Title: The Social Epidemiology of Tuberculosis in South Africa: A Multilevel Analysis

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Abstract

Increased risk of tuberculosis is widely recognized to be associated with increased poverty, yet there have been few analyses of the social determinants of tuberculosis, particularly in high-burden settings. We conducted a multilevel analysis of self-reported tuberculosis disease in a nationally-representative sample of South Africans based on the 1998 Demographic and Health Survey (DHS). Individual- and household-level demographic, socio-behavioral and socioeconomic risk factors were taken from the DHS; data on community-level socioeconomic status (including measures of absolute wealth and income inequality) were derived from the 1996 national census. Of the 13,043 DHS respondents, 0.5% reported having been diagnosed with tuberculosis disease in the past 12 months and 2.8% reported having been diagnosed with tuberculosis disease in their lifetime. In a multivariate model adjusting for demographic and behavioral risk factors, tuberculosis diagnosis was associated with cigarette smoking, alcohol consumption and low body mass index, as well as lower levels of personal education, unemployment and reduced household wealth. In a model including individual- and household-level risk factors, high levels of community income inequality were independently associated with increased prevalence of tuberculosis (adjusted odds ratio for lifetime tuberculosis comparing the most unequal quintile to the middle quintile of inequality: 2.37, 95% confidence interval: 1.59-3.53). These results provide novel insights into the socioeconomic determinants of tuberculosis in developing-country settings, although the mechanisms through which income inequality may affect tuberculosis disease require further investigation.

Introduction

Tuberculosis (TB) is the world's leading curable cause of infectious disease mortality, with a disproportionate burden of disease falling on low- and middle-income countries (World Health Organisation, 1999). Globally the incidence of tuberculosis is increasing, fueled in part by the concurrent epidemic of HIV/AIDS, particularly in sub-Saharan Africa. Tuberculosis is traditionally regarded as a disease of poverty and many aspects of low socioeconomic status (SES), for example overcrowding and malnutrition, are accepted individual-level risk factors for the disease. The distribution of tuberculosis within populations has also been historically associated with group-level socioeconomic factors, including industrialization, globalization and structural adjustment programs, as reflected in inadequate social conditions for the poorest in society (Dubos & Dubos, 1992; Gandy & Zumla, 2003; McKeown, 1976).

South Africa has a long history of high tuberculosis burden, and currently the tuberculosis incidence of over 550 cases per 100,000 per annum is the second highest in the world (Maher & Raviglione, 2005). As in other countries, the tuberculosis epidemic in South Africa can be traced in part to the poor working and living conditions that surrounded early industrialization. Many of these conditions persist to this day, and along with the HIV/AIDS epidemic, have fueled the current high levels of tuberculosis disease in this country (Packard, 1989).

Despite the acknowledgement that tuberculosis has strong socioeconomic determinants, there has been surprisingly little epidemiological research into the pathways through which SES might increase the risk of tuberculosis. Ecologic studies conducted in the United States and Britain have found crude associations between tuberculosis rates across areas and low levels of education, high levels of poverty, government social support, social deprivation and income inequality (Krieger, Waterman, Chen, Soobader & Subramanian

2003; Parslow, El-Shimy, Cundall & McKinney, 2001; Spence, Hotchkiss, Williams & Davies, 1993; Tocque, Regan, Remmington, Beeching, Jamieson, Syed et al., 1999). Evidence from ecologic studies in Brazil and South Africa supports the existence of this relationship in middle-income countries (Munch, Van Lill, Booysen, Zietsman, Enarson & Beyers, 2003; Souza, Ximenes, Albuquerque, Lapa, Portugal, Lima et al., 2000). Finally, individual-level studies of the link between low SES and high risk of tuberculosis have found associations in poorer, high tuberculosis-burden settings, such as southern India and Guinea-Bissau (Gustafson, Gomes, Vieira, Rabna, Seng, Johansson et al., 2004; Shetty, Shemko, Vaz & D'Souza, 2006), although not in Malawi (Glynn, Warndorff, Malema, Mwinuka, Pönnighaus, Crampin, et al., 2000).

