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\textbf{ABSTRACT}

For decades, the US public and private sectors have committed substantial resources towards cancer research, but the societal payoff has not been well-understood. We quantify the value of recent gains in cancer survival, and analyze the distribution of value among various stakeholders. Between 1988 and 2000, life expectancy for cancer patients increased by roughly four years, and the average willingness-to-pay for these survival gains was roughly $322,000. Improvements in cancer survival during this period created 23 million additional life-years and roughly $1.9 trillion of additional social value, implying that the average life-year was worth approximately $82,000 to its recipient. Health care providers and pharmaceutical companies appropriated 5–19\% of this total, with the rest accruing to patients. The share of value flowing to patients has been rising over time. In terms of economic rates of return, R&D investments against cancer have been a success, particularly from the patient’s point of view.

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\textsuperscript{1} See http://dtp.nci.nih.gov/timeline/noflash/milestones/M4_Nixon.htm.

\textsuperscript{2} Moreover, the number of deaths from cancer has been increasing, owing in large part to advances in technology that have reduced the probability of dying from heart disease (Honore and Lleras-Muney, 2006).

I will also ask for an appropriation of an extra $100 million to launch an intensive campaign to find a cure for cancer, and I will ask later for whatever additional funds can effectively be used. The time has come in America when the same kind of concentrated effort that split the atom and took man to the moon should be turned toward conquering this dreaded disease. Let us make a total national commitment to achieve this goal.

President Richard M. Nixon, January 1971 State of the Union Address

Eleven months later, President Nixon formalized the war on cancer with the passage of the National Cancer Act, an act designed to promote the discovery of new treatments for cancer and to encourage early detection and prevention of the disease. More than three decades later, cancer remains an important cause of morbidity and mortality in the United States. In 2003 alone, cancer accounted for nearly 25\%, or roughly 556,000, of all deaths, and represented the second-leading cause of mortality in the US.\textsuperscript{2} Today, nearly 1.5 million cases of cancer are diagnosed in the US annually.

While cancer continues to claim lives, the last three decades have witnessed tremendous strides in our understanding and treatment of the disease. Scientists have identified many of the genes responsible for cancer, discovered new chemotherapeutic and biologic approaches to treating the disease, and developed new imaging techniques to detect cancer earlier. Accompanying these scientific advances have been perhaps equally important public health campaigns which have led to improved screening and earlier detection of the disease.

Despite these advances, there is significant debate over whether the war on cancer is being “won.” Criticisms generally center on two closely related issues. The first is that there has been little progress in cancer survival, and that the progress that has been made is due to the development of expensive treatments that marginally prolong life (Epstein, 2005; Faguet, 2005). Indeed, a large body of work has questioned the cost-effectiveness of recent cancer treatments.

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(Berenson, 2005; Hillner and Smith, 2007; Kolata and Pollack, 2008; Shih and Halpern, 2008). This has influenced public opinion among physicians as well. For example, a recent survey of academic oncologists found that only 25% of those surveyed believe that recently developed therapies for metastatic colorectal and non-small cell lung cancers offer a good value (Nadler et al., 2006). Second, some argue that too much effort has been focused on developing such costly treatments, at the expense of prevention and early detection (Faguet, 2005). In response to both criticisms, proponents of the war on cancer argue that there have been substantial increases in cancer survival. For example, the five-year relative survival rate for all cancers diagnosed between 1996 and 2003 was 66%, up from 50% in the mid-1970s (American Cancer Society, 2008). Improvements in survival have been particularly dramatic for breast cancer, colon cancer, and non-Hodgkin’s lymphoma, all of which have seen significant advances in therapy, screening, or both (Breen et al., 2001; Espey et al., 2007; Pulte et al., 2008). Despite this controversy, there has been little effort to explicitly analyze the benefits and costs of the war on cancer. The sole exception we are aware of is Lichtenberg (2004), who estimates that newer cancer drugs developed between 1975 and 1995 increased the life expectancy of a cancer patient by one year and increased the cost of cancer care by $3,000 per patient.

To address this important question, our paper evaluates the costs and benefits of investments in R&D that began with the War on Cancer. We also evaluate the distribution of net social gains across patients and firms. Gains in cancer R&D will affect cancer survival through three mechanisms. First, R&D advances may prevent the disease altogether, through the development of vaccines, changes in behavior, and the development of screening tests to identify pre-malignant disease. Second, technological advances or efforts to increase screening may result in the detection of malignant disease at earlier stages. This may improve survival, since cancer is typically easier to treat at earlier stages. Finally, therapeutic advances may improve survival directly. For example, better treatment for late-stage cancer can improve survival even if detection remains unchanged.

In prior work (Sun et al., 2010), we calculated cancer survival gains between 1988 and 2000 and decomposed these into the shares accounted for by treatment and detection improvements, respectively. Overall, we found that cancer survival increased significantly—for instance, survival for all cancers combined increased by 3.9 years between 1988 and 2000, while survival for breast cancer and non-Hodgkin's lymphoma increased by 3.6 and 3.5 years, respectively. The majority of these gains were due to improvements in treatment, which accounted for roughly 80% of survival gains for both breast cancer alone and for all cancers combined, and for nearly all of the survival gains for non-Hodgkin’s lymphoma. In particular, we found that changes in the probability of early detection were much less quantitatively significant than improvements in survival at a given stage of the disease.

The earlier analysis focused on the relative contributions of detection and treatment to survival. In this study, we compare the social value of these survival gains to their cost, to gain some insight into the social welfare effects of the war on cancer. Moreover, there continues to be debate over how much producers and manufacturers earn, and whether this comes at the expense of consumer surplus for patients. Therefore, we also ask how these social gains (or costs) were shared between patients and producers. A program that generated gains in social surplus that flowed exclusively, or primarily, to producers, would be viewed quite differently by policymakers and the public, compared to one that generated gains for patients.

We quantify the costs and benefits of cancer R&D, focusing on the benefits of earlier detection of malignant disease and improved cancer therapies. We begin by calculating the value of improved cancer survival from 1988 to 2000. We calculate that the average newly diagnosed cancer patient, earning the average US income, would be willing to pay approximately $31,000 annually to retain year 2000 survival prospects, as compared with the prospects available in 1988. This is equal to roughly half of full income. The high rates of mortality associated with cancer magnify the value of even small absolute survival gains, since greater value is placed on a given gain in longevity, when an individual has a shorter life expectancy (Becker et al., 2007). For example, since the value of consumption and wealth are significantly lower after death, individuals may be willing to pay nearly their entire end-of-life wealth for as little as a few extra weeks of life. In fact, this is one explanation for the emergence of expensive biologic treatments for cancer, which increase life-expectancy quite modestly—in some cases, just weeks—and yet still face high demand (Goldman et al., 2010). In addition, our results also suggest that wealthier individuals would be willing to pay a larger share of their income for these higher survival prospects. In economic parlance, this implies that cancer treatment behaves like a “luxury good,” in the sense that expenditure shares rise with income.

Aggregating over patients, we estimate that these improvements in survival generated approximately 23 million additional life-years, and $1.9 trillion of value for patients. These numbers imply that cancer patients were willing to pay $833 billion over this time period, while the net surplus to patients was approximately $1.9 trillion.

We are deliberately conservative in our approach to estimating the rate of return earned by the war on cancer. First, we do not incorporate the value generated by cancer prevention, and count only the benefits that accrue from 1988 to 2000, well after R&D initiated in the early 1970s would come to fruition. Against these benefits, we counted the total cost of (and profits from) cancer care provided from 1988 to 2000, and all cancer R&D spending from 1970 to 2000. Using these inputs, we calculate that cancer care providers (drug companies, hospitals, doctors, and health professionals) earned at most $393 billion in profits over this time period, while the net surplus to patients was approximately $1.9 trillion.

We are deliberately conservative in our approach to estimating the rate of return earned by the war on cancer. First, we do not incorporate the value generated by cancer prevention, and count only the benefits that accrue from individuals who acquire cancer. Second, while we count all cancer R&D expenditures from 1970 onwards as the total size of investment, we count net benefits (gains in survival, less medical costs) from 1988 to 2000 only. We restrict our analysis partly due to limitations present in data for earlier years, but also to recognize the lags inherent in the medical R&D cycle. For instance, it would be inappropriate to count benefits that accrue from therapies developed prior to 1970. Typically, drug development takes 10 to 15 years from inception to launch (DiMasi et al., 2003); enforcing an 18-year lag thus seems a conservative assumption. Moreover, we also over-count R&D costs by including some spending—towards the late 1990s, for instance—whose benefit has not yet been realized or measured.

