical care (carotid endarterectomies and rehabilitation). The remaining 32 percent of mortality reduction was attributed to unmodeled public health improvements, such as reduced smoking and hypertension not achieved through medical treatment.

▶ **LUNG, BRONCHUS, AND TRACHEA CANCER:** We did not identify any suitable existing models specific to lung, bronchus, and trachea cancer. We therefore used Surveillance, Epidemiology, and End Results Program data to create a new model (appendix exhibit A8).[7] Age-adjusted lung or bronchus cancer incidence fell 25 percent between 1990 and 2015, whereas mortality decreased 31 percent. Thus, decreasing incidence accounted for 81 percent of the decrease in mortality. Because smoking is the primary risk factor for lung cancer, we attributed 81 percent of reduced mortality to public health.

For these cancers, average five-year survival after diagnosis improved from 13 percent to 18 percent during 1990–2015. Stage migration has likely had only small impacts in lung cancer; screening was recommended only at the end of our study period, and symptomatic lung cancers

EXHIBIT 2

Top contributors to US life expectancy changes, by category, 1990–2015

Categories/causes of death	Years of life expectancy gained or lost	Contribution to overall life expectancy change (%)
Circulatory system		
Ischemic heart disease	1.76	53
Cerebrovascular diseases	0.33	10
Malignant neoplasms		
Malignant neoplasm of trachea, bronchus, lung	0.34	10
Malignant neoplasm of breast	0.13	4
Malignant neoplasm of colon, rectum, anus	0.12	4
Trauma		
Motor vehicle accident	0.19	6
Assault (homicide)	0.11	3
Neurological		
Alzheimer disease	−0.14	−4
Dementia, excluding Alzheimer disease	−0.19	−6
Other		
Infant mortality	0.28	8
HIV/AIDS	0.24	7
Accidental poisoning or overdose	−0.32	−9
Total	2.86	85

SOURCE Authors' analysis of data from the National Vital Statistics System. **NOTES** Limited to causes corresponding with increases or decreases of more than 0.1 years. See online appendix exhibits A1–A5 for further detail (see note 7 in text). Figures may differ from totals in text and table because of rounding.

generally grow rapidly. Further, there was little change in stage distribution of diagnosis; such changes accounted for only 4 percent of mortality reduction. We characterized this improvement as other/unknown.

Meta-analyses suggest a reduction in mortality risk of 13 percent in association with chemotherapy for treatment of non-small-cell lung cancer (roughly 80–90 percent of lung cancers). The share of patients with lung, bronchus, and trachea cancer receiving chemotherapy increased from 28 percent of known patients in 1990 to 40 percent in 2015. Even without accounting for advances in therapeutic effectiveness, increasing chemotherapy use accounted for 5 percent of reduced mortality. We attributed the residual mortality improvement (11 percent) to other nonpharmaceutical medical advances, such as improvements in surgery and radiotherapy.

▶ **BREAST CANCER:** We identified a 2018 Cancer Intervention and Surveillance Network article attributing reduced breast cancer mortality to advances in treatment and screening.[9] The article described findings from six models analyzing contributors to trends in breast cancer incidence and avoided mortality between 1975 and 2012. Averaging across models, 60 percent of improvement was attributable to medications, 31 percent of improvement was attributable to screenings, and 9 percent of improvement was unexplained (see appendix exhibit A9).[7]

▶ **COLORECTAL CANCER:** The Microsimulation Screening Analysis Cancer Intervention and Surveillance Network Colorectal Cancer Model has been used to estimate the source of longevity gains for colorectal cancer.[10,11] The most recent Microsimulation Screening Analysis results are from the period 1990–2000. The models were then used to forecast to 2015 under various scenarios. The "optimistic" scenario matched well with observed trends for the most important contributors to colorectal cancer incidence and mortality (appendix exhibit A10).[7] In this scenario, 27 percent of improved survival was attributable to pharmaceuticals (chemotherapy), 42 percent was attributable to medical care (cancer screening), and 31 percent was attributable to public health (especially decreased smoking).[10,11]

▶ **MOTOR VEHICLE ACCIDENTS:** Two studies in the traffic safety literature can be used to estimate the impact of medical care on motor vehicle accident fatalities. One study modeled traffic fatalities per 100,000 people at the state-year level as a function of automobile characteristics, road characteristics, and the White infant mortality rate for the state-year. The latter served as a proxy for the impact of medical technology.[12] We used this model to estimate the percentage of the time series change in motor vehicle accident fatalities attributable to medical advances (appendix exhibit A11).[7] The estimate suggests that improved medical care accounted for 10 percent of the reduction in motor vehicle traffic fatalities. We attributed the residual 90 percent to public health, such as improvements in vehicle safety.

