Sources of Black-White Differences in Cancer Screening and Mortality

Abstract

In 1971, President Nixon initiated the “war on cancer”, which led to dramatic increases in spending on cancer research and treatment. After a steep increase in overall cancer death rates in the early 1990s that has been attributed to tobacco-related cancers, age-adjusted death rates declined substantially. However, not all population subgroups have benefited equally from these advances. Today, blacks have higher mortality and poorer survival rates compared to whites for nearly all cancer sites. We explore black-white mortality differentials for three groups of cancers: 1) tobacco-related, 2) screening-related, and 3) less medically amenable cancers. Using the theory that social conditions act as fundamental causes of diseases, we estimate logistic regression and survival models to examine the associations between race, socioeconomic status and cancer screening and mortality.
Background

In 1971, President Nixon initiated the “war on cancer”, which led to dramatic increases in spending on cancer research and treatment. After a steep increase in overall cancer death rates in the early 1990s that has been attributed to tobacco-related cancers, age-adjusted death rates declined substantially between 1990 and 2006, from 279.8 to 221.1 per 100,000 among men and from 175.3 to 153.7 per 100,000 among women. Age-adjusted cancer death rates in 2006 are below their 1970 levels (Jemal, Ward, and Thun 2010). The major drivers of these declines are favorable changes in health behaviors (e.g., smoking cessation), the development and dissemination of screening technologies, and improvements in treatment for specific cancers. With some caveats regarding the psychological costs and skyrocketing monetary costs of certain treatments and technology-intensive screening procedures, most accounts conclude that we are “winning the war on cancer” (Cutler 2008; Jemal, Ward, and Thun 2010). However, not all population subgroups have benefited equally from these advances.

This is a cause for concern since it speaks to rising inequality where a major cause of death is concerned – cancer remains the second leading cause of death in the United States, accounting for 23.2% of all deaths in 2007 (Xu et al. 2010). The theory that social conditions act as fundamental causes of disease is an appropriate lens through which to examine the sources of disparities in cancer mortality (Link and Phelan 1995). This “fundamental cause” perspective predicts that improvements in health, combined with existing social and economic inequalities, give rise to health disparities because of groups’ differential access to health-enhancing resources (Phelan and Link 2005). Cancer is a prime example of a disease for which innovations in detection and treatment emerged in the past four decades, and we have been able to observe the mortality-reducing impact of these innovations.

In the early 1980s, before the advent of widespread screening for most cancers, black-white disparities in breast and colorectal cancer mortality rates were relatively small or nonexistent (Figures 1 and 3). Subsequently, however, mortality disparities have widened over time for most screening-related cancers. Today, blacks experience higher mortality and poorer survival rates compared to whites for nearly all cancer sites (Jemal et al. 2008). Results of other studies that have examined the relationship between race and socioeconomic status (SES) and cause-specific mortality, including causes considered...
medically amenable versus those that are not (Phelan et al. 2004), are also consistent with the predictions of fundamental cause theory (Macinko and Elo 2009; Phelan and Link 2005).

If access to resources and medical technology play an important role in the above findings, we would expect that the relationship between race, SES and cancer screening would also have changed over time. In this paper, we examine whether aggregate-level patterns of mortality decline are consistent with the timing of widespread adoption of screening technologies and the associations between race, SES and cancer screening and mortality at the individual level using data from the National Health Interview Surveys (NHIS).

Data and Methods

We explore black-white mortality differentials for three groups of cancers: 1) tobacco-related, 2) screening-related, and 3) less medically amenable cancers (see Table 1 for the listing of cancers in each category). Using aggregate-level data, we will examine how trends in mortality differences from screening-related cancers match up to the timing of widespread screening, how the trends in mortality differences from tobacco-related cancers match up to differences in cohort smoking histories, and how trends for groups (1) and (2) compare to trends in mortality from less medically amenable cancers.