This paper is outlined as follows. Section 1 describes our framework for valuing gains in cancer survival. Section 2 explains the
1. Framework for evaluating the war on cancer

Our analysis proceeds in three steps. First, we estimate the value to individuals of gains in cancer survival. Second, we decompose these gains into those accounted for by improved treatment and earlier detection. Finally, we estimate the aggregate social value of cancer gains, compare these to costs, and examine the distribution of gains across patients and producers. This section describes, in that order, the framework we use to answer each of these questions.

1.1. Valuing gains in cancer survival

Our framework for valuing gains in cancer survival is based on the approach utilized by Becker et al. (2005). These authors derive valuation formulas for infra-marginal changes in longevity. 4 It is important to think of cancer survival changes as infra-marginal, because the alternative to treatment is often rapid mortality, not slight increments in mortality risk. Indeed, infra-marginal valuation should ideally be the standard approach to estimating the value of discrete survival gains from any disease. Changes in health-related mortality risk seem poorly matched to natural experiments that estimate the willingness-to-pay for marginal risk reduction.

Following Becker et al. (2005), we consider a hypothetical life-cycle individual (HLCI) whose disease begins in a given year and who faces that year’s cross-sectional survival and annual income for the remainder of his or her life. The cross-sectional survival is assumed to reflect the detection and treatment technologies available at that time. In order to calculate the individual value of improved cancer survival in that year, we ask how much a HLCI contracting cancer would be willing to pay to avoid the less favorable survival curves from the earlier year. This amount represents the patient’s willingness-to-pay for the improved survival probabilities. We can then do this separately for the increment in survival generated by better treatment and better detection.

Let \( S' = \{ S_{t} \} \) represent the vector of survival probabilities, based on the available treatment technology present in year \( t \). \( S_{t} \) represents the probability that the patient survives at least \( j \) years after the disease has begun in year \( t \), given that the disease is detected at stage \( i \). For example, \( S_{1980}^{\text{early,}j} \) is the probability that an individual who gets cancer in 1980 and is diagnosed early will live at least \( j \) years past 1980. Equivalently, \( S_{1980}^{\text{late,}j} \) is the probability that an individual who gets cancer in 1980 and is ultimately diagnosed late will live at least \( j \) years from 1980. Note that in this example, 1980 is the date of incidence, as opposed to the date of detection. For notational simplicity, we omit the superscript \( t \) where possible, with the understanding that survival profiles generally vary over time.

We can capture the salient features of the problem by supposing that an individual’s survival profile depends on when his disease gets detected, and the survival probabilities of cancer patients diagnosed at various stages. Define \( p \) as the probability of early detection. Define \( S \) as a vector of survival probabilities for various stages of diagnosis. Finally, define lifetime full-income as \( Y \). The individual’s indirect utility then depends on these three “goods,” according to \( e(p, S, Y) \).

In a simple environment with a single consumption good, a complete contingent claims market, and an interest rate equal to the individual’s rate of discount, consumption profiles will be constant. Therefore, if \( y \) represents an individual’s full income in a single period, it will be the case that consumption \( c = y \) in each period. As a result, indirect utility can be represented as:

\[
V(p, S, y) = u(y) \cdot \sum_{i} p_i A(S_i)
\]

(1)

Here, \( A(S_i) = \sum_{j=0}^{\infty} \left( \frac{1}{1 + r} \right)^j S_{ij} \), which is the expected value of an annuity paying one unit of consumption each year to an individual with the survival profile defined by \( S_{ij} \).

Once we quantify \( V(p, S, y) \), we can define the willingness to pay for arbitrary increments in survival and detection. In particular, an individual currently enjoying \( p' \) and \( S' \) is willing to pay wtp to avoid having to face \( p \) and \( S \), where:

\[
V(p', S', y - \text{wtp}) = V(p, S, y)
\]

(2)

This equation provides us with the conceptual means to calculate the value to patients of changes in survival and detection probabilities.

1.2. Valuing improvements in treatment and detection

Gains in cancer survival may be driven by advances in both treatment and detection. Advances in treatment, such as the development of new chemotherapeutic regimens or surgical techniques, improve survival by delaying progression of the disease or eliminating it outright. Improvements in detection, which can occur through the development of new technologies and/or public health efforts to increase screening, improve survival by allowing more patients to be diagnosed, and therefore treated, earlier. Given the potential importance of each of these mechanisms in accounting for overall gains in survival, it is useful to decompose the overall willingness-to-pay for survival improvements into the portion due to treatment versus detection.

Eq. (2) characterizes the individual’s willingness-to-pay for a bundle of detection and survival probabilities. Analogous to the well-known Blinder–Oaxaca decomposition in labor economics (Blinder, 1973; Oaxaca, 1973), we can decompose the total change in value into the component driven by changes in detection probabilities, holding survival profiles constant, and the remaining component driven by changes in survival profiles alone. Specifically, we can write:

\[
\Delta V = V(p', S', y) - V(p, S, y)
\]

\[
= \left[ V(p', S', y) - V(p', S, y) \right] + \left[ V(p', S, y) - V(p, S, y) \right]
\]

(3)

\[
= \Delta V_{\text{treat}} + \Delta V_{\text{detect}}
\]

The first term in square brackets is the increase in lifetime indirect utility due to improvements in treatment: this is defined as changes in the survival profile, holding the probability of early detection constant. The second is the increase in utility due to improvements in detection: analogously, this is defined as changes in the probability of early detection, holding survival profiles constant. If we were quantifying marginal changes in treatment, \( dV/d\lambda \), their monetary values would be approximately equal to \( dV/d\lambda \), where \( \lambda \) is the marginal utility of income. Since we are quantifying infra-marginal changes \( \Delta V \), the analogous approach is to divide by the average utility of income, \( M \). This is defined in terms of the individ-

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4 This infra-marginal valuation approach has also been used to value improvements in HIV/AIDS survival (Philipson and Jena, 2006) and to calculate the general equilibrium effects of large improvements in mortality (Jena, Mulligan, Philipson, and Sun, 2008).
ual's willingness-to-pay and utility change, as in:

\[ M = \Delta V_{\text{wtp}} \]

(4)

Dividing both sides of (3) by (4):

\[ \text{wtp} = \frac{\Delta V_{\text{detect}}}{M} + \frac{\Delta V_{\text{treat}}}{M} \]

(5)

The first term is the approximate monetary value of detection gains and the second the approximate value of treatment gains. These are approximations, because each change in lifetime indirect utility is divided by the average utility of income, calculated across the entire improvement in survival, not just the specific portion due to detection or to treatment.

1.3. Estimating aggregate consumer surplus from improvements in cancer survival

Equation (2) allows us to calculate the annual amount an individual is willing to pay for a given improvement in cancer survival from a baseline year \( t \) to year \( t' \). We define this annual willingness to pay as \( \text{wtp}_{t,t'} \). The lifetime willingness to pay, \( \text{wtp}_{t'} \), for the improved survival is the expected present value of the stream of these annual willingness to pay terms:

\[ \text{WTP}_{t,t'} = \text{wtp}_{t,t'} \cdot \sum_{i,j} p_i^A(S'_i) \]

(6)

The individual's lifetime consumer surplus from gains in survival equals this lifetime willingness-to-pay, net of lifetime spending on cancer. Expected lifetime spending for a patient whose disease begins in year \( t \) can be defined as \( C_t \), where:

\[ C_t = \sum_{i} p_i^A \left( \sum_{j=0}^{\infty} \frac{S^j_{i,t}}{(1 + r)^j} c^i_j \right) \]

(7)

In this expression, \( c^i_j \) is the average cost (across all disease stages) \( j \) years after cancer strikes. The net cost of moving to year \( t' \) technology from year \( t \) technology is thus \( C_{t'} - C_t \). As a result, the individual's lifetime consumer surplus from this movement in technology can be represented as:

\[ \text{CS}_{t,t'} = \text{WTP}_{t,t'} - (C_{t'} - C_t) \]