A second model relates mortality for people in motor vehicle accidents to the receipt of high-level trauma care.[13] The study showed that the relative risk for death was 0.71 for people transported to a Level I or II trauma center compared with people transported to other locations. Using data on the share of people taken to Level I or II trauma centers over time implies that 10 percent of reduced motor vehicle accident fatalities were due to greater access to Level I or II trauma centers (appendix exhibit A11).[7] The alignment of results across models provided additional support for attributing 10 percent of reduced motor vehicle accident fatalities to other medical care and 90 percent to public health.

▶ **HOMICIDE:** We updated the approach used by Anthony Harris and colleagues in 2002 to estimate the contribution of medical care to reduced homicide.[14] These authors proposed that aggravated assaults be viewed as potential homicides where the outcome was not death. Change in the incidence of aggravated assault over time

EXHIBIT 3

Estimated impact of pharmaceuticals, other medical care, and public health on changes in US mortality, by cause of death, 1990–2015

Categories/ causes of death	Contribution to mortality reduction (%)	Contribution to mortality changes (%)				Comments
		Public health	Pharma-ceuticals	Other medical care	Other/ unexplained	
CIRCULATORY SYSTEM						
Ischemic heart disease	53	39	52	7	2	Most important pharmaceutical therapies: statins, antihypertensives Most important public health improvements: reductions in cholesterol, hypertension, and smoking
Cerebrovascular disease	10	32	60	8	—	Most important contributors: antihypertensives, statins, warfarin
MALIGNANT NEOPLASMS						
Malignant neoplasms of trachea, bronchus, lung	10	81	5	11	4	Reduced incidence of lung cancer used as proxy for public health factors
Malignant neoplasm of breast	4	—	60	31	9	Figures reflect 1990–2012 "Other medical care" reflects screening
Malignant neoplasm of colon, rectum, anus	4	31	27	42	—	Figures reflect experience (1990–2000) and projection (2000–15) "Other medical care" reflects screening
TRAUMA						
Motor vehicle accident	6	90	—	10	—	White infant mortality rate used as proxy for medical care Confirmatory findings from second model
Homicide	3	91	—	9	—	Aggravated assault rate used as proxy for nonmedical contributors to homicide
NEUROLOGICAL						
Alzheimer disease	–4	—	—	—	100	Possible changes in coding practices
Dementia, excluding Alzheimer disease	–6	—	—	—	100	Possible changes in coding practices
OTHER						
Infant mortality	8	39	21	20	20	See exhibit 4 and appendix exhibit A13[a]
HIV/AIDS	7	—	76	24	—	Figures reflect physician survey Confirmatory evidence from timing of HAART introduction
Accidental poisoning or overdose	–9	4	96	—	—	Includes deaths related to opioid crisis
TOTAL						
All causes	85	44	35	13	–7	

SOURCE Authors' analysis of data from the National Vital Statistics System and sources cited in text. **NOTES** Figures reflect 1990–2015 unless otherwise indicated. Figures may differ from totals in text and table because of rounding. HAART is highly active antiretroviral therapy. [a]See note 7 in text.

may thus be viewed as change in the risk for death from homicide not attributable to changes in the deadliness of the average aggravated assault, reporting patterns, or medical care.

According to federal Uniform Crime Reporting statistics, aggravated assaults fell by 44 percent between 1990 and 2015. During the same period, the homicide rate fell by 48 percent. The additional reduction in homicide deaths of 4 percentage points is 9 percent of the decline in homicides. Thus, we attributed 9 percent of reduced homicide mortality to medical care and the remaining 91 percent to public health. As with motor vehicle accidents, the medical care component was mostly nonpharmaceutical; we recorded it accordingly (exhibit 3).

