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Distal and variably proximal causes: Education, obesity, and health

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ABSTRACT

Medical sociologists hold that social conditions generate disparities across a host of health conditions through exposure to a variety of more proximate risk factors. Though distal and proximal causes jointly influence disease, the nature of risk accumulation may differ appreciably by the link of a proximal cause to the outcome in question. This paper employs a representative sample of over 3000 American older adults to examine whether position in the educational gradient amplifies the effect of obesity on two health outcomes. Results indicate that educational inequalities amplify the effect of high body mass index on disability (unstandardized coefficients across education groups range from −.05 [ns] to .26 [$p < .01$] among overweight respondents yet reach .17 [ns] to .73 [$p < .001$] among severely obese adults), but fail to amplify the consequences of severe obesity in the case of C-reactive protein (CRP) levels. Instead, educational gradients in CRP are most pronounced at lower levels of body mass. Sex-specific analyses further clarify these patterns, as the connections between CRP and body mass are particularly strong among women. We conclude that risk accumulation processes differ based on the proximity of causes to the health outcome under examination.

Introduction

One of medical sociology's most cogent insights is that social conditions are a distal but fundamental cause of disease (Link & Phelan, 1995; Lutfey & Freese, 2005; Phelan, Link, Diezroux, Kawachi, & Levin, 2004). Understanding the root conditions that generate health disparities in a population has provided an important corrective to the dominant biomedical emphasis on proximate causes of disease. The fecundity of this insight has been borne out by recent efforts that showcase the primacy of social class in shaping population exposure to disease risk, both in dynamic historical context (Chang & Lauderdale, 2009) and above and beyond competing, alternative fundamental causes (Link, Phelan, Miech, & Westin, 2008).

At the same time, however, our field is less clear on the *interplay between distal factors and more proximate causes*, such as how risks accumulate for people with high status but poor health lifestyles versus materially disadvantaged people with healthy lifestyles. The perspectives of cumulative advantage/disadvantage and "double jeopardy" are hinged on the concept of risk accumulation but seldom make much of the distinction between distal and proximal risk factors (c.f., Singh-Manoux, Clarke, & Marmot, 2002). Further, proximate causes themselves vary in their proximity to a health outcomes. Some proximate causes have a tight, causal biological link to a health outcome and explain much of its variation, whereas others play a looser, far less etiologic role.

To make the case more concretely, consider smoking; the carcinogens in cigarette smoke react with proteins to form adducts in organ tissue (Phillips, Hewer, Martin, Garner, & King, 1988). Thus, smoking is an *extremely* proximate risk factor for adduct levels in the lung. Some proximate causes, though closer in a causal sense than more distal social conditions, remain further away on the etiological pathway to sickness or disease. Keeping with the same example, smoking would likewise serve as a proximate risk factor for mortality, but the proximity is far more removed than when we were considering adducts. While nicotine exposure is assuredly a hazard, there are many other steps (biological and non-biological) along the way before smoking results in death. We view this issue, *differences in risk proximity*, as an opportunity to investigate competing models of risk accumulation in the interaction of distal social causes and more proximal biological risk factors. Is the conjoint influence of these factors conditional on the outcome in some systematic way? And how do distal causes condition the effect of known proximate causes when risk proximity itself is variable?

In undertaking these questions we focus on body mass index (BMI) as a significant health risk factor, recognizing that high body

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mass is (1) a non-trivial public health issue of substantive interest for medical sociologists and (2) related to a broad range of health conditions.

Accordingly, we assess BMI's relationship with two health outcomes conjointly with educational attainment, a central element of socioeconomic status (SES) and a key axis of social inequality. Educational disparities and BMI are each associated with numerous health risks that are evident across a variety of bodily systems. Reflecting distal and proximate causes, respectively, educational attainment and BMI are negatively associated in most developed societies (Wang & Beydoun, 2007), and each risk exerts an independent effect on health net of the other (Borg & Kristensen, 2000).

When considered jointly, however, results tend to be less consistent. Most evidence reveals that social disadvantage amplifies the negative effect of other, more proximate risk factors (e.g., Rask, O'Malley, & Druss, 2009), but other studies show that the social gradient varies across health indicators such that high status groups may be at higher risk for selected outcomes (e.g., Rosero-Bixby & Dow, 2009). The purpose of this study is to examine the set of conditions under which educational gradients *amplify* the health risks of high BMI and compare them to the scenarios where the effects of education fail to amplify the independent health risks of high BMI. We focus on two outcomes with varying proximity to BMI—C-reactive protein and disability—in hopes of demonstrating that amplification effects depend in part upon the proximity of the cause to the health problem in question.

Competing models of SES and health risk

Conceptualizing risks as both distal and proximal is becoming more common in medical sociology and social epidemiology. Link and Phelan's (1995) *fundamental cause theory*, for instance, articulates that social class is not merely a placeholder variable that stands as a proxy for the "real" causes of illness and disease. Rather, social conditions are causally generative of population health patterns by initiating a multiplexity of mechanisms (Lutfey & Freese, 2005). This insight motivates interest in understanding social structure and legitimates distal factors as important for the study of human health. Social epidemiological risk factor accumulation models further extend this perspective. For instance, rather than treating key proximal risks as control variables in the relationship of SES and health, an alternate route of inquiry considers the conjoint influence of distal and proximal risks (Smith & Hart, 2002).

One explanation of how distal and proximal risks accumulate interactively is that people with low education may be more vulnerable to the results of health-compromising behaviors or endogenous risk factors than are highly-educated people. We refer to this model as *amplified risk accumulation*. With more compensatory mechanisms at their disposal, people with high levels of education could counteract much of the harm caused by known cardiovascular risk factors (e.g., high body mass). For people with low education, however, exposure to the varied risks associated with their social standing would generate additional disadvantage with each accruing proximate risk (e.g., high BMI).

Several empirical studies also support this model of risk accumulation. Pampel and Rogers (2004), for instance, found a positive interaction between social status and smoking status for morbidity risk: the risk of low social standing exacerbates the damaging health consequences of smoking cigarettes. Krueger and Chang (2008) document a similar pattern, showing that the deleterious effects of physical inactivity are much starker among the poor than those with greater resources. Likewise, the mortality and hospitalization consequences of heavy drinking are magnified among manual workers relative to non-manual workers (Makela & Paljarvi, 2008).

An alternative model, likewise premised on the logic of conjoint effects, anticipates that educated people would stand to lose the most from health-damaging choices or endogenous biological risks (e.g., high BMI). This specification is based upon the recognition that people of high education benefit from a variety of assets, including better access to exercise and good nutrition, a sophisticated understanding of medical care, and safer work conditions, among other advantages. In contrast, each additional risk may exert diminishing returns among less advantaged people, as they have "less to lose" once other health disadvantages are taken into account (Blaxter, 1990; Pampel & Rogers, 2004). Under this scenario, education gradients taper off at the high end of the BMI distribution because the person's risk factors approach saturation. Following Pampel and Rogers (2004), this can be referred to as the *Blaxter hypothesis,* as it is derived from Mildred Blaxter's specification in Health and Lifestyles (1990).

The Blaxter thesis has received limited support from subsequent research. Some corroborating evidence has come from studies using aggregated neighborhood-level data (Duncan, Jones, & Moon, 1993), but more recent studies have failed to support it (Krueger & Chang, 2008; Pampel & Rogers, 2004). That being said, relatively few studies examine conjoint effects between distal social causes and more proximal biological or behavioral ones.

Of course, a third and very common approach to studying the accumulation of risk factors is premised on an additive model. From this perspective, education is associated with neither amplified nor diminished returns to a risk factor (Christiansen & Kooiker, 1999; Marang-van de Mheen, Smith, & Hart, 1999). Pampel and Rogers (2004) find this pattern when examining ascribed statuses (i.e., race and ethnicity), in that racial minorities are at increased risk of mortality, but being of minority status does not alter the effect of smoking.

It is possible that educational attainment could amplify, weaken, or be irrelevant for the effect of high BMI on health outcomes. We provide a systematic test of these possibilities on two health outcomes that were selected to illustrate proximal and distal associations with BMI. We expect that amplification is more likely for outcomes reflecting distal rather than proximal associations.

Obesity and health outcomes

C-reactive protein (CRP)

The first outcome that we examine is a measure with a direct, physiological link to BMI. In recent years, the integration of biomeasures into survey research has opened a new vista for understanding how risk factors independently and jointly exact a toll on health. Capitalizing on this development, we examine CRP, a nonspecific marker of inflammation that is a key biomeasure for cardiovascular disease risk for both men and women (Ridker, Cushman, Stampfer, Track, & Hennekens, 1997; Ridker, Hennekens, Buring, & Rifai, 2000). Released as a part of the large systemic immune cascade in response to infection or injury, CRP is regulated by proinflammatory cytokines, including tumor necrosis factor-α and interleukin-6 (Tamakoshi et al., 2003).

Heightened CRP levels are also associated with obesity and contribute to the concern about why obesity is a danger for the development of metabolic syndrome and cardiovascular disease. An emergent literature documents that adipose tissue is related to inflammatory biomarkers such as CRP; and this relationship has been reported in a variety of samples (e.g., Wisse, 2004). Although adipocytes, the cells that make up fat, were long considered to function mainly as passive energy stores, more recent research