(8)

Using individual consumer surplus values, we can then aggregate up to total consumer surplus. Since the willingness to pay for survival gains, and therefore consumer surplus, is a function of income in our model, the total consumer surplus accruing to a cohort of cancer patients whose disease begins in a given year \( t' \), \( \text{CS}_{t'} \), can be calculated by integrating over the income distribution and multiplying by the incidence of cancer in that year, \( N_t' \):

\[ \text{CS}_{t'} = N_t' \int \text{CS}_{t,t'}(y) f(y) dy \]

(9)

In this expression, \( \text{CS}_{t,t'}(y) \) is the consumer surplus for a patient with income \( y \); \( f(y) \) is the distribution of income, and \( N_t' \) is the number of patients whose disease begins in the year \( t' \). The aggregate consumer surplus across all cohorts ever afflicted with cancer is the present discounted sum of the consumer surplus accruing to each cohort:

\[ \text{CS} = \sum_{t' \geq t} \frac{\text{CS}_{t'}}{(1 + r)^{t'-t}} \]

(10)

2. Data and methods

In the previous section, we outlined a theoretical framework for calculating the consumer surplus arising from gains in cancer survival and estimating the relative contributions of advances in cancer treatment and detection. Implementing our approach requires several pieces of information, including: (1) assumptions about the nature of the utility function, (2) estimates of stage-conditional survival and detection probabilities, (3) estimates of the income distribution, and (4) historical estimates of cancer spending. The following sections describe, in this order, how we incorporate each of these elements into our analysis.

2.1. Parameterizing the willingness to pay

Following Becker et al. (2005), we assume that the instantaneous utility function takes the following form:

\[ u(c) = \frac{c^{\gamma} - (1/\gamma)}{1 - (1/\gamma)} + \alpha \]

(11)

The parameter \( \alpha \) is a normalization factor that determines the level of consumption at which the individual would be indifferent between life or death (at which point utility equals zero), and \( \gamma \) is the intertemporal elasticity of substitution. Following these authors, we assume that \( \gamma = 1.25 \) and \( \alpha = -14.97. \)

Given expressions (2) and (11), it is straightforward to show that the closed-form solution for the willingness to pay for an improvement in detection and treatment prospects from year \( t \) to \( t' \) is:

\[ \text{wtp}_{t,t'} = \gamma - \left[ \sum_{i} p_i^A(S'_{i,t'}) \left[ \gamma^{y_i^{t-t'}} \left[ 1 - \frac{y_i^{t-t'}}{\gamma} \right] \right] - \alpha \right] \left[ 1 - (1/\gamma) \right] \]

(12)

2.2. Estimates of survival and detection probabilities

Our estimates of survival and detection probabilities are drawn from prior work (Sun et al., 2010), which we briefly summarize here. In that analysis, we use data from the Surveillance, Epidemiology, and End Results (SEER) program to estimate changes in cancer survival and detection between 1988 and 2000. SEER is a database of reports from 17 cancer registries, maintained by the National Cancer Institute, which has tracked cancer incidence since 1973. We restricted our focus to tumors from the nine original SEER registries present in 1973, which cover roughly 10% of the US population. When an individual living in an area covered by a registry is diagnosed with cancer, a report is sent to SEER describing that individual’s age at diagnosis, year of diagnosis, and gender. In addition, SEER collects data about the tumor itself, including its location and stage at diagnosis. Once diagnosed, patients are (generally) actively followed up until they die or are otherwise lost to follow-up. For each patient, SEER then reports the number of years for which the patient was known to be alive, as well as the patient’s status at the end of that time (death or lost to follow-up).

We focused our analysis on all cancers combined, as well as the following individual cancers: breast, colorectal, lung, pancreas, and non-Hodgkin’s lymphoma. SEER data provide four classifications for stage: in situ, localized, regional metastasis, and distal metastasis. Because the proportion of patients diagnosed with in situ cancer was relatively small for most of the cancers we examined, we combined the in-situ and localized stages. We thus

\[ For\ further\ justification\ of\ these\ parameter\ assumptions,\ please\ see\ Becker,\ Philipson,\ and\ Soares\ (2005).\]
modeled breast, colorectal, lung, and pancreatic cancers as proceeding through three stages: in situ/localized, regional metastasis, and distal metastasis. For non-Hodgkin’s lymphoma and all cancers combined, differences in coding across cancers and years required us to model disease in two stages: a late stage comprising of distal metastasis and an early stage consisting of all other stages combined.

While further details of the methodology used to estimate survival and detection probabilities can be found in our complementary analysis, two points should be stressed. First, we took a period (cross-sectional) approach to estimating survival, rather than a cohort (longitudinal) approach. Formally, the period probability, in year $t$ of surviving from year $5$ to year $6$, is calculated as the fraction of patients acquiring the disease in year $t−6$ who survived from year $t−1$ to year $t$. The cohort approach, on the other hand, would calculate this probability as the fraction of people acquiring the disease in year $t$ who survived from year $t+5$ to year $t+6$.

We employ the period approach for two reasons. First, it facilitates calculation of long-term survival probabilities, which are necessary to calculate accurate estimates of life expectancy.6 Second, the period approach to calculating survival probabilities in year $t$ reflects the state of year $t$ technology. The cohort-based approach, on the other hand, amalgamates technology across all years during which the cohort will live. Of course, the period approach limits the number of years available to us: for instance, to calculate the 1988 probability of surviving 6 years, we will need to observe patients who entered the registry in 1982. While SEER data were collected beginning in 1973, reliable data on staging were only available for many cancers starting in 1982. While we could therefore begin our analysis with cancers starting in 1983, as mentioned above, the period approach limits the number of years available to us for earlier years. As a result, we focused our efforts on survival gains between 1988 and 2000: doing so allows us to estimate survival for patients in 1988 for up to six years using the SEER data directly. Since about half of cancer patients survived six years in 1988, we viewed this as the minimum acceptable panel length for our first year of analysis.

Second, we used all-cause mortality to estimate survival, as opposed to disease-specific mortality. All-cause mortality is sensitive to gains from treatment of competing risks for cancer, and it avoids issues associated with cause-of-death misattribution (Moy et al., 2001; Black et al., 2002). One drawback of using all-cause mortality, however, is the possibility of capturing secular improvements in mortality that have little to do with improvements in cancer treatment or detection. As a result, we assessed the sensitivity of our results by re-estimating consumer surplus values using cancer-specific mortality. Our results are largely unchanged, and perhaps even suggest larger gains in mortality.

Important to our analysis, SEER data report survival from the date of diagnosis, not the date of incidence. This can result in “lead-time bias” in our estimates of survival because individuals who are detected earlier may appear to live longer, even if earlier detection provides no therapeutic benefit. If the transition time between stages of the disease is known, survival from the date of incidence can be estimated as the observed survival from the date of diagnosis at a given stage plus the average transition time from incidence to that stage. For example, if the observed post-diagnosis survival for a given cancer is 5 years for individuals with regional disease, and it is known that it takes 2 years for the cancer to progress from in situ/local disease to regional disease, then the estimated survival from date of incidence would be 7 years. Note that for this estimation strategy to deliver reliable estimates of survival after cancer incidence, the transition times between stages must be calculated in the absence of treatment. Such transition times may, of course, be difficult to observe in actual practice. To deal with this issue, we examine the sensitivity of our results to lead time bias by subjecting the estimation to a range of transition time assumptions, from: (a) for in situ/local patients, the date of diagnosis and the date of incidence are the same, to (b) for later stage patients, the disease takes on a range of transition times of up to three years between each stage. For diseases with three stages such as breast, colon, pancreatic, and lung cancer, this amounted to an assumed six-year transition time between early and late disease.7 We report baseline results assuming no transition time between stages, and then show how our results vary with different transition time assumptions. Our qualitative conclusions for value, net benefit, and the distribution of gains are all quite insensitive to transition times between stages.