▶ **ALZHEIMER DISEASE AND DEMENTIAS:** The reasons for underlying apparent increases in recorded mortality from Alzheimer disease and other dementias are unclear, as mortality trends were age adjusted. There are no established modifiable risk factors for Alzheimer disease, and the overall trend across known risk factors for non-Alzheimer dementia did not clearly worsen during 1990–2015 (appendix exhibit A12).[7]

There is generally "a blurred distinction between death with dementia and death from dementia."[15] Vital records–based estimates of

changes in dementia mortality may be particularly influenced by changes in disease awareness and place of death that took place between 1990 and 2015.[15] Given the absence of clear medical or public health explanations for the increases in dementia-related mortality, as well as the plausibility of changes in coding practices as the key explanatory factor, we attributed mortality increases to other/unexplained.

▶ **INFANT MORTALITY:** No comprehensive models to explain overall changes in infant mortality were identified. We therefore investigated the factors contributing to improved survival for the five conditions responsible for the greatest reductions in infant mortality between 1990 and 2015 (90 percent of the total; exhibit 4). Appendix exhibit A13 contains details.[7]

Reductions in sudden infant death syndrome accounted for 39 percent of all infant mortality gains. Sudden infant death syndrome–related improvements were attributed to advances in public health, particularly campaigns to prevent stomach sleeping in the 1990s. Reduced mortality from respiratory distress syndrome accounted for 23 percent of gains (exhibit 4). According to data from two neonatal hospital quality improvement collaboratives (appendix exhibits A13.1–A13.3),[7] the use of surfactant and antenatal steroids, both of which markedly reduce the risk for death among low-birthweight babies, increased sharply in the 1990s. We estimated that increasing use of these pharmaceuticals explained 89 percent of the observed respiratory distress syndrome mortality reduction. The large increase in life expectancy resulting from infant mortality reductions in the 1990–94 period is temporally consistent with advances associated with sudden infant death syndrome and respiratory distress syndrome (appendix exhibit A5, panel D).[7]

Fourteen percent of reduced infant mortality was a result of decreases in fatal congenital anomalies of the heart, most likely driven by nonpharmaceutical medical innovations. Eight percent of reduced infant mortality was attributed to reduced death from chromosomal anomalies. We attributed these reductions to increases in selective terminations and categorized them as other/unknown. Finally, 6 percent of reduced infant mortality was a result of reductions in lung-related congenital anomalies. There were important advances in surgery and diagnostic technique over our period of interest, particularly for diaphragmatic hernia repair, which we characterized as other medical care. Appendix exhibit 13.1 provides details and supporting references.[7]

Aggregating, we estimated that 39 percent of reduced infant mortality was attributable to public health, 21 percent to pharmaceuticals, and 20 percent to other medical care (exhibit 4).

▶ **HIV:** The literature review did not identify a suitable model to explain HIV mortality improvement. We therefore relied primarily on expert

EXHIBIT 4

Estimated impact of public health, pharmaceuticals, and other medical care on changes in US infant mortality, by cause of death, 1990–2015

		Contribution to mortality changes (%)				
Causes of death	Contribution to mortality reduction (%)	Public health	Pharmaceuticals	Other medical care	Other/ unexplained	Comments
Sudden infant death syndrome	39	100	—	—	—	Spread of safe sleep practices Reductions in cigarette smoking among pregnant women
Respiratory distress syndrome	23	—	89	—	11	Increased use of surfactant and antenatal steroids in preterm births
Congenital anomalies of the heart	14	—	—	100	—	Interventional cardiac procedures and associated technology (for example, miniaturization of tools, increases in NICU access) Improved prenatal diagnosis
Chromosomal anomalies	8	—	—	—	100	Edwards and Patau syndromes are in this category Increases in prenatal screening and selective terminations
Congenital anomalies of the lung	6	—	—	100	—	Increasing use of diaphragmatic hernia repair
Other	10	—	—	—	100	Not investigated
Total		39	21	20	20	

SOURCE Authors' analysis of sources in online appendix exhibit A13 (see note 7 in text).