In the individual-level mortality analysis, we focus on cancers for which screening and early detection are important for reducing the risk of death (breast, cervical, colorectal, and prostate) and cancers linked to smoking (lung, bronchus, trachea, lip, larynx, bladder, esophagus). We use data from the NHIS, which have included questions about the receipt of various screening procedures for preventable cancers in several years between 1987 and 2010. The NHIS is representative of the civilian noninstitutionalized population and, importantly for our study, oversamples blacks, Hispanics, and Asians. These data (through NHIS 2004) have also been linked to the National Death Index (NDI) through 2006, achieving a lengthy mortality follow-up that closely corresponds to the experience of the general U.S. population (Ingram, Lochner, and Cox 2008), and can be used to examine predictors of cancer mortality.

The NHIS provides information on race/ethnicity, education, family income, smoking behavior, regular source of care and health insurance coverage. We hypothesize that screening varies by race/ethnicity and SES, that these differentials have declined over time as screening has become more common, and that differentials by race/ethnicity can be largely explained by SES, regular source of care and health insurance coverage. However, because the NHIS does not include information on stage at
We further hypothesize that a large fraction of the variation in cancer mortality by race/ethnicity can be explained by SES, access to a regular source of care and health insurance coverage. In order to test these hypotheses, we will use Cox proportional hazards and discrete-time hazard models. Table 2 lists the known risk factors for cancers for which effective screening exists. We will be able to control for some of them, such as cigarette smoking and body mass index, in our analysis. We also plan to extend this analysis by considering Hispanics. Aggregate data on Hispanic ethnicity is only available after 1999; however, we will be able to explore whether the determinants of cancer screening and mortality differ between Hispanics and non-Hispanics for the full study period using the NHIS data.

**Preliminary Results**

Figures 1-6 show trends in mortality for six cancer sites from 1979-2007 for blacks and whites, differentiating between Hispanics and non-Hispanics when the data allow (1999-onwards). For breast, colorectal, and prostate cancer, three cancer sites for which effective screening exists, we observe a widening in the black-white mortality differential (Figures 1, 3, and 4). In general, the timing of increases in the black-white mortality gap appear to be consistent with the introduction of screening technologies, allowing for a reasonable lag period. The narrowing black-white mortality gap in cervical cancer mortality may be expected since Pap smear was introduced several decades earlier than the other screening technologies (the 1940s rather than the 1980s, see Table 2). Cancer of the lung, bronchus, and trachea is the key example of tobacco-related cancer. For males, death rates peak in the early 1990s and are higher for blacks than whites throughout the period, and for females, death rates have continued to rise and are slightly higher for whites than for blacks (Figure 5). These patterns are consistent with what is known about cohort smoking histories and differences in smoking cessation rates between blacks and whites (Burns et al. 1997). Figure 6 shows age-adjusted death rates for lymphoid leukemia, a cancer that

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1. While stage at diagnosis is an important contributor to black-white differences in cancer mortality, it does not entirely account for those differences. Within each stage of diagnosis for almost all cancer sites, five-year relative survival is lower for blacks than for whites, and survival differences persist after controlling for stage at diagnosis (Clegg et al. 2002; Shavers and Brown 2002).

2. Data for Hispanic blacks may be unreliable due to small subpopulation sizes.
was considered less medically amenable in the U.S. in the 1980s and for which effective screening does not exist (Phelan et al. 2004). For both males and females, blacks and whites experience similar mortality rates, and the trend is fairly flat through the entire time period.

In our next steps, we will use individual-level data from the NHIS to test our hypotheses regarding the associations between race, SES and cancer screening and mortality.

References


Figures and Tables

**Figure 1.** Age-Standardized Breast Cancer Death Rate by Race/Ethnicity, Females 35+, 1979-2007

**Figure 2.** Age-Standardized Cervical Cancer Death Rate by Race/Ethnicity, Females 35+, 1979-2007
Figure 3a. Age-Standardized Colorectal Cancer Death Rate by Race/Ethnicity, Females 35+, 1979-2007

Figure 3b. Age-Standardized Colorectal Cancer Death Rate by Race/Ethnicity, Males 35+, 1979-2007
Figure 4. Age-Standardized Prostate Cancer Death Rate by Race/Ethnicity, Males 35+, 1979-2007

Figure 5a. Age-Standardized Lung Cancer Death Rate by Race/Ethnicity, Females 35+, 1979-2007