### 2.3. Estimates of income

We used the Medical Expenditure Panel Survey (MEPS), a nationally representative survey of the US civilian population, to estimate the income distribution for US cancer patients in 2000.8 We dropped respondents with negative or zero personal income. Note that MEPS only reports earned income, while full income, which reflects the value of leisure time, is the relevant measure. Following Murphy and Topel (2006), we assume that (full-time employed) individuals work 2,000 hours annually and have 4,000 hours available annually for work and leisure, so that full income is equal to twice earned income. While there are other data sources with higher-quality income data, MEPS permits us to identify the income distribution for cancer patients specifically.9

In Fig. 1 below, we plot the year 2000 cumulative distribution of full income for cancer patients and the general population, using US Census data to calculate the latter. Generally, cancer patients are wealthier than the general population; the median full income for a cancer patient is $54,756 (in year 2006 dollars), compared to $49,842 in the general population. However, Fig. 1 does suggest that the cumulative distribution function (cdf) for the general population crosses the cdf for the cancer population, so that at the tail end of the distribution, cancer patients are poorer.

### 2.4. Estimates of cancer spending

In order to estimate consumer surplus, we must estimate $C^t$, the expected lifetime spending of an individual afflicted with cancer whose disease begins in year $t$. Recall that $C^t = \sum_i p^t_i \sum_j (S_{ij} / (1 + r))^t c^t_j$, where $c^t_j$ is the average cost (across all disease stages) $j$ years after cancer strikes. Because direct estimates of $c^t_j$ are not available, we estimate lifetime cancer spending as follows.

---

6 For example, for patients who were diagnosed with cancer in 2000, the probability of surviving 20 years is estimated using the data from patients who were initially diagnosed in 1980. By contrast, the cohort-based approach would require following patients whose disease began in 2000 until 2020.

7 As we discuss in the next section, these ranges were chosen because they fit the available literature on transition times between cancer stages.

8 We used the “total person-level” income variable (TTLP00X), which is the sum of all personal income except taxes and sales income, as a measure of individual income. For retirees, income is the sum of wages, pension payments, transfer payments, and income from investments.

9 In sensitivity analyses, we used the Current Population Survey (CPS) income distributions for the entire US population. As shown in Appendix Table A2, the results were qualitatively similar.
Table 1

<table>
<thead>
<tr>
<th>Cancer</th>
<th>Change in life expectancy (years)</th>
<th>Change in detection probability (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Overall</td>
<td>In Situ/Local</td>
</tr>
<tr>
<td>All cancers combined</td>
<td>3.9</td>
<td>3.7</td>
</tr>
<tr>
<td>Breast</td>
<td>3.6</td>
<td>0.0/2.1</td>
</tr>
<tr>
<td>Colorectal</td>
<td>1.7</td>
<td>0.86</td>
</tr>
<tr>
<td>Lung</td>
<td>0.83</td>
<td>1.9</td>
</tr>
<tr>
<td>NHL</td>
<td>3.5</td>
<td>3.3</td>
</tr>
<tr>
<td>Pancreas</td>
<td>0.46</td>
<td>1.1</td>
</tr>
</tbody>
</table>

Notes: NHL = non-Hodgkin’s lymphoma. Numbers in parentheses indicate negative values. All cancers combined and non-Hodgkin’s lymphoma were modeled as two-stage diseases, so the results for in situ/local disease represent in situ/local as well as regional disease. All cancers combined includes the entire universe of cancers described in Methods, including diseases apart from breast, colorectal, lung, NHL, and pancreas.

First, we adopt a cross-sectional approach to estimating spending, so that spending in the 20th year of the disease for patients whose disease began in 2000 is estimated by the year 2000 spending of patients whose disease initially began in 1980. In this case, the following relationship holds:

\[ E_t = \sum_{j=t}^{\infty} N_j e_j \]  

(13)

\[ E_t \] represents total spending on cancer in year \( t \) and \( N_j \) is the number of patients whose disease began \( j \) years ago, and who are alive in year \( t \). Finally, \( e_j \) is spending by these individuals in year \( t \). \( N_j \) can be estimated using SEER data, which tracks patients over time. Additionally, Brown et al. (2001) find that historically, national expenditures on cancer equal 5% of total health expenditures, so we estimate \( E_t \) to be 5% of national health expenditures.10

Even with these assumptions, more structure is needed to identify \( c_j \). We assume that that the share of total lifetime spending that is spent in each year of the disease remains constant over time, so that spending in any given year of the disease is a fixed multiple of spending in the initial year of the disease. Mathematically, this implies that:

\[ c_j = \alpha_j c_0 \]  

(14)

where \( c_j \) is cancer spending in the first year of disease for someone who gets cancer in year \( t \) and \( \alpha_j > 1 \) is the proportion by which spending \( j \) years later is greater than initial spending. \( \alpha_j \) is assumed to be independent of the year an individual gets cancer. We estimate \( \alpha_j \) using data from the Medical Expenditure Panel Surveys (MEPS), which provides data on whether a patient has ever had cancer, the year the disease began, and medical spending in the current year. Because MEPS has relatively few cancer patients, we pooled all cancer patients between 1996 and 2004, resulting in a sample of 4,314 individuals who have had cancer for an average of 4.5 years.11 Using this sample, we estimate spending on cancer care for each individual as their medical spending in a given year minus the average spending for non-cancer patients of the same age and gender. These estimates of cancer spending, as well as the number of years the individual reports having cancer, are then used to estimate \( \alpha_j \). With estimates of \( \alpha_j \), \( E_t \), and \( N_j \), expressions (13) and (14) can be used to directly solve for the entire sequence of \( c_j \). This, in turn, allows us to calculate lifetime spending on cancer by year of cancer incidence. Intuitively, information on incidence, survival, and the relative growth in spending from the date of incidence, allow us to construct a time-series for medical spending.12

3. Results

3.1. Increases in cancer detection and survival after treatment

Table 1 shows changes in life expectancy and the probability of early detection between 1988 and 2000 for all cancers combined, as well as the individual tumor types of breast cancer, colorectal cancer, lung cancer, pancreatic cancer, and non-Hodgkin’s lymphoma. (Note that the “all cancers combined” group also includes tumor types not individually reported in the table, such as prostate cancer.) For all cancers combined, we find that overall life expectancy increased by 3.9 years between 1988 and 2002, driven predominantly by increases in life expectancy for early stage patients (3.7 years). Indeed, nearly all of the cancers we examined had

10 National health expenditure data are obtained from the Centers for Medicare & Medicaid Services.

11 MEPS does not contain patients who were institutionalized or hospitalized at the time of the survey, which may cause us to underestimate costs. However, note that we use MEPS to estimate the distribution of costs over the life cycle, as opposed to the total level of cancer costs, so this omission will only affect us to the degree that it is not evenly distributed across the cancer lifecycle, and even then, would only affect our estimate of how costs are distributed across the lifecycle, not the total level of costs.

12 In estimating cancer spending, we inflated all dollar values to year 2006 dollars. In addition, we used a sixth order polynomial regression to smooth out and extrapolate values of \( \alpha_j \).
increases in survival at earlier disease stages, with generally little improvement in survival for patients who were diagnosed at distal metastasis. However, one notable exception is non-Hodgkin’s lymphoma, where survival gains for distal metastasis (3.4 years) were on the same order as gains for early stage disease (3.3 years).

Breast cancer and non-Hodgkin’s lymphoma also saw reasonably large increases in the percentage of patients diagnosed at earlier stages. Between 1988 and 2000, the percentage of breast cancer patients diagnosed at the in situ/localized stage increased by 8.1%, and the number of patients diagnosed at an early stage for non-Hodgkin’s lymphoma increased by 4.1%. For all cancers combined, the percentage of patients diagnosed with early stage disease increased by 5.6%. In contrast, the remaining cancers we studied (colorectal, lung and pancreatic) saw more modest increases in the percentage of patients diagnosed at earlier stages. Note that, due to limitations in the staging data for prostate cancer, the SEER data do not admit analysis of improvements in prostate cancer detection, which is commonly thought to have played a relatively large role in survival for that disease.14

3.2. Individual value of cancer survival gains

Given our estimates of survival and detection probabilities, we can use the methodology described earlier to calibrate the value of cancer survival gains between 1988 and 2000. For each cancer in our analysis and for all cancers combined, Table 2 presents a number of metrics for the value consumers place on the survival gains observed between 1988 and 2000: (1) annual willingness to pay, in each year of their remaining life; (2) cumulative lifetime willingness to pay; and (3) the implied value of a statistical life-year, calculated by dividing cumulative willingness-to-pay for improved survival, by the observed increase in life expectancy drawn from Table 1.

