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A fundamental cause approach to the study of disparities in lung cancer and pancreatic cancer mortality in the United States

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ABSTRACT

This study examines how associations between socioeconomic status (SES) and lung and pancreatic cancer mortality have changed over time in the U.S. The fundamental cause hypothesis predicts as diseases become more preventable due to innovation in medical knowledge or technology, individuals with greater access to resources will disproportionately benefit, triggering the formation or worsening of health disparities along social cleavages. We examine socioeconomic disparities in mortality due to lung cancer, a disease that became increasingly preventable with the development and dissemination of knowledge of the causal link between smoking cigarettes and lung cancer, and compare it to that of pancreatic cancer, a disease for which there have been no major prevention or treatment innovations. County-level disease-specific mortality rates for those ≥45 years, adjusted for sex, race, and age during 1968–2009 are derived from death certificate and population data from the National Center for Health Statistics. SES is measured using five county-level variables from four decennial censuses, interpolating values for intercensal years. Negative binomial regression was used to model mortality. Results suggest the impact of SES on lung cancer mortality increases 0.5% per year during this period. Although lung cancer mortality rates are initially higher in higher SES counties, by 1980 persons in lower SES counties are at greater risk and by 2009 the difference in mortality between counties with SES one SD above compared to one SD below average was 33 people per 100,000. In contrast, we find a small but significant reverse SES gradient in pancreatic cancer mortality that does not change over time. These data support the fundamental cause hypothesis: social conditions influencing access to resources more greatly impact mortality when preventative knowledge exists. Public health interventions and policies should facilitate more equitable distribution of new health-enhancing knowledge and faster uptake and utilization among lower SES groups.

Introduction

How do we understand why large socioeconomic disparities have emerged over the past few decades for some major causes of death and not others? One line of thinking focuses attention on the grinding, self-reinforcing, accumulating, wear and tear that the deprivation of lower socioeconomic status (SES) imposes on those who encounter it. This stress-based explanation is clearly critically important for understanding the prominence and persistence of socioeconomic gradients in morbidity and mortality. At the same time, the main mechanistic explanation it provides is one that should express itself in the same way at different times. If the wear and tear of low SES is evident among current cohorts and was also evident among cohorts 30 years ago, we would expect, given this explanation, that SES gradients in mortality would be evident at both time periods. Additionally, Krieger, Chen, Kosheleva, and Waterman (2012) note that while it is true that SES is associated with many causes of death, it is also true that there are some for which the gradient is much smaller, is narrowing, or may even be non-existent. Why should this be so? We propose that the theory of fundamental social causes, which offers a framework for making predictions for when disparities in health outcomes may arise, can help us understand why socioeconomic gradients vary across time and between causes of death. In particular, we examine whether we can expect disparities to emerge when life-enhancing information is produced that is not evenly distributed or able to be acted on

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across socioeconomic strata and conversely whether we can expect the absence of disparities when our capacity to prevent death is limited.

To exemplify how, we first provide an overview of the theory of fundamental social causes and the types of predictions the theory makes regarding socioeconomic inequalities in mortality. Next, taking a comparative case-study approach, we use fundamental cause theory to examine two contrasting causes of death, one that is susceptible to human intervention and one that is not. Specifically, we examine lung cancer, currently the largest cause of cancer death both throughout the world (GLOBOCAN, 2008) as well as in the U.S. (National Cancer Institute, 2010), and pancreatic cancer, currently the fourth largest cause of cancer death in the U.S. (National Cancer Institute, 2010). As we will see, lung cancer became largely preventable through smoking avoidance, while pancreatic cancer generally remained impervious to human intervention, thus providing a useful and interesting comparison to test the relevance of fundamental cause theory.

Fundamental causes

In their original statement of the theory, Link and Phelan (1995) argue that social inequalities in mortality are large, persistent, and growing because socioeconomic status acts as a "fundamental cause" of inequalities in health. According to the fundamental cause theory, SES operates in this way because those with greater access to resources are better able to deploy those resources to avoid poor health outcomes, including mortality, than those with less access to resources. Phelan, Link, Diez-Roux, Kawachi, and Levin (2004) note that preventative knowledge is an important catalyst for SES inequalities in mortality: as we learn more about how to prevent or treat disease, the benefits of this new knowledge are not distributed equally throughout the population, but are instead harnessed more securely by individuals and groups who are less likely to be exposed to discrimination and who have greater access to resources such as knowledge, money, power, prestige, and beneficial social connections. This triggers the formation or exacerbation of health disparities along typical social cleavages such as SES (Link & Phelan, 1995).

The fundamental cause theory offers a framework for making predictions for when disparities in health outcomes may arise. We apply this framework to understand how socioeconomic inequalities in lung and pancreatic cancer mortality have changed over time. In particular, for lung cancer, we examine the mortality trajectories in light of the fact that the disease became largely preventable with the development and dissemination of knowledge of the causal link between smoking cigarettes and lung cancer. Under the fundamental cause theory, for a disease like lung cancer for which a major cause of disease was discovered, we would expect that those with greater access to resources would be in a better position to be aware of the new information and also be able to take action to change health-related behaviors based on the new information compared to those with less access to resources.

In addition, Freese and Lutfey (2011) emphasize that socioeconomic inequality impacts health because individuals exist within social contexts. Thus, they highlight "spillover" effects, which occur, for example, when individuals, regardless of their individual behaviors, benefit from the actions of their social relations. Considering smoking, for example, individuals with higher SES are less likely to start and more likely to quit smoking in response to their social context, even if they are indifferent to their own health. The converse is also true: an individual's exposure to smoking is influenced by his or her proximity to others who smoke. For example, if an individual stops smoking but his household contacts still smoke, he will still be exposed to secondhand smoke.

In accordance with fundamental cause theory, we would therefore expect lung cancer mortality rates to decrease earlier in more advantaged populations and for socioeconomic disparities in lung cancer mortality to emerge or worsen. In contrast, for a disease like pancreatic cancer, for which there have been no major developments in prevention or treatment, we would expect no dramatic changes in mortality rates and no major differences between groups to emerge over time as belonging to an advantaged group should confer no major mortality benefit when there are no innovations.

While fundamental cause theory offers a framework for making predictions under different general disease prevention and treatment scenarios, it is also important to understand the particular circumstances surrounding a given disease. Therefore, we use a case study approach to investigate lung cancer and pancreatic cancer as causes of death, whether there were advances that occurred in the prevention or treatment of these two diseases, and the timing of any advances. As detailed below, while we find that there was a building of knowledge and awareness of how to prevent lung cancer, no major innovations in the prevention or treatment of pancreatic cancer occurred. Our findings then serve as the basis for the hypotheses we test.

Case studies: lung and pancreatic cancer

Claims of the harmful effects of tobacco on health in general date back centuries (Harley, 1993; Redmond, 1970; Walker, 1980) and anecdotal reports of a potential link between tobacco and lung cancer in particular date back to the early 1900s (Tylecote, 1927; Wynder & Graham, 1950). However, epidemiological and scientific support for a casual relationship between smoking tobacco and developing lung cancer built gradually as early case series examinations of the 1920s and 1930s in Europe gave way to 1930s and 1940s case–control studies (also initially in Europe) with increasingly more rigorous methodology, and eventually prospective cohort studies. Evidence linking smoking and lung cancer especially grew during the 1950s. For example, in 1950, four case–control studies were published that found an association between smoking and lung cancer (Doll & Hill, 1950; Levin, Goldstein, & Gerhardt, 1950; Schrek et al., 1950; Wynder & Graham, 1950). By this time, the lung cancer mortality rate among US men had more than quadrupled since the 1920s, mirroring the pattern of increased tobacco consumption with an approximately 20 year lag (Warner & Robert Wood Johnson Foundation, 2006).

Publications in the lay press helped to bring the body of evidence of the association between tobacco and lung cancer building (and debated (Doll & Hill, 1999)) in the biomedical literature to the general public. For example, in December 1952, *Reader's Digest* published an article entitled, "Cancer by the Carton," which highlighted medical research on the relationship between smoking and lung cancer and discussed the hazards of smoking (Norr, 1952). The piece noted the "suspicious parallel" between the increase in cigarette consumption and the increase in deaths due to lung cancer (Norr, 1952, p. 738). Similarly, in 1953, Time Magazine's article, "Beyond Any Doubt," (1953) reported that accumulating data suggested that while no known carcinogen existed in cigarettes, those who got and ultimately died from lung cancer were mostly long-time cigarette smokers.

Corroborating the findings of case–control studies, in 1954 and 1956, Doll and Hill published results of a prospective cohort study of medical doctors in the United Kingdom (Doll & Hill, 1954, 1956), which demonstrated a direct, dose–response relationship between the amount smoked and the lung cancer mortality rate. Furthermore, because of the similar smoking habits among doctors in urban and rural areas and the robust dose–response relationship