Our results suggest the importance of minimizing cost-related barriers to key preventive and chronic care services.

opinion, as reported in a survey of sixteen physicians specializing in the treatment of HIV.[16] Surveyed physicians estimated that pharmaceuticals accounted for 76 percent of reduced HIV morbidity and mortality between 1990 and 2015. Nonpharmaceutical medical technologies, such as diagnostic testing, accounted for nearly all the remainder. Responding physicians reported that other factors, including public health, accounted for only 0.3 percent of improvement.

Observed trends support these estimates. Seventy-one percent of the reduction in HIV-related deaths between the peak of the HIV/AIDS epidemic in 1994–95 and 2015 took place in 1996–97 (appendix exhibit A14), when highly active antiretroviral therapy first became widely available.[7,17] Similarly, Frank J. Palella Jr. and colleagues showed that protease inhibitors (an essential element of highly active antiretroviral therapy) reduced mortality by 70 percent for people with low CD4+ cell counts.[17]

The low estimate for net benefit from public health also appears reasonable. Male-to-male sexual contact is the most common mechanism of HIV transmission in the US. Risky sexual practices among men who have sex with men increased between 1992 and 2013.[18] Therefore, although transmissions might have increased faster but for public health efforts, it is unlikely that public health efforts contributed to improvements between 1990 and 2015.

▶ **ACCIDENTAL POISONING OR OVERDOSE:** We used vital statistics data to understand changes in the factors contributing to fatal accidental poisoning or overdose. Ninety-six percent of the increase in poisoning or overdose was attributable to increases in fatal prescription and nonprescription drug use, particularly opioids; the remaining component was attributable to other sources such as alcohol consumption (data not shown). The prescription drug component of opioid deaths is most readily characterized as pharmaceutical. The nonprescription component is somewhat more difficult, as it reflects the use of heroin and fentanyl. Evidence suggests that many people transitioned into these substances after prescription opioids were made more difficult to obtain.[19,20] Thus, we attributed these deaths to pharmaceuticals as well, even if some of the "technology" was related to the ability to supply illegal drugs.

SENSITIVITY ANALYSIS In the sensitivity analysis, we varied the characterization of three contributors to changing mortality: overdoses, cancer screenings, and selective pregnancy terminations for genetic anomalies (appendix exhibit A15).[7] Our overall findings were sensitive to reallocating opioid-related mortality from pharmaceuticals to public health (for example, lack of sufficient Food and Drug Administration and Drug Enforcement Administration oversight). In this alternative scenario, 35 percent of gains were due to public health gains and 44 percent of gains were due to pharmaceutical gains.

Discussion

We studied contributors to life expectancy changes for twelve conditions accounting for 2.9 years of improved life expectancy in the US between 1990 and 2015 (exhibit 2). We found great variation in the key drivers of mortality change across causes. In our primary analysis, 44 percent of improvement was attributable to public health, 13 percent was attributable to nonpharmaceutical medical care, and 35 percent was attributable to pharmaceuticals (exhibit 3). The share of survival deterioration attributed to pharmaceuticals (–9 percent) was outweighed by the share of improvement attributed to pharmaceuticals (44 percent) (based on authors' calculations of unrounded data from exhibit 3). There was also a residual of –7 percent attributable to other/unexplained factors.

In addition to heterogeneity in the drivers of mortality avoidance across conditions, there was heterogeneity in the extent to which improvements were attributable to new technologies versus greater diffusion of existing technologies. For example, the 1990s saw the introduction of highly active antiretroviral therapy and the widespread diffusion of statins, which were crucial to mortality reduction for HIV and ischemic heart disease, respectively. There were also increases in the use of surfactant and antenatal steroids, technologies that reduced infant mortality. However, reductions in life expectancy attributable to opioids underscore the potential for severe harm when technologies diffuse beyond appropriate populations.

We found additional heterogeneity in the pace of mortality reduction over time and across con-

ditions. Paralleling the work of Anne Case and Angus Deaton,[21] we observed a sharp slowdown in 2010–15 in improvement in ischemic heart disease mortality (appendix exhibit A5).[7] This change contrasts with trends for other causes of death, as well as longitudinal trends in heart disease–specific mortality in peer countries.[21] Explaining this slowdown requires further research.