Overall, our results suggest that survival gains between 1988 and 2000 were of tremendous value to individuals with cancer. For all cancers combined, patients whose disease began in 2000 were willing to pay an average of $322,438 (or $30,737 annually) in order to avoid the less favorable technologies of 1988. Given the estimated improvement in life expectancy, this reflects a value of a statistical life year of roughly $82,676. Breast cancer patients were willing to pay roughly $28,000 annually for survival gains, while colon cancer patients were willing to pay roughly $20,000 annually. Intriguingly, the largest values were for lung cancer patients ($37,777), non-Hodgkin’s lymphoma patients ($33,812 annually), and colorectal cancer patients ($44,666 annually). For non-Hodgkin’s lymphoma, a large value is not particularly surprising, given that survival increased by at least 3.5 years over this period. Overall, our estimates suggest that patients’ willingness to pay for cancer survival improvements between 1988 and 2000 imply a value of a statistical life year of roughly $80,000 for most cancers, although in the case of pancreatic cancer, the implied value of a life year is roughly $56,000.

In Table 2, we estimated the value of cancer survival gains using the calibrated model of lifetime utility discussed in Section 2. An alternative method for estimating the value of cancer survival improvements would be to multiply the estimated gains in life expectancy by an assumed value of a statistical life year. Commonly used values of a statistical life year vary widely. At the lower end, $50,000 per year is the cost-effectiveness threshold used by many public payers (Devlin and Parkin, 2004). Based on a review of the literature, Cutler and Richardson (1997) estimate a value of $100,000, and using the demand for dialysis, Lee et al. (2009) estimate the value of a life year at $129,000. At the higher end, Murphy and Topel (2006) estimate values between $100,000 and $350,000 at various points during the life cycle, while in a review of the literature, Aldy and Viscusi (2007) find values ranging from $300,000 to $1 million. Given this range of values, Table 3 shows the value of 1988–2000 cancer survival gains assuming a $50,000, $100,000, or $300,000 value of a statistical life year. Using a $100,000 value of a life year leads to lifetime value of cancer survival gains similar to those shown in Table 2, which is not surprising given the values of statistical life-year implied by our analysis. At $50,000 per life year, our estimated survival gains are worth roughly $195,000 for all cancers combined, and at $300,000 per life year, they are worth roughly $1 million.

Given an average annual full income of $66,890 in the MEPS sample, Table 2 implies that individuals getting cancer in 2000 would be willing to pay a significant fraction of their full income, nearly half, in order to avoid the less favorable technologies of 1988. In Fig. 2, we explore this point further by graphing how the calibrated willingness to pay for the observed improvements in survival varies with income.

In line with Table 2, our results suggest that individuals with cancer were willing to pay a large share of their annual income for survival gains since 1988, particularly among those with the highest levels of full income. Generally speaking, we find that survival gains have been generally worth around 50–60% of income for all cancers combined and non-Hodgkin’s lymphoma, with lower levels for breast, lung, and colorectal cancer, and much higher levels.

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14 The SEER variables used to assign a stage to prostate cancer are not defined until 1995.
for pancreatic cancer. Indeed, we find that in some cases, individuals with pancreatic cancer would be willing to pay nearly 80% of their full income for survival gains.

The high willingness to pay for improved survival as a share of full income for pancreatic cancer is at odds with the conventional wisdom, for two reasons. First, pancreatic cancer survival improved by less than one year. However, theoretical work by Becker et al. (2007) argues that terminally ill patients may place unusually high value on incremental survival gains. The intuition behind their result relies on a familiar diminishing returns argument. While healthy people may be willing to pay moderate amounts for modest survival gains, terminally ill patients, who are endowed with little health, may be willing to pay far larger amounts for similarly sized gains. Put differently, because the value of wealth at death is zero or close to it for most individuals, the willingness to pay for even minor improvements in longevity may be large at the end of life.15 Put differently, the gains in treatment account for such a large percentage of the value of survival gains, given the advances in early detection shown in Table 1. Our results suggest that the gains in treatment have been even larger. Put differently, the gains in stage-conditional survival have been larger than the gains in survival due to the movement of patients into earlier stages at the time of detection.

3.3. Individual consumer surplus arising from improvements in cancer survival

Having calculated an individual patient’s annual willingness to pay for improvements in survival, we now turn to the issue of lifetime consumer surplus, which requires us to calculate the lifetime value of survival gains and the increase in expected lifetime costs. Fig. 3 presents our estimates of lifetime spending for cancer patients between 1988 and 2000. Fig. 3 shows a gradual decline between 1990 and 2000, due primarily to the 3% annual rate of discount applied, as discussed in equation (10). In the absence of discounting across cohorts, our results suggest that the lifetime cost of cancer care increased by roughly $52,000, a 142% increase. In comparison, the medical CPI increased by 90% over this time period.16 Table 4 presents estimated lifetime costs for an individual in each cohort of patients between 1989 and 2000 as well as individual estimates of the lifetime value of observed survival gains. These are used to calculate the individual lifetime consumer surplus for each cohort.

The first column in Table 5 presents the estimated increase in life expectancy for patients whose disease begins in the given year, compared to patients whose disease begins in 1988. The second column represents the lifetime willingness to pay for this survival consumption and leisure, rather than increasing consumption and leisure in a given year. As a result, wealthier people are likely to place more value on survival gains, causing health to resemble the economic definition of a “luxury” good.

Table 4 shows that the value of 1988–2000 survival gains shown in Table 2 and Fig. 1 were primarily driven by advances in treatment, rather than advances in detection.

For all cancers combined, the willingness to pay for treatment advances alone comprises 97% of the total value of cancer gains. Treatment advances have been particularly important in the case of non-Hodgkin’s lymphoma and pancreatic cancer, where nearly all of the value of survival gains is due to treatment advances. For breast and lung cancer, roughly 90% of the value of survival gains from 1988 to 2000 appears to result from advances in treatment, while for colorectal cancer, 75% of the total value of survival gains is due to the value of treatment advances. At first glance, it may seem surprising that the gains in treatment account for such a large percentage of the value of survival gains, given the advances in early detection shown in Table 1. Our results suggest that the gains in treatment have been even larger. Put differently, the gains in stage-conditional survival have been larger than the gains in survival due to the movement of patients into earlier stages at the time of detection.

15 One prediction of the Becker et al. analysis is that the estimated value of a statistical life year would be quite large for very small increments in survival, as in pancreatic cancer, but our estimates suggest that values of a life year do not differ much across cancers. This discrepancy is largely an artifact of our calibration procedure. In our approach, wealth accumulation in our model starts at time zero (when cancer first appears), and life expectancy with pancreatic cancer is low. Thus, while individuals with pancreatic cancer may be willing to spend larger shares of their remaining wealth for a given unit increase in life expectancy, the overall willingness to pay per unit will not be higher simply because wealth is lower.

16 In addition, using Medicare claims data, Warren et al. (2008) estimate that between 1991 and 2002, the cost of initial cancer treatment (treatment from two months before to twelve months after diagnosis) increased by $7,319 for lung cancer, $5,345 for colorectal cancer, and $4,189 for breast cancer, while the cost of initial treatment for prostate cancer fell by $196. Our estimates of the lifetime cost for all cancers combined appear larger than these findings, and may thus be overstating the increases in cancer cost, while understating the gains in consumer surplus.
improvement, while the third column shows the increase in the lifetime costs of cancer care. Finally, the last column shows the lifetime consumer surplus associated with the improvement in cancer survival. Between 1989 and 2000, the average lifetime cost of cancer care was roughly $41,000 higher (averaged across all years) compared to 1988. The lifetime cost of cancer for an individual getting cancer in 2000 was approximately $37,000 higher. The lifetime value of cancer survival gains vastly exceeded this increase in cost, and led to substantial consumer surplus. Indeed, we find that lifetime consumer surplus has been rapidly increasing over time. In 1989, for example, the average gain in consumer surplus per patient was $31,000, while in 2000, the average gain in surplus was roughly $285,000; this implies an annual increase of 21%. This rapid increase is generated by substantial gains in survival, accompanied by relatively little growth in the costs of cancer care, relative to 1988 levels.