3 Includes cancers of the lung, bronchus, and trachea
Figure 5b. Age-Standardized Lung Cancer Death Rate by Race/Ethnicity, Males 35+, 1979-2007

Figure 6a. Age-Standardized Lymphoid Leukemia Death Rate by Race/Ethnicity, Females 35+, 1979-2007

4 Includes cancers of the lung, bronchus, and trachea
Table 1. Grouping of Cancers

<table>
<thead>
<tr>
<th>Screening-Related</th>
<th>Tobacco-Related</th>
<th>Less Amenable&lt;sup&gt;5&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast</td>
<td>Bladder</td>
<td>Brain</td>
</tr>
<tr>
<td>Cervix</td>
<td>Esophagus</td>
<td>Gallbladder and extrahepatic bile ducts</td>
</tr>
<tr>
<td>Colorectal</td>
<td>Larynx</td>
<td>Heart and mediastinum</td>
</tr>
<tr>
<td>Prostate</td>
<td>Lip</td>
<td>Lymphoid leukemia</td>
</tr>
<tr>
<td></td>
<td>Lung, bronchus, and trachea</td>
<td>Ovary</td>
</tr>
</tbody>
</table>

<sup>5</sup> Taken from Phelan et al. (2004), based on the degree to which deaths were amenable in the United States during the 1980s.
<table>
<thead>
<tr>
<th>Cancer Sites</th>
<th>Risk Factors</th>
<th>Screening</th>
<th>Treatment</th>
<th>Relative 5-Year Survival Rate</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco-related</td>
<td>Lung, trachea, bronchus, lip, larynx, bladder, esophagus</td>
<td>Tobacco, occupational exposure (radiation or asbestos), diet (low fruit and vegetable intake), family history</td>
<td>Chest x-rays, sputum cytology, spiral/helical CT scans of the lungs</td>
<td>Surgery, chemotherapy, radiation</td>
<td>12&lt;sup&gt;5&lt;/sup&gt; 15&lt;sup&gt;5&lt;/sup&gt;</td>
</tr>
<tr>
<td>Colorectal</td>
<td>Obesity, inflammatory bowel disease, family history, lack of physical activity, diet (low fruit and vegetable intake or low fiber and high-fat), alcohol and tobacco use</td>
<td>Colonoscopy, sigmoidoscopy, fecal occult blood test (FOBT)</td>
<td>Colonoscopy was approved for use in 1978.</td>
<td>Surgery, chemotherapy, radiation</td>
<td>55 65</td>
</tr>
<tr>
<td>Breast</td>
<td>Age, ages at menarche, first birth, and menopause, number of births, family history, ionizing radiation, HRT and oral contraceptive use, obesity, lack of physical activity</td>
<td>Mammography, clinical breast exam, breast self-exam</td>
<td>Mammography usage started in the 1960s; widespread use began in the early 1980s.</td>
<td>Surgery, chemotherapy, radiation, hormonal therapy</td>
<td>77 90</td>
</tr>
<tr>
<td>Prostate</td>
<td>Age, family history, race</td>
<td>Prostate specific antigen test (PSA), digital rectal exam (DRE)</td>
<td>PSA was approved for use in 1986.</td>
<td>Surgery, chemotherapy, radiation, hormonal therapy</td>
<td>95 99</td>
</tr>
<tr>
<td>Cervix</td>
<td>HPV, HIV, smoking, oral contraceptive use, having 3 or more children</td>
<td>Pap smear</td>
<td>The Pap smear was introduced in the U.S. in the 1940s.</td>
<td>Surgery, chemotherapy, radiation</td>
<td>62 73</td>
</tr>
</tbody>
</table>

<sup>2</sup> Not available for Hispanics.
<sup>3</sup> Effective screening for tobacco-related cancers is relatively limited. Lung cancer screening has been found to lead to the detection of cancer at earlier stages, but not to improvements in survival (Cutler 2008).
<sup>4</sup> Lung cancer is often detected at later stages and therapy is not highly effective; medical care is often palliative.
<sup>5</sup> Applies to cancer of the lung and bronchus.