We identified 3 percent of life expectancy gains as resulting from increases in breast and colorectal cancer screening (exhibit 3). The benefits of these screenings have been debated. For example, H. Gilbert Welch and colleagues argue that mammography and colorectal cancer screening have much smaller, if any, effects on overall mortality than on disease-specific mortality.[22] However, the most current reviews suggest reduced overall mortality in conjunction with these two types of screening, although estimates are not statistically significant.[23,24] In addition, the most recent screening modalities have not been fully evaluated (for example, colonoscopy instead of sigmoidoscopy).

Apart from screening, our models found that key gains in other (nonpharmaceutical) medical care included surgical care for adults with ischemic heart disease and stroke and for babies with congenital anomalies. Other nonpharmaceutical interventions were important for people with lung cancer.

Public health improvements accounted for the largest part of mortality improvement overall (44 percent), outranking any other driver analyzed (exhibit 3). This extends previous work suggesting that public health is the dominant determinant of longevity in general.[2] In our work, public health improvements were driven by increased adoption of risk reduction practices known before the 1990s, such as smoking reduction and seatbelt usage, as well as by important "low-tech" breakthroughs, such as awareness of the danger of stomach sleep for infants. Improved traffic safety was also a big contributor to improved health.

Our emphasis on public health and pharmaceuticals as the key drivers of reduced mortality is specific to 1990–2015. Had we studied the 1980s, other (nonpharmaceutical) medical care likely would have been assigned greater responsibility for improved outcomes, given the 250 percent growth in cardiovascular procedures over that decade[25] and the fact that statins only became available in the late 1980s. Had we studied only the years after 2010, our estimate of the net benefit of pharmaceuticals would have been reduced, given the acceleration of the opioid epidemic over this period. Even an analysis of the drivers of life expectancy change during 1990–2015 conducted some years from now might yield revised estimates as knowledge of the impacts of various interventions changes.

If we could translate knowledge from existing public health "wins" to areas with less success, longevity gains could be very large.

Our work sought to explain the reasons for mortality reduction and did not consider differing effects by race, ethnicity, geography, and education. Future work should address this important limitation. Future work should also consider the drivers of morbidity reduction. Global Burden of Disease Study data suggest that disability and mortality moved in tandem during 1990–2015 (appendix exhibit A16).[7] However, the extent to which the drivers of mortality reduction are also drivers of morbidity reduction is unclear, as we did not examine many of the conditions most responsible for reduced disability between 1990 and 2015 (including mental illness and visual impairment).

Policy Implications

Our findings have implications for the ongoing debate regarding the value of health care spending in general, and spending on pharmaceuticals specifically. Although our findings do not speak directly to the value of treating additional people with medications, they do underscore the central role of medications overall in explaining reduced mortality. Policy making on drug pricing should consider the implications of potential legislation across the full spectrum of conditions—both those where the societal return on drug investments is high and those where expected value is more ambiguous.

Our results also suggest the importance of minimizing cost-related barriers to key preventive and chronic care services. For example, coverage expansions through the Affordable Care Act have been associated with increases in early-stage cancer diagnoses[26] as well as increased use of heart disease medication.[27] For the in-

sured, elimination of cost sharing for primary prevention may have led to increases in cancer screenings.[26] Given substantial underuse of high-value care, longevity gains might have been larger if coverage gains had been more complete and if out-of-pocket spending for secondary preventive services had also been reduced. Expanding coverage and advancing value-based insurance design[28] therefore remain important needs.

Conclusion

Our results emphasize the need to build on existing public health successes. In recent decades, smoking has become much less prevalent and driving has become safer. Simultaneously, obesity has increased and opioid-related mortality has soared. It is not clear what most explains this mixed record: the intrinsic nature of the behaviors or the lack of appropriate interventions. If we could translate knowledge from existing public health "wins" to areas with less success, longevity gains could be very large. ■