3.4. Aggregate consumer and producer surpluses

Given our estimates of individual lifetime consumer surplus gains arising from improvements in cancer survival, Table 6 presents our estimates of gains in aggregate consumer surplus, which is simply the number of newly incident cases in a given year.
multiplied by the individual consumer surplus gains reported in Table 5.

In the aggregate, we find that patients have received tremendous value from treatment and detection advances since 1988, with estimated total consumer surplus gained of roughly $1.9 trillion for all cancer patients whose disease began between 1989 and 2000. Table 6 also presents estimates of gains in producer surplus.

The ranges for producer surplus represent different means by which costs of care are translated into profits. We assume that profit margins range from the high margins enjoyed by producers of on-patent pharmaceuticals, to the lower margins enjoyed by hospitals. For drugs, Caves et al. (1991), Grabowski and Vernon (1992) and Berndt et al. (1996) estimate that producer variable costs are 20% of sales. On the other hand, Gaynor and Vogt (2003) find that hospitals have profit margins of 25%. Consistent with these estimates, we allow producer profits to range between 20% and 80% of sales. Since health care spending on cancer is, of course, a combination of drug therapies, as well as physician and hospital compensation, actual profit margins are likely to fall somewhere between the two bounds we use. Even with the most generous assumptions on producer sur- pluses, Table 6 illustrates that while producers have benefited from increases in cancer surplus, these gains have been small relative to the gains experienced by cancer patients. We estimate the producer surplus to be $98–393 billion from 1988 to 2000.

The sum of producer and consumer surpluses arising from improvements in cancer survival from 1988 to 2000 is $2.0 trillion to $2.3 trillion. In order to calculate the social surplus arising from these gains in survival, we must deduct the amount spent on cancer R&D. There are two challenges to estimating this quantity. First, a significant proportion of R&D comes from pharmaceutical firms, which do not typically report R&D by disease category. Second, ideally, we would calculate the R&D spending which produced the observed survival gains between 1988 and 2000. However, given that biomedical research often takes time to be translated into clinical practice, it is difficult to directly determine which part of R&D in a given year produced these gains. For example, it is likely that some of the survival gains between 1988 and 2000 were due to research spending in earlier years, while some of the R&D spending between 1988 and 2000 did not produce the observed survival gains during this time period.

To address these issues, we construct an upper bound on R&D spending as follows. First, we calculate the total value of National Cancer Institute spending between 1971 and 2000, using data on the Institute’s annual appropriations (Appendix Table A1). After converting each year’s appropriations to 2006 dollars using the Consumer Price Index and discounting to 1988 at a 3% interest rate, we find that the total value of cancer R&D spending was roughly $82 billion. Assuming that NCI spending represents 25% of all cancer R&D spending leads to an upper bound for cancer R&D of roughly $300 billion. This estimate is likely an upper bound for two reasons. First, as already discussed, it is unlikely that all spending between 1971 and 2000 contributed to the survival gains between 1988 and 2000. Second, it is likely that NCI spending accounts for more than 25% of total cancer R&D. For example, Jaffe (1996) finds that, generally speaking, the federal government accounts for 35% of R&D spending. Using $300 billion as an upper bound for cancer R&D, we estimate the total social surplus of 1988–2000 survival gains to be roughly $1.6–1.9 trillion, with 82–95% of this surplus accruing to patients and 5–19% accruing to producers.

3.5. Sensitivity analyses

In this section, we describe analyses used to determine the sensitivity of our results to potential sources of bias. The first we consider is the possibility of lead time bias. Lead time bias occurs because registry data such as SEER report survival from the date of diagnosis, not the date of incidence. As a result, improvements in detection lead to the inclusion of patients at earlier stages of disease. These individuals will have longer remaining life expectancy regardless of whether the disease is detected at the “early” (two years post-incidence) or “late” (eight years post-incidence) stages. In this case, registry data will erroneously show that early detection leads to a six year survival improvement. Thus, lead time bias will tend to overstate the importance of detection improvements. As the example above shows, lead time bias can be overcome by adding the transition time between stages to the observed survival for patients whose disease is diagnosed at later stages. Knowledge of transition times allows accurate calculation of the actual life expectancy from the date of diagnosis, rather than the potentially misleading life expectancy from the date of diagnosis.

Since the transition time between incidence and diagnosis is generally unknown, we examine the degree to which variability in the transition time impacts our findings. As discussed earlier, we consider the range of assumptions on transition times, from zero to three years for all of the cancers we consider except breast cancer, where we consider a transition time of zero to two years between stages. For cancers with three designated stages, such as colon, pancreatic, and lung cancer, this amounts to an assumed six year transition time between early and late disease. There are two major concerns with our approach. The first is the plausibility of a six-year transition time, in the absence of treatment, from early stage to metastatic disease. While there are few studies that have assessed the progression of cancer in the absence of treatment, the extant literature suggests the transition time between early stage and malignant disease is roughly two years or less. For example, Raz et al. (2007) examined survival among 1,324 California patients who were untreated for stage I disease due to patient refusal, death prior to surgery, or contraindications due to risk factors, and found a median survival of 9 months. In the case of breast cancer, Bloom et al. (1962) found the median survival for stage I, stage 2, and stage 3 breast cancer to be 47.3 months, 39.2 months, and 22 months, respectively. Based on these numbers, the average time to transition between grades is about one year, although the staging system used by Bloom, the Manchester system, is a clinical system that differs slightly from the AJCC system used in SEER.17

The second issue concerns the limits of our database itself. With a six-year transition period between early and metastatic disease, individuals whose disease begins in 2000 will not be diagnosed at the metastatic stage until 2006—however, the last year of observation in our data is 2003. Therefore, for our sensitivity analysis, we consider how assuming a three-year transition period between stages affects the value of survival gains from 1988 to 1997.18 Table 7 displays the results.

Not surprisingly, survival gains between 1988 and 1997 tend to be smaller than the gain between 1988 and 2000, although interestingly, the opposite is true for pancreatic cancer. As a result, the value of 1988–1997 survival gains tends to be smaller than the value of 1988–2000 gains. The three-year transition time generally

17 A further review of the limited literature examining transition times between cancer stages can be found in our earlier work
18 Because breast cancer is modeled as a four stage disease a three year transition time between stages would lead to nine year transition period between early and late stage disease. Therefore, for breast cancer, we assume a two year transition time between stages, which amounts to a six year transition from early to metastatic disease.


Table 7

<table>
<thead>
<tr>
<th>Cancer</th>
<th>Survival gain</th>
<th>Lifetime WTP</th>
<th>% Gains due to treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Transition time</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0 year</td>
<td>3 year</td>
<td>$302,280</td>
</tr>
<tr>
<td>All cancers combined</td>
<td>3.12</td>
<td>3.14</td>
<td>-0.011</td>
</tr>
<tr>
<td>Breast</td>
<td>2.80</td>
<td>2.63</td>
<td>0.007</td>
</tr>
<tr>
<td>Colorectal</td>
<td>0.821</td>
<td>0.876</td>
<td>-0.025</td>
</tr>
<tr>
<td>Lung</td>
<td>0.584</td>
<td>0.707</td>
<td>-0.001</td>
</tr>
<tr>
<td>NHL</td>
<td>2.01</td>
<td>3.07</td>
<td>-0.011</td>
</tr>
<tr>
<td>Pancreas</td>
<td>0.509</td>
<td>0.672</td>
<td>0.001</td>
</tr>
</tbody>
</table>

* Result for breast cancer assumes a 2-year transition time, in order to maintain comparability. SEER reports breast cancer in four stages, while the other tumor types are broken down into three stages.

Table 8


<table>
<thead>
<tr>
<th>Ever had</th>
<th>Diabetes</th>
<th>Heart disease</th>
<th>High blood pressure</th>
<th>Lung disease</th>
<th>Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>1994</td>
<td>0.022</td>
<td>-0.011</td>
<td>0.002</td>
<td>-0.013</td>
<td></td>
</tr>
<tr>
<td>1995</td>
<td>0.049</td>
<td>0.007</td>
<td>0.136</td>
<td>-0.005</td>
<td></td>
</tr>
<tr>
<td>1996</td>
<td>0.002</td>
<td>-0.025</td>
<td>-0.054</td>
<td>0.035</td>
<td></td>
</tr>
<tr>
<td>1997</td>
<td>0.045</td>
<td>0.029</td>
<td>0.027</td>
<td>-0.004</td>
<td></td>
</tr>
<tr>
<td>1998</td>
<td>0.019</td>
<td>0.042</td>
<td>-0.079</td>
<td>-0.014</td>
<td></td>
</tr>
<tr>
<td>1999</td>
<td>-0.044</td>
<td>-0.096</td>
<td>-0.025</td>
<td>-0.035</td>
<td></td>
</tr>
<tr>
<td>2000</td>
<td>0.054</td>
<td>0.025</td>
<td>-0.054</td>
<td>-0.026</td>
<td></td>
</tr>
<tr>
<td>2001</td>
<td>0.126</td>
<td>-0.081</td>
<td>-0.022</td>
<td>-0.022</td>
<td></td>
</tr>
<tr>
<td>2002</td>
<td>-0.006</td>
<td>0.048</td>
<td>-0.062</td>
<td>0.013</td>
<td></td>
</tr>
<tr>
<td>2003</td>
<td>0.044</td>
<td>-0.116</td>
<td>-0.031</td>
<td>0.072</td>
<td></td>
</tr>
<tr>
<td>2004</td>
<td>0.037</td>
<td>-0.009</td>
<td>0.055</td>
<td>0.034</td>
<td></td>
</tr>
<tr>
<td>2005</td>
<td>-0.008</td>
<td>0.123</td>
<td>0.067</td>
<td>0.007</td>
<td></td>
</tr>
<tr>
<td>Baseline mean</td>
<td>0.237</td>
<td>0.323</td>
<td>0.719</td>
<td>0.156</td>
<td></td>
</tr>
<tr>
<td>Adjusted R-squared</td>
<td>0.027</td>
<td>0.022</td>
<td>0.019</td>
<td>0.013</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>1007</td>
<td>1002</td>
<td>1006</td>
<td>1007</td>
<td></td>
</tr>
</tbody>
</table>

Notes: Each column is a regression of the variable in the column title on the reported year dummies (1993 is the excluded category), age dummies (70–74, 75–79, 80–84, >85), male, non-hispanic black, and hispanic origin. The sample contains all individuals aged >65, newly diagnosed with breast, prostate, lung, or colorectal cancer; data are taken from the Medicare Current Beneficiary Surveys, 1992–2005. Significance levels are based on standard errors clustered at the individual-level. * p < 0.05, ** p < 0.01, *** p < 0.001.

has modest effects on survival, with the lone exception of non-Hodgkin’s lymphoma. Our results suggest that taking lead time bias into account has little effect on the estimated share of gains due to treatment, with the exception of breast cancer, where taking lead time bias into account increases the share of gains due to treatment from 68 to 82 percent. Willingness-to-pay also typically does not change by more than 10%, with the exception of lung cancer, where the longer transition time substantially raises the value of survival gains. In sum, lead time bias likely has quantitatively modest impacts on our results; if anything, it causes us to underestimate the value of survival gains, and the share of survival accounted for by improved treatment.

“Length bias” refers to unobserved changes in tumor and/or patient characteristics, which may lead us to overestimate survival gains, and the share of gains due to treatment. Improvements in detection will tend to sample slower-growing tumors with more favorable survival characteristics. This is one way in which length bias may cause a spurious increase in survival. Changes in patient characteristics over time may also create length bias. For example, improvements in treating other co-morbid conditions, such as cardiovascular disease, may result in a healthier patient population over time. On the other hand, reductions in background mortality may result in an older, sicker population of incident cases. Overall, secular trends in mortality and co-morbidity may produce length bias of indeterminate sign.

While we are unable to deal directly with unobservable changes in tumor and/or patient characteristics, we assessed the direction and degree of this bias by evaluating changes in the observable characteristics of cancer patients, between 1992 and 2005, using the Medicare Current Beneficiary Survey (MCBS). The MCBS is a nationally representative data set designed to ascertain health status, utilization, and expenditures for the Medicare population. The sample frame consists of aged and disabled beneficiaries enrolled in Medicare Part A and/or Part B, although we use only the aged in this analysis. The MCBS attempts to interview each person twelve times over three years, regardless of whether he or she resides in the community, a facility, or transitions between community and facility settings. The disabled (under 65 years of age) and the oldest-old (85 years of age or over) are over-sampled. The first round of interviewing was conducted in 1991. Originally, the survey was a longitudinal sample with periodic supplements and indefinite periods of participation. In 1996, the MCBS switched to a rotating panel design with limited periods of participation. Each fall a new panel is introduced, with a target sample size of 12,000 respondents, and each summer a panel is retired. The MCBS contains detailed self-reported information, including the prevalence of various conditions.

Using the MCBS, we calculated regression-adjusted trends in co-morbidity prevalence among incident and prevalent cancer cases among the 65+ population. We ran separate regressions for the fol-
Table 9

<table>
<thead>
<tr>
<th>Ever had</th>
<th>Diabetes</th>
<th>Heart disease</th>
<th>High blood pressure</th>
<th>Lung disease</th>
<th>Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>1993</td>
<td>0.012</td>
<td>−0.003</td>
<td>0.026**</td>
<td>0.014</td>
<td>0.003</td>
</tr>
<tr>
<td>1994</td>
<td>0.011</td>
<td>−0.002</td>
<td>0.045**</td>
<td>0.014</td>
<td>0.006</td>
</tr>
<tr>
<td>1995</td>
<td>0.010</td>
<td>−0.019</td>
<td>0.026</td>
<td>0.018</td>
<td>0.001</td>
</tr>
<tr>
<td>1996</td>
<td>0.020</td>
<td>−0.028</td>
<td>0.034</td>
<td>0.005</td>
<td>0.010</td>
</tr>
<tr>
<td>1997</td>
<td>0.018</td>
<td>−0.032</td>
<td>0.053</td>
<td>0.019</td>
<td>−0.009</td>
</tr>
<tr>
<td>1998</td>
<td>0.013</td>
<td>−0.017</td>
<td>0.082***</td>
<td>0.025</td>
<td>−0.009</td>
</tr>
<tr>
<td>1999</td>
<td>0.003</td>
<td>−0.021</td>
<td>0.083***</td>
<td>0.019</td>
<td>−0.009</td>
</tr>
<tr>
<td>2000</td>
<td>0.017</td>
<td>−0.083***</td>
<td>0.067</td>
<td>0.011</td>
<td>−0.006</td>
</tr>
<tr>
<td>2001</td>
<td>0.038**</td>
<td>−0.026</td>
<td>0.095***</td>
<td>0.011</td>
<td>−0.011</td>
</tr>
<tr>
<td>2002</td>
<td>0.028</td>
<td>−0.023</td>
<td>0.117***</td>
<td>−0.004</td>
<td>−0.001</td>
</tr>
<tr>
<td>2003</td>
<td>0.038**</td>
<td>0.004</td>
<td>0.131***</td>
<td>−0.001</td>
<td>0.003</td>
</tr>
<tr>
<td>2004</td>
<td>0.043**</td>
<td>−0.009</td>
<td>0.156***</td>
<td>0.009</td>
<td>0.001</td>
</tr>
<tr>
<td>2005</td>
<td>0.045***</td>
<td>−0.007</td>
<td>0.165***</td>
<td>0.024</td>
<td>−0.007</td>
</tr>
<tr>
<td>Baseline mean</td>
<td>0.119</td>
<td>0.350</td>
<td>0.451</td>
<td>0.140</td>
<td>0.079</td>
</tr>
<tr>
<td>Adjusted R-squared</td>
<td>0.011</td>
<td>0.023</td>
<td>0.025</td>
<td>0.001</td>
<td>0.007</td>
</tr>
<tr>
<td>N</td>
<td>16295</td>
<td>16186</td>
<td>16289</td>
<td>16294</td>
<td>16295</td>
</tr>
</tbody>
</table>

Notes: Each column is a regression of the variable in the column title on the reported year dummies (1992 is the excluded category), age dummies (70–74, 75–79, 80–84, >85), male, non-hispanic black, and hispanic origin. The sample contains all individuals aged >65, ever diagnosed with breast, prostate, lung, or colorectal cancer; data are taken from the Medicare Current Beneficiary Surveys, 1992–2005. Significance levels are based on standard errors clustered at the individual-level.

* p < 0.05.
** p < 0.01.
*** p < 0.001.

4. Conclusion

Despite the abundant research documenting gains in cancer survival, there have been few attempts to characterize the value of cancer R&D. We provide some evidence relevant to this issue, by measuring the value of cancer R&D, relative to its costs. We also calculate how the social gains from R&D have been distributed between patients and producers, and the size of the respective roles played by improved treatment and earlier detection. Our findings suggest that cancer R&D has led to significant gains in cancer survival, which have been large relative to the costs incurred in their pursuit. And while some cancers have experienced larger reductions in morbidity and mortality than others, today’s cancer patients can expect to live longer and healthier lives than their counterparts from earlier decades; these longevity increases far outweigh the costs of achieving them.

Across all cancers, the gains in survival have been worth around half of full income, and even those in the bottom 10% of the income distribution would be willing to pay around 40% of their income for the observed gains in cancer survival. In the aggregate, we find the value of gains in cancer survival to be $1.9 trillion to date, of which 5% to 19% has been appropriated by producers in the form of profits. To provide some context for this number, we estimate that from 1988 to 2000, cancer afflicted roughly 9.5 million Americans. Across the newly diagnosed individuals in all these cohorts, we estimate that life expectancy increased by an average of 3.9 years per patient, compared to its 1988 level. This implies the creation of 23 million life-years. As a result, the average life-year created was worth approximately $86,000 to its recipient. Moreover, we find that, at most, $300 billion has been spent on cancer research, implying that private and federal R&D efforts have enjoyed very large average returns. Our results are consistent with earlier research valuing survival gains for other diseases experiencing substantial

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19 Heart disease includes patients with myocardial infarction, arteriosclerosis, and coronary heart disease.

20 In line with work by Hall and Jones (2007), our results also suggest that cancer is a “luxury good,” in the sense that wealthier patients are willing to devote larger shares of their income to pay for gains in cancer survival.
improvements in mortality. For example, Philipson and Jena (2006) find that in the case of AIDS, producers were able to appropriate approximately 5% of the social surplus arising from innovations in HIV/AIDS medications. By comparison, we find that producers have been able to appropriate 5–19% of the social surplus generated by cancer survival gains from 1988 to 2000. Overall, our results are likely conservative—our estimates of increases in the cost of cancer care are larger than increases in the medical CPI as well as increases in the costs of cancer care reported in the literature. Moreover, we also likely overstate R&D spending for the reasons previously stated. Even with these conservative estimates, we still find that patients’ willingness to pay largely dwarfs the costs of cancer care and R&D. Indeed, even if our estimates overstate the value of cancer gains by twofold, we would still find that cancer R&D has been worthwhile.

Our analysis has focused on the average return to cancer research. This is distinct from the return on the marginal cancer research dollar. Naturally, marginal returns will be lower than average returns. High average returns do not imply that marginal returns exceed marginal cost. The value of the marginal dollar on cancer research is still an open question, and one that is worth further investigation. While the War on Cancer has, on average, paid returns that exceed marginal cost, the value of gains for patients whose cancer occurs after 2000. Even in the absence of any further technological progress in cancer, these patients would still benefit from technologies discovered to date; therefore, our estimates will underestimate the total social value by excluding this group. Second, our analysis assumes that there are no technology improvements for individuals whose cancer begins in a given year. For example, we assume that individuals whose cancer began in 1995 face the 1995 technology for the entire course of their disease. Third, our analysis does not take into account the potential gains from cancer prevention, whether from changes in behavior or from detection of premalignant lesions as occurs with cervical and colorectal cancer.

Our results should be interpreted as a first effort to investigate the value of gains in cancer survival. Future research can extend it in several directions. First, as discussed, progress needs to be made on estimating the marginal return to cancer R&D. Second, our definition of early-stage cancer is very broad, as it includes all individuals for whom the cancer has not metastasized. While this limitation was driven by the data, future analyses could examine how finer definitions of stage affect the results. Third, our limited approach towards modeling the transition time between cancer stages could be replaced by more detailed and clinically specific estimates. In addition, with more years of data, one could examine the sensitivity of our results to assuming longer transition times between stages. Finally, more refined estimates of cancer spending and R&D are useful and could impact our results, particularly if there have been trends in the percentage of health care dollars devoted to cancer.

Acknowledgements

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Appendix A.

Tables A1 and A2.

<table>
<thead>
<tr>
<th>Year</th>
<th>NCI appropriation (§ billions)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1971</td>
<td>1.896</td>
</tr>
<tr>
<td>1972</td>
<td>2.930</td>
</tr>
<tr>
<td>1973</td>
<td>3.481</td>
</tr>
<tr>
<td>1974</td>
<td>3.410</td>
</tr>
<tr>
<td>1975</td>
<td>3.809</td>
</tr>
<tr>
<td>1976</td>
<td>3.845</td>
</tr>
<tr>
<td>1977</td>
<td>3.757</td>
</tr>
<tr>
<td>1978</td>
<td>3.623</td>
</tr>
<tr>
<td>1979</td>
<td>3.399</td>
</tr>
<tr>
<td>1980</td>
<td>3.104</td>
</tr>
<tr>
<td>1981</td>
<td>2.701</td>
</tr>
<tr>
<td>1982</td>
<td>2.462</td>
</tr>
<tr>
<td>1983</td>
<td>2.313</td>
</tr>
<tr>
<td>1984</td>
<td>2.362</td>
</tr>
<tr>
<td>1985</td>
<td>2.419</td>
</tr>
<tr>
<td>1986</td>
<td>2.468</td>
</tr>
<tr>
<td>1987</td>
<td>2.558</td>
</tr>
<tr>
<td>1988</td>
<td>2.498</td>
</tr>
<tr>
<td>1989</td>
<td>2.522</td>
</tr>
<tr>
<td>1990</td>
<td>2.415</td>
</tr>
<tr>
<td>1991</td>
<td>2.392</td>
</tr>
<tr>
<td>1992</td>
<td>2.545</td>
</tr>
<tr>
<td>1993</td>
<td>2.424</td>
</tr>
<tr>
<td>1994</td>
<td>2.372</td>
</tr>
<tr>
<td>1995</td>
<td>2.292</td>
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<tr>
<td>1996</td>
<td>2.275</td>
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<tr>
<td>1997</td>
<td>2.301</td>
</tr>
<tr>
<td>1998</td>
<td>2.350</td>
</tr>
<tr>
<td>1999</td>
<td>2.559</td>
</tr>
<tr>
<td>2000</td>
<td>2.735</td>
</tr>
<tr>
<td>Total</td>
<td>82.215</td>
</tr>
</tbody>
</table>

Notes: Data on NCI appropriations come from the annual NCI Factbook for the years shown above. All dollar amounts shown are in 2006 dollars, discounted to 1988 at a 3% interest rate.

<table>
<thead>
<tr>
<th>Cancer</th>
<th>Annual value of survival gains ($)</th>
<th>Lifetime value of survival gains ($)</th>
<th>Implied value of a life year ($)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All combined</td>
<td>30,900</td>
<td>324,149</td>
<td>83,115</td>
</tr>
<tr>
<td>Breast</td>
<td>28,066</td>
<td>361,554</td>
<td>100,431</td>
</tr>
<tr>
<td>Colorectal</td>
<td>20,588</td>
<td>155,979</td>
<td>91,752</td>
</tr>
<tr>
<td>Lung</td>
<td>37,962</td>
<td>68,695</td>
<td>82,765</td>
</tr>
<tr>
<td>NHL</td>
<td>33,985</td>
<td>275,055</td>
<td>78,587</td>
</tr>
<tr>
<td>Pancreas</td>
<td>44,861</td>
<td>25,817</td>
<td>56,123</td>
</tr>
</tbody>
</table>

Notes: All Amounts are in Year 2006 dollars, discounted to 1990 at a 3% rate. The annual value of survival gains is an individual’s annual willingness to pay, in each year of her remaining life, for observed survival improvements. The lifetime value of survival gains is calculated by multiplying the annual value by the total discounted life expectancy. The implied value of a life year is calculated by dividing the lifetime value of survival gains by the increase in life expectancy.

References


