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Income inequality and sexually transmitted in the United States: Who bears the burden?

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ABSTRACT

Three causal processes have been proposed to explain associations between group income inequality and individual health outcomes, each of which implies health effects for different segments of the population. We present a novel conceptual and analytic framework for the quantitative evaluation of these pathways, assessing the contribution of: (i) absolute deprivation – affecting the poor in all settings – using family income; (ii) structural inequality – affecting all those in unequal settings – using the Gini coefficient; and (iii) relative deprivation – affecting only the poor in unequal settings – using the Yitzhaki index. We conceptualize relative deprivation as the interaction of absolute deprivation and structural inequality. We test our approach using hierarchical models of 11,183 individuals in the National Longitudinal Study of Adolescent Health (Add Health). We examine the relationship between school-level inequality and sexually transmitted infections (STI) – self-reported or laboratory-confirmed Chlamydia, Gonorrhoea or Trichomoniasis. Results suggest that increased poverty and inequality were both independently associated with STI diagnosis, and that being poor in an unequal community imposed an additional risk. However, the effects of inequality and relative deprivation were confounded by individuals' race/ethnicity.

Introduction

There is a growing body of evidence that higher levels of income inequality both across and within countries are associated with worse health outcomes (Kondo et al., 2009; Wilkinson & Pickett, 2006). Multiple causal processes relating income inequality to health have been proposed (Kawachi, 2000), but there is considerable debate as to whether these relationships are truly causal, and if so how the mechanisms might vary by health outcome (Deaton, 2003; Gravelle, 1998).

Theoretically, socioeconomic status (SES) can pattern sexually transmitted infection (STI) risk in multiple ways. These include affecting whom one selects as a partner, and thus how likely the partner is to be infectious, and the actions an individual takes within a relationship (e.g. number of partners, sexual behaviour with each partner) (Bärnighausen & Tanser, 2009; Boerma & Weir, 2005; Poundstone, Strathdee, & Celentano, 2004).

Causal mechanisms that explain an empirical association between inequality and health have been divided into three broad categories (Leigh, Jencks, & Smeeding, 2009; Nilsson, 2009), each of which implies different segments of the community will be affected. It is possible to envisage pathways leading from inequality to STIs that are specific to each category.

Causal mechanisms linking inequality to STIs

First, the absolute deprivation hypothesis (ADH) posits that inequality is associated with ill-health for the poorest through its relationship with the distribution of income in a community. Ceteris paribus, more unequal communities will have more individuals living at both high and low income levels than similar, more equal communities. Given the empirically observed concave relationship between resources and health, such that there are positive but diminishing marginal returns health returns to additional income, greater inequality at any given mean level of income thus leads to lower average health, since decreased income harms the poor in the unequal community more than increased income benefits the rich (Leigh et al., 2009).

ADH mechanisms relating inequality to STIs might include access to sexual health education and care, which are likely to be lower for poorer individuals, and partner choices – since income affects where one can afford to socialize. If both mechanisms act in

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concert, differential distribution of knowledge and care resources across social strata would reinforce high STI rates amongst the poorest.

Second, the structural inequality hypothesis (SIH) focuses on the idea that the structure of unequal societies harms the health of everyone within them. Greater inequality may cause weaker social bonds and less social cohesion. Weak social ties can lead to lower levels of public good provision, either due to failure to work together to secure such goods or because community members have less in common, thus lowering the likelihood of a majority supporting provision of any given good (Kawachi & Berkman, 2000; Leigh et al., 2009).

Additionally, heterogeneity in economic circumstances may reduce interaction between community members. This can affect health by reducing the diffusion of healthy behaviours, limiting informal social control of unhealthy behaviours or by generating distrust leading to increased anxiety or depression – each of which can lead to poorer physiological and behavioural outcomes (Kawachi & Berkman, 2000; Kubzansky & Kawachi, 2000). For example, if lower social bonds lead to increased propensity to commit crime against others, this might lower physical mobility within neighbourhoods, increase stress levels and of course increase violence-related ill-health.

Community heterogeneity may also ensure increased assortativity of social mixing by increasing the proportion of unlike people who are socially or geographically proximate. This may lead to increased diffusion of behaviours or infections, if disease prevalence or health behaviours differ systematically by social groups. The direction of effect in such a situation is ambiguous, since those with poorer health might impact the healthier, or vice versa. In the case of infectious diseases, mixing of heterogeneous risk groups has been shown theoretically to lead to slower disease spread but ultimately higher total prevalence (Doherty, Shiboski, Ellen, Adimora, & Padian, 2006; Garnett & Anderson, 1996).

SIH mechanisms might include reduced provision of sexual health services – perhaps because there is inadequate funding to support their provision. Alternatively, differential sexual mixing patterns, specifically more mixing between high and low SES groups in more unequal communities, would increase the spread of STIs across all social strata within these communities.

Finally, the relative deprivation hypothesis (RDH) suggests that inequality affects the worst-off in unequal communities uniquely, by increasing their social distance from their relevant reference group – others living nearby (Spriggs, Halpern, Herring, & Schoenbach, 2009). This reference group provides an expectation for normative living standards and behaviours (Runciman, 1966; Webber, 2007). When the worse-off are unable to achieve this standard of living due to limited resources, the resulting stress and shame may lead to worse health through either psychosocial or behavioural pathways (Kondo, Kawachi, Subramanian, Takeda, & Yamagata, 2008). RDH mechanisms linking relative deprivation to STIs seem most likely to arise through behavioural mechanisms. For example, the perceived gap between individuals and their better-off neighbours could lead to increased use of alcohol and other substances, leading in turn to more risky sexual behaviour.

It is notable that each set of mechanisms predicts that different segments of the population will be affected (Fig. 1). ADH mechanisms will put the poor at increased risk relative to the rich, regardless of the level of community inequality. SIH mechanisms place all those living in more unequal communities at increased risk, relative to those living in more equal communities. Finally, RDH mechanisms will affect only the poor within unequal communities. Of course, some, all, or none of these mechanisms may be present in a given context. Understanding where the burden lies is essential for careful targeting of prevention and treatment interventions.

	Non-poor Family	Poor Family
Equal Community		Absolute deprivation
Unequal Community	Structural inequality	Absolute deprivation Structural inequality Relative deprivation

Fig. 1. Conceptual map of economic disadvantages.

Empirical links between SES and STIs

Empirically, sexual behaviour and STI rates have been shown to be associated with socioeconomic conditions at the individual and group levels in North America and Europe, both due to individual factors such as poverty and education, and group-level factors such as high neighbourhood poverty, deprivation and social disorganization, and low social capital and collective efficacy (Bauermeister, Zimmerman, & Caldwell, 2011; Browning, Leventhal, & Brooks-Gunn, 2004; Dupéré, Lacourse, Willms, Leventhal, & Tremblay, 2008; Hogben & Leichliter, 2008; Holtgrave & Crosby, 2003; Krieger, Waterman, Chen, Soobader, & Subramanian, 2003; Ramirez-Valles, Zimmerman, & Newcomb, 1998).

These associations are particularly notable amongst women (Zierler & Krieger, 1997) and in the African-American community (Adimora & Schoenbach, 2002). The effect-modification of SES by race/ethnicity and gender is unsurprising, given that both factors strongly pattern partner choices, sexual experiences and STI risk (Adimora & Schoenbach, 2005). Previous studies of the association of STIs with education and poverty have found effects to vary by race and gender (Annang, Walsemann, Maitra, & Kerr, 2010; Newbern, Miller, Schoenbach, & Kaufman, 2004).

Research on the relationship between income inequality and STIs in the United States has been limited. To date, the only two analyses have been ecological: one of neighbourhood-level inequality in Massachusetts and Rhode Island (Krieger et al., 2003) and the other of state-level inequality nationally (Holtgrave & Crosby, 2003). Both studies found inequality to be positively associated with reported STI rates.

In the present study we examined whether the three mechanisms explain any association between income inequality and STI acquisition risk amongst young adults in the United States. To do this we propose a novel approach which links commonly used economic measures to specific theoretical causal mechanisms.

Methods

We conducted a secondary data analysis using Waves I–III of the National Longitudinal Survey of Adolescent Health (Add Health). Add Health is a nationwide survey which sampled adolescents from 80 US high schools and 52 of these schools' largest feeder schools (Harris et al., 2009), and followed them into young adulthood. Schools were selected so as to ensure coverage across regions, levels of urbanicity, school sizes and types, and race/ethnicity. Wave I (1994–95) surveyed a sample of all enrolled students in grades 7 through 12 at home, Wave II (1996) re-surveyed those who had been in grades 7 through 11 at Wave I, and Wave III (2001–02; ages 18–26) re-interviewed all Wave I respondents. Understanding sexual behaviour and health was one of the primary interests in the design of Add Health (Resnick et al., 1997).