All authors report grant support from Pharmaceutical Research and Manufacturers of America. Jason Buxbaum reports research support from the Agency for Healthcare Research and Quality (Grant No. T32-HS000055). Buxbaum has received consulting income from the University of Michigan. Michael Chernew is chair of the Medicare Payment Advisory Commission, in Washington, D.C. He has received consulting income from Madelena Consulting. He has received speaking fees from American College of Cardiology, American Medical Association, America's Health Insurance Plans, BCBS of Florida, HealthEdge, Humana, MedAxiom and Washington University. He sits on the Advisory Board for Archway Health, MITRE, Virta Health, and National Institute for Health Care Management. He provides editorial services for Elsevier Inc. and MJH Life Sciences. He has research grants from Arnold Ventures, Agency for Healthcare Research and Quality, Centers for Medicare and Medicaid Services, National Institutes of Health, Blue Cross Blue Shield Association, Health Care Service Corporation, Ballad Health, Peterson Center on HealthCare, Robert Wood Johnson Foundation, and Commonwealth Fund. He reports having equity in Archway Health and V-BID Health. A. Mark Fendrick has received consulting income from AbbVie, Amgen, Centivo, Community Oncology Association, Covered California, EmblemHealth, Exact Sciences, Freedman Health, GRAIL, Harvard University, Health & Wellness Innovations, Health at Scale Technologies, MedZed, Penguin Pay, Risalto, Sempre Health, State of Minnesota, Department of Defense, Virginia Center for Health Innovation, Wellth, and Zansors. He has received research support from the Agency for Healthcare Research and Quality, Arnold Ventures, Gary and Mary West Health Policy Center, National Pharmaceutical Council, Patient-Centered Outcomes Research Institute, Robert Wood Johnson Foundation, State of Michigan, and Centers for Medicare and Medicaid Services. He reports having equity in V-BID Health. David Cutler has received consulting income from the American Economic Association, Colorado Center for Nursing Excellence, *Journal of the American Medical Association*, and the Scientific Advisory Board of F-Prime Capital Partners. He has been retained by counsel for plaintiffs to provide expert services in pending litigation involving opioid pharmaceuticals. The content is solely the responsibility of the authors and does not necessarily represent the official views of the Agency for Healthcare Research and Quality or the Medicare Payment Advisory Commission. The authors gratefully acknowledge the assistance of Kaushik Ghosh and Ken Langa.

NOTES

1 Centers for Medicare and Medicaid Services. National health expenditure data: historical [Internet]. Baltimore (MD): CMS; 2019 Dec 19 [cited 2020 Jul 29]. Available from: https://www.cms.gov/Research-Statistics-Data-and-Systems/Statistics-Trends-and-Reports/NationalHealthExpendData/NationalHealthAccountsHistorical.html

2 Kaplan RM, Milstein A. Contributions of health care to longevity: a review of 4 estimation methods. Ann Fam Med. 2019;17(3):267–72.

3 Cutler DM, Rosen AB, Vijan S. The value of medical spending in the United States, 1960–2000. N Engl J Med. 2006;355(9):920–7.

4 Cutler DM, Ghosh K, Messer KL, Raghunathan TE, Stewart ST, Rosen AB. Explaining the slowdown in medical spending growth among the elderly, 1999–2012. Health Aff (Millwood). 2019;38(2):222–9.

5 Howard DH, Bach PB, Berndt ER, Conti RM. Pricing in the market for anticancer drugs. J Econ Perspect. 2015;29(1):139–62.

6 Howard DH, Chernew ME, Abdelgawad T, Smith GL, Sollano J, Grabowski DC. New anticancer drugs associated with large increases in costs and life expectancy. Health Aff (Millwood). 2016;35(9):1581–7.

7 To access the appendix, click on the Details tab of the article online.

8 Ogata S, Nishimura K, Guzman-Castillo M, Sumita Y, Nakai M, Nakao YM, et al. Explaining the decline in coronary heart disease mortality rates in Japan: contributions of changes in risk factors and evidence-based treatments between 1980 and 2012. Int J Cardiol. 2019;291:183–8.

9 Plevritis SK, Munoz D, Kurian AW, Stout NK, Alagoz O, Near AM, et al. Association of screening and treatment with breast cancer mortality by molecular subtype in US women, 2000–2012. JAMA. 2018;319(2):154–64.

10 Edwards BK, Ward E, Kohler BA, Eheman C, Zauber AG, Anderson RN, et al. Annual report to the nation on the status of cancer, 1975–2006, featuring colorectal cancer trends and impact of interventions (risk factors, screening, and treatment) to reduce future rates. Cancer. 2010;116(3):544–73.

11 Vogelaar I, van Ballegooijen M, Schrag D, Boer R, Winawer SJ, Habbema JD, et al. How much can current interventions reduce colorectal cancer mortality in the U.S.? Mortality projections for scenarios of risk-factor modification, screening, and treatment. Cancer. 2006;107(7):1624–33.

12 Noland RB, Zhou Y. Has the Great Recession and its aftermath reduced traffic fatalities? Accid Anal Prev. 2017;98:130–8.

13 Melton SM Jr, McGwin G, Abernathy JH 3rd, MacLennan P, Cross JM, Rue LW 3rd. Motor vehicle crash–related mortality is associated with prehospital and hospital-based resource availability. J Trauma. 2003;54(2):273–9, discussion 279.

14 Harris AR, Thomas SH, Fisher GA, Hirsch DJ. Murder and medicine: the lethality of criminal assault 1960–1999. Homicide Stud. 2002;6(2):128–66.

15 Ganguli M, Rodriguez EG. Reporting of dementia on death certificates: a community study. J Am Geriatr Soc. 1999;47(7):842–9.
16 Wamble DE, Ciarametaro M, Dubois R. The effect of medical technology innovations on patient outcomes, 1990–2015: results of a physician survey. J Manag Care Spec Pharm. 2019;25(1):66–71.
17 Palella FJ Jr, Delaney KM, Moorman AC, Loveless MO, Fuhrer J, Satten GA, et al. Declining morbidity and mortality among patients with advanced human immunodeficiency virus infection. N Engl J Med. 1998;338(13):853–60.
18 Hess KL, Crepaz N, Rose C, Purcell D, Paz-Bailey G. Trends in sexual behavior among men who have sex with men (MSM) in high-income countries, 1990–2013: a systematic review. AIDS Behav. 2017;21(10):2811–34.
19 Alpert A, Powell D, Pacula RL. Supply-side drug policy in the presence of substitutes: evidence from the introduction of abuse-deterrent opioids. Am Econ J Econ Policy. 2018;10(4):1–35.
20 Evans WN, Lieber EMJ, Power P. How the reformulation of OxyContin ignited the heroin epidemic. Rev Econ Stat. 2018;101(1):1–15.
21 Case A, Deaton A. Mortality and morbidity in the 21st century [Internet]. Washington (DC): Brookings Institution; 2017 Mar 23 [cited 2020 Jul 29]. Available from: https://www.brookings.edu/bpea-articles/mortality-and-morbidity-in-the-21st-century/
22 Welch HG, Schwartz L, Woloshin S. Overdiagnosed: making people sick in the pursuit of health. Boston (MA): Beacon Press; 2011.
23 Jodal HC, Helsingen LM, Anderson JC, Lytvyn L, Vandvik PO, Emilsson L. Colorectal cancer screening with faecal testing, sigmoidoscopy, or colonoscopy: a systematic review and network meta-analysis. BMJ Open. 2019;9(10):e032773.
24 Barbeau P, Stevens A, Beck A, Skidmore B, Arnaout A, Brackstone M, et al. Breast cancer screening: Part A. An evidence report to inform an update of the Canadian Task Force on Preventive Health Care 2011 guideline [Internet]. Ottawa: Ottawa Hospital Research Institute; 2017 [cited 2020 Jul 29]. Available from: https://canadiantaskforce.ca/wp-content/uploads/2019/02/Systematic-Review-Evidence-Report_v2_FINAL.pdff
25 Gillum BS, Graves EJ, Jean L. Trends in hospital utilization: United States, 1988–92. Vital Health Stat 13. 1996;(124):1–71. Table 19.
26 Zhao J, Mao Z, Fedewa SA, Nogueira L, Yabroff KR, Jemal A, et al. The Affordable Care Act and access to care across the cancer control continuum: a review at 10 years. CA Cancer J Clin. 2020;70(3):165–81.
27 Ghosh A, Simon K, Sommers BD. The effect of health insurance on prescription drug use among low-income adults: evidence from recent Medicaid expansions. J Health Econ. 2019;63:64–80.
28 Chernew ME, Rosen AB, Fendrick AM. Value-based insurance design. Health Aff (Millwood). 2007;26(2):w195–203. DOI: 10.1377/hlthaff.26.2.w195.