Recent decades have seen increasing attention in public health paid to quantitative study of social and economic factors as determinants of health outcomes. This field of social epidemiology recognizes the importance of both individual- and group-level socioeconomic risk factors in determining an individual's health outcomes (Berkman & Kawachi, 2000). In particular, it differentiates between two classes of mechanisms by which observed group-level associations may causally operate: a compositional effect occurs when the group-level outcome is due solely to the aggregation of individual-level characteristics of members, while a contextual effects arises when the nature of the environment in which group members live determines their health outcomes (MacIntyre, Ellaway & Cummins, 2002). Examples of the former include the average number of years of education and mean income in an area; examples of the latter include the level of public services provided by government and the distribution of wealth in an area. Research has tended to focus on compositional effects, perhaps because they can be calculated directly through the aggregation of individual-level data, something not possible for contextual phenomena. In particular, studies of health

involving income-based community-level effects more frequently consider poverty in absolute than in relative terms.

Existing epidemiological insights into the relationships between SES and TB risk come almost exclusively from either individual- or group-level (ecological) studies, yet such studies are unable to distinguish the compositional and contextual effects of socioeconomic factors on TB risk. A comprehensive investigation of the relationship between SES and tuberculosis needs to jointly consider the effect of both individual- and community-level measures of SES. Such an approach requires a multilevel analysis, incorporating variables at different levels of aggregation, to differentiate between compositional and contextual health effects (Diez-Roux & Aiello, 2005). To better understand the potential compositional and contextual influences of SES on tuberculosis risk, we conducted a multilevel analysis of the relative importance of demographic, socio-behavioral and socioeconomic individual-level risk factors, alongside group-level measures of SES, in determining tuberculosis outcomes in South Africa. Our primary hypothesis was that group-level income inequality is an important determinant of tuberculosis disease, independent of typically-used measures of SES.

Methods

This secondary analysis combined data from two cross-sectional surveys, the 1998 South African Demographic and Health Survey (SADHS) and the 1996 South African national census. Demographic and Health Surveys (DHS) are an international series of nationally representative surveys conducted in middle- and lower-income countries (Fisher & Way, 1988). The 1998 SADHS was the first DHS conducted in South Africa and had a household response rate of 97% and an adult response rate of 90% (*South African Demographic and Health Survey*, 2002). The study population for our analysis was the 13,826 respondents to the SADHS adult health questionnaire, from which those individuals

with missing data were excluded. *Household* characteristics from the SADHS household questionnaire were linked to each respondent in the adult health questionnaire. *Community-level* characteristics were assigned to each individual from a 10% random sample of the 1996 South African census (Statistics South Africa, 1998). These community-level characteristics were defined according to an individual's Magisterial District of residence (MD: the basic administrative unit of the nation; there were 345 MDs in 1998, with an average population of 130,000).

The SADHS protocol was approved by the research ethics committee of the South African Medical Research Council and by the National Department of Health and informed consent was obtained from all participants prior to its conduct. This analysis was approved by the Research Ethics Committee of the University of Cape Town.

Measures

The two outcome measures were: (i) whether a participant in the SADHS had been told by a healthcare professional in the past year that they had tuberculosis ("recent tuberculosis"); and (ii) whether they had ever been so told ("lifetime tuberculosis"). In addition, a wide range of individual- and household-level variables believed to be associated with tuberculosis disease were included in the analysis (Table 1). Of note, the CAGE questionnaire is a four-question method for assessing alcohol dependency, with a score greater than one associated with being at risk of alcoholism; it has been validated in a rural South African setting (Claasen, 1999). Body mass index (BMI) was directly measured in the SADHS. This index and whether a household had ever gone without a meal due to a lack of funds were used as proxies for malnutrition. The number of adults per bedroom in a household provided a measure of household crowding.

We used a household asset index as a measure of household wealth. Using wealth as a measure of SES takes account of all resources available to a household, rather than only the current inflow as provided by cash income (Myer, Ehrlich & Susser, 2004). The index was independently developed through factor analysis of nine questions in the SADHS covering: main source of drinking water; type of toilet facility; type of fuel used; number of rooms used for sleeping; main material of floors and walls; affordability of food; and household and individual ownership of specific assets (Booysen, 2002). Households were ranked by this index and then split into five quintiles of wealth.

At the community level measures of SES were constructed for each MD participating in the SADHS, based on household-level employment and income data from the national census. Each individual in the census reported their usual annual income as falling into one of 14 bands ranging from none to more than R360,000 ($53,614, 2006 exchange rate (Bank of Canada, n.d.)). An average point value was assigned to each income band from figures previously used by Statistics South Africa (Statistics South Africa, n.d.), and the total of all individual incomes in each household aggregated to create an estimate of each household's total income. Respondents also indicated whether or not they were employed at the time of the census.

Magisterial Districts were ranked into quintiles for each of four community-level SES measures: two measures of absolute poverty (headcount poverty rate and unemployment rate) and two measures of income inequality (Gini coefficient and Robin Hood index). The headcount poverty rate measured the proportion of households in each MD earning less than R500 ($74) per adult-equivalent (children were valued at 0.5 adults) per month. The unemployment rate was the proportion of households without an employed adult member at the time of the census. The Gini coefficient is a standardized measure of the absolute

differences between each household's size-adjusted income and every other household's size-adjusted income. The Robin Hood index is a calculation of how much must be taken from the rich and given to the poor in order to equalize incomes (Kennedy, Kawachi & Prothrow-Stith, 1996). For both the Gini and Robin Hood measures, higher values indicate greater income inequality.

Analytic methodology

Data were analyzed using Stata version 9.2 (StataCorp; College Station, TX, USA). Variables analyzed in quintiles used the middle quintile as the reference category. Tests of equality of variables were conducted using a Wald χ^2 test; all statistical tests were two-sided at α=0.05. All analyses were adjusted for the SADHS sampling strategy using survey weights. Bivariate relationships between the two outcome measures and each independent variable were examined using simple logistic regression to generate odds ratios (OR) and 95% confidence intervals (CI).

Multivariate analysis was carried out using a hierarchical model that included fixed effects and group-level intercepts as random effects (Bryk & Raudenbush, 1992; Snijders & Bosker, 1999). This allowed for the three-level, inherently nested nature of the data: individuals nested within households, and households nested within MDs. Hierarchical models allow the additional information provided by knowing which household or community an individual comes from to be taken into account in the modeling the relationship between independent and dependent variables. All multivariate models were run as three-level, hierarchical models using the GLLAMM routine in Stata (Rabe-Hesketh, Pickles & Skrondal, 2004) which were then further adjusted for clustering at the provincial level.

All explanatory variables found to be associated with either dependent variable in bivariate analysis were included in the multivariate model, with the exception of 'ever consumed alcohol' since it was highly correlated with 'CAGE score greater than 1', and 'ever worked in a mine' which was less predictive than the similar 'ever worked in a gold mine'. For each outcome, a first regression model was constructed to examine individual- and household-level variables. Subsequently, each community-level variable was added in turn, since the high degree of correlation between these four measures made their joint inclusion inadvisable.

Results

The study sample consisted of the 13,043 respondents (94.3% of the SADHS population) with information on all variables of interest (Table 1). The sample was 59% female, 75% African, 11% coloured, 10% white and 4% Asian.[1]

In this sample 69 individuals (0.5%) reported having been told they had tuberculosis in the past 12 months. After adjusting for the survey design, the population incidence rate was 422 cases per 100,000 per annum. A total of 369 respondents (2.8%) reported having ever been told they had tuberculosis disease, including the 69 (18.7%) who had been so informed in the past year; after adjustment the lifetime prevalence was 2365 per 100,000 persons.

Thirty-six percent of respondents had been employed during the previous year, with those aged 30-44 years and men mostly likely to report recent employment. The level of education in the sample decreased with increasing age, from an average of 9.4 years for those aged under 30 years to 5.6 years for those aged over 45 years, and was significantly higher for women than for men. Over 10% of men in the sample had ever worked on a mine, and this was strongly associated with increasing age. Just over a quarter of individuals had ever

smoked 100 or more cigarettes, and almost two-fifths had ever drunk alcohol. Both smoking and alcohol consumption was associated with increasing age and male gender. Less than 10% of participants had a BMI below 18.5 ('underweight'), with those aged under 30 years being at greatest risk; men were twice as likely to be underweight than women.

Eighty-three percent (287 of 345) of the MDs in South Africa were surveyed by the SADHS. Within these, an average of 17% of households earned less than R500 per adult-equivalent, while an average of 38% of households had no employed adult member. The mean community Gini coefficient was 0.67 and the mean Robin Hood index was 47.8. For all four measures of community-level SES those aged 30-44 years lived in communities with slightly higher SES than respondents of other ages while women lived in slightly more deprived communities than men.

Associations with recent and lifetime tuberculosis

Bivariate risk factors for recent and lifetime tuberculosis disease are shown in Table 2. Women were less likely than men to have been recently diagnosed with tuberculosis, and significantly less likely to have been ever diagnosed. Similarly, those aged over 30 years were more likely to have had tuberculosis recently or ever, but this association was only significant in the case of lifetime disease. Compared to Africans, whites and Asians were significantly less likely to have had either recent or lifetime tuberculosis, while coloured respondents were significantly more likely to have had lifetime disease.

For almost all behavioral and socioeconomic risk factors, unadjusted point estimates for recent tuberculosis were close to those for lifetime tuberculosis, but associations reached statistical significance more often for lifetime disease. Education was protective of tuberculosis disease, at a rate of a 10% reduction in the odds of disease for every additional year of education completed. Individuals who had worked in the past year had 30% reduced

odds of recent tuberculosis, and 40% reduced odds of lifetime disease. Ever having worked in a mine was associated with a 2.3 times increase in the odds of lifetime tuberculosis while having ever worked on a gold mine was associated with a more than doubling of the prevalence of both recent and lifetime diagnosis.

Living in a household which had ever missed meals due to a lack of funds was associated with a more than doubling in the odds of lifetime and recent disease. Living in a household with higher crowding was associated only with recent tuberculosis. Individuals living in households falling into quintiles two through four of the household asset index had similar prevalence of lifetime and recent tuberculosis. Those in the least wealthy quintile were at roughly double the odds of those living in the middle quintile for both outcomes, and this difference achieved statistical significance for lifetime illness. Participants in the wealthiest quintile had a reduced prevalence of any tuberculosis diagnosis compared to those in the middle quintile, by as much as 85% in the case of recent disease.

The crude relationships between community-level SES measures and tuberculosis are shown in Table 3. The average level of income poverty seen in the community in which an individual was resident had little effect on their likelihood of reporting tuberculosis disease, with the exception of those in quintile 4 who had a significantly reduced prevalence of recent diagnosis. Living in a MD in either of the two lowest unemployment rate quintiles was associated with a significantly reduced odds of tuberculosis compared to living in an MD in the middle quintile.

The two measures of community-level inequality showed a threshold relationship between level of inequality and tuberculosis diagnosis: individuals living in the two most unequal quintiles according to the Gini coefficient were at more than double the prevalence lifetime tuberculosis that those living in the middle quintile. Slightly smaller associations

were observed using the Robin Hood index. Although increasing inequality according to the Robin Hood index was associated with recent tuberculosis diagnosis, this did not achieve statistical significance.

Multivariate associations with tuberculosis

Many of the crude associations involving risk factors for tuberculosis did not persist in multivariate analysis (Table 4). Adjusted analyses containing individual- and household-level variables found being female to be associated with a 31% reduced odds of recent tuberculosis, although this result was not statistically significant. After adjusting for other risk factors, the race of an individual was not significantly associated with either tuberculosis measure.

The number of years of education completed by an individual had a marginally significant association with tuberculosis, with each additional year being associated with a 3 to 4% reduction in the odds of tuberculosis disease. Working in the past year reduced an individual's odds of disease by at least 25%, although this was significant only in the case of lifetime tuberculosis.

The individual socio-behavioral risk factors which had been strong predictors of disease in crude analysis – smoking, alcohol consumption and low BMI – remained strongly predictive of tuberculosis in adjusted analysis for both recent and lifetime disease. The associations between tuberculosis and household crowding and meals missed were attenuated in multivariate analysis, although having ever missed meals remained weakly associated with lifetime disease. The inverse relationship between household wealth quintile and tuberculosis remained in adjusted analysis. A significant protective effect (OR: 0.25, 95% CI: 0.13-0.51) was observed in the wealthiest quintile for recent disease, while being resident in the poorest quintile was associated with increased odds of lifetime disease (OR: 1.68, 95% CI: 1.10-2.55), compared to the middle quintile.

The introduction of each of the community-level SES variables into the multivariate model (Table 4) had little effect on the more proximal associations reported above. Regressions containing MD-level poverty and unemployment did not change the point estimates for the household asset score or recent employment history respectively, but the crude associations between the community-level variables and tuberculosis were attenuated (not shown).

Regression models containing community-level Gini coefficients found that the associations seen in crude analysis continued to hold. In particular, those living in MDs ranked in the two quintiles with the greatest income inequality had more than double the odds of developing tuberculosis compared to those living in the middle quintile, after adjusting for lower-level demographic, behavioral and socioeconomic variables including absolute wealth. Regression models involving the Robin Hood index as the measure of income inequality (not shown) produced similar associations.

Discussion

This analysis of a nationally-representative sample of the South African population found the prevalence of self-reported tuberculosis disease to be associated with low personal, household and community SES. Low levels of personal education, unemployment and a low level of household wealth were associated with increased odds of tuberculosis. Low levels of community-level employment and wealth did not appear to have an independent effect on tuberculosis occurrence, once individual and household effects were taken into account. However, individuals living in areas of high inequality had an increased prevalence of tuberculosis disease, independent of their individual- and household-level risk factors. These results were strikingly similar for both recent (12-month) and lifetime measures of tuberculosis disease.

This study adds several new dimensions to current understandings of the social determinants of tuberculosis. It is one of few studies to examine the associations between SES variables and tuberculosis after adjustment for a range of established demographic and socio-behavioral risk factors (such as BMI, alcohol consumption and smoking). The multilevel analytic approach allows for the differentiation between community- and individual-level mechanisms in the relationship between SES and tuberculosis. Furthermore, these data allow strong inferences to be drawn regarding risk factors for tuberculosis disease across the country. A nationally-representative cross-sectional survey provided evidence on individual and household characteristics, while South African census data provided robust estimates of the true community-level SES characteristics across the nation.

Several potential shortcomings nonetheless require consideration in interpreting these results. The outcome measures used are self-reported data on past diagnosis of tuberculosis by a doctor or nurse, rather than laboratory-confirmed disease. These measures may have diverged if respondent recall was imperfect or if access to healthcare varied systematically. Evidence from South Africa suggests that reliance on self-reported past tuberculosis diagnosis may lead to an underreporting of lifetime disease relative to chest radiography (teWaterNaude, Ehrlich, Churchyard, Pemba, Dekker, Vermeis et al., 2006). If such underreporting is not correlated with the explanatory variables in this analysis then any bias introduced would mean that the reported results underreport the true associations. The most likely systematic variation would be for individuals of higher SES to have had greater access to healthcare, and thus increased likelihood of tuberculosis detection. Such a phenomenon would lead to an inverse association between SES and tuberculosis risk being obscured, again suggesting that the real associations may be stronger than those identified here. This potential for bias warrants further investigation.

Given the cross-sectional nature of the two datasets used, the possibility of reverse causation also requires consideration as an explanation for some of these results. This is particularly likely for analyses involving lifetime tuberculosis, since a previous episode of tuberculosis disease may reduce an individual's ability to work and in turn their SES. The measure of recent tuberculosis diagnosis should, however, be less susceptible to this problem. The similarity of associations between SES measures and tuberculosis for the two measures of tuberculosis suggests that such an explanation plays a limited role here.

Although the data are nationally representative for South Africa in the late 1990s, it is not clear to what degree these results are more widely applicable, particularly since South Africa is a middle-income nation with an unusually high level of income inequality. While the link between income inequality and tuberculosis may hold in high-inequality settings, in line with existing evidence of a threshold effect for inequality and health (Pickett & Pearl, 2001; Subramanian, Delgardo, Jadue, Vega & Kawachi, 2003; Subramanian & Kawachi, 2004), the generalizability of these results to high-income, low-inequality settings is unclear. Similarly, the generalizability of the SES-tuberculosis relationship reported in this analysis may be limited when looking at poorer countries with less variation in absolute income and in income inequality.

South Africa's historic experience of spatial planning along racial lines may have led to tuberculosis epidemiology in this setting differing from elsewhere. The MDs used as communities in this study are large, often encompassing both formal towns and their peri-urban settlements. Any given MD would therefore include a number of race-group areas within a single 'community'. As a result, it may be that income inequality in this analysis is a proxy for inequality in provision of other services – for example housing, education or

medical care – by the Apartheid state along racial lines. If this were so, the result may not be reproducible in other settings.

The absence of data on HIV infection, an established risk factor for tuberculosis disease which is prevalent in South Africa (the national HIV prevalence in 1998 is estimated to have been 5.9% (Actuarial Society of South Africa, 2005)), is a principal limitation in these data. In the absence of this explanatory variable, any risk factor that is correlated with HIV infection will appear to be more closely associated with tuberculosis disease than is truly the case. For example, if HIV is associated with low SES, as has been reported in South Africa (Shisana & Simbayi, 2002), then the true relationship between tuberculosis and SES may be less pronounced than the reported results suggest. However, the absence of data on individual HIV status is of less concern for interpretation if HIV is considered to be a mediator in the pathway between social determinants and tuberculosis. This will be the case if HIV is a risk factor for the outcome (tuberculosis), and a result of the exposure (low SES), i.e. if it is socially patterned. This argument is supported by broad evidence that social deprivation is linked to many health outcomes, including sexually-transmitted diseases (Brandt, 1987), and more specifically by evidence that HIV infection is a result of both poverty and economic inequality in poor and unequal settings such as Haiti and Peru (Farmer, 1999). If so, the inclusion of more distal, social causes of tuberculosis would take into account this mediation process, reducing the potential for confounding due to omitted variables. Indeed, in such a situation, the inclusion of HIV status might be considered an overadjustment of the analysis.

Implications of this study

The findings observed here require further investigation in different settings, especially those with different SES profiles and burdens of tuberculosis infection. This is particularly the case for the recent tuberculosis results, which were underpowered and

particularly likely to have been affected by the absence of HIV data. The addition of anonymous HIV testing to recent DHS studies offers the possibility of conducting such an analysis in the future. Nevertheless, several of the findings are of interest in the study of tuberculosis in particular and in the field of social epidemiology more generally.

This analysis found alcohol abuse, cigarette smoking and low BMI each independently to be a risk factor for tuberculosis in South Africa, even after adjusting for the SES of individuals. One previous review of tuberculosis and smoking reported that smoking was rarely found to be independently associated with tuberculosis after adjustment for socioeconomic factors, although smoking has been generally found to be crudely associated with the disease (Maurya, Vijayan & Shah, 2002). A recent meta-analysis, however, found the relationship between smoking and tuberculosis to be statistically significant, and stronger in those studies which adjusted for SES than those that did not (Lin, Ezzati & Murray, 2007). Relatively little epidemiological research has been conducted on the relationship between alcohol and tuberculosis infection or disease in South Africa, although alcohol is frequently mentioned in the context of non-adherence to treatment (e.g. Jakubowiak, Bogorodskaya, Borisov, Danilova, & Kourbatova, 2007) . This study's findings make the link between alcohol abuse, smoking and tuberculosis in settings where all three are common, and the implications of this link for tuberculosis control programs, an important area of future research.

The significant crude associations seen for tuberculosis and a history of gold mine work and overcrowding were attenuated in the multivariate model. This analysis does not, therefore, reproduce previously-reported associations between tuberculosis and these variables. However, given the established relationship between exposure to silica in gold mining and tuberculosis (teWaterNaude et al., 2006) and the significant associations between

a history of gold mining and poverty, a lack of education, rural residence and BMI (not shown), it is possible that a multivariate model including all these variables amounted to overadjustment.

The finding that tuberculosis is simultaneously associated with several measures of individual- and household-level SES may provide preliminary insight into how socioeconomic factors influence health outcomes. This analysis found low educational achievement, recent unemployment and high levels of household deprivation each to be independently associated with increased odds of tuberculosis. These findings are broadly supportive of the existing literature that finds each of these measures to be correlated with poor health. In high-income settings, however, the joint inclusion of SES measures often leads to a loss of significance for some of them (Chan-Yeung, Yeh, Tam, Kam, Leung, Yew et al., 2005; Davey-Smith, Hart, Hole, MacKinnon, Gillis, Watt et al., 2004; Holtgrave & Crosby, 2004). Such findings suggest an etiological mechanism in which SES measures act through a single causal pathway with high substitutability (Figure 1a). This study, however, found that the inclusion of any one SES measure has little impact on the relationship between tuberculosis and any other SES measure, suggesting that in this setting each SES measure may act through a separate causal pathway (Figure 1b).

This analysis also adds to an existing literature that has found income inequality to be associated with various health outcomes, particularly in high-inequality settings (Kawachi, 2000; Kawachi & Blakely, 2001; Pickett & Pearl, 2001; Subramanian et al., 2003; Subramanian & Kawachi, 2004). It is important to note that these findings of association do not necessarily imply causality, but may reflect inequality as a confounder, perhaps of a true relationship between income inequality and HIV (Holtgrave & Crosby, 2003; Drain, Smith,

Hughes, Halperin & Holmes, 2004), or of psychosocial stress and illness (Deaton, 2003; McEwen, 1998).

The emphasis of existing tuberculosis prevention policies in South Africa is on disease treatment. The evidence presented in this study suggests that greater emphasis on social and economic policies that focus on assisting the poor and marginalized is needed in the discourse of tuberculosis control. In order to implement such policies effectively it is necessary to understand the causal mechanisms at play. In the case of income inequality several candidates exist, each of which is consistent with the finding in this study of inequality being harmful for the most deprived sections of society rather than being protective for the rich. First, inequality may affect a community's access to healthcare. More unequal communities may have more limited access to healthcare for their poorest members than similarly poor individuals in more egalitarian settings (Subramanian & Kawachi, 2004). In this case poor people in unequal settings would be expected to have worse health than their wealth level would otherwise predict (Figure 1c, pathway i). Second, inequality may affect the structure of communities in which it is prevalent by causing higher levels of psychosocial stress than more egalitarian ones (Wilkinson & Pickett, 2006). This stress may raise an individual's risk of tuberculosis (it may also mediate the relationship between household overcrowding and tuberculosis). The result would be higher levels of tuberculosis in more unequal settings (Figure 1c, pathway ii). Third, in an unequal community it is likely that the poorest members will be marginalized due to weaker bonds between members. This may cause more unequal areas to suffer poorer health outcomes, either because of poor diffusion of health information and healthy behavioral norms, or due to a reduced capacity to lobby for the provision of healthcare and other public services (Kawachi & Berkman, 2000). Marginalization may therefore act as a mediator in either of the other two causal mechanisms (Figure 1c, pathway iii). These three mechanisms are not exhaustive, but rather indicative of possible pathways

between income inequality and disease outcomes. It is important that in future research these and other hypothetical causal mechanisms are tested empirically, since different pathways may entail different practical interventions to lessen the burden of tuberculosis.

Conclusion

This study presents novel insights into the associations between socioeconomic conditions and risk of tuberculosis disease. The use of a social epidemiology framework to analyze risk factors for tuberculosis, particularly in sub-Saharan Africa, is a young endeavor. These findings require further investigation, but highlight the importance of including measures of community-level SES, particularly emergent characteristics such as income inequality, in future research on tuberculosis. Despite concerns over the generalizability of the context, this study suggests that both individual- and community-level SES may play an important role in determining an individual's risk of becoming ill with tuberculosis.

[1] Apartheid sought to categorize all South Africans into one of four racial groups: Asian (or Indian), African (or black), coloured and white. The social stratification by racial group reflects large and enduring historical disparities in SES, quality of housing and services and access to medical care, and consequently in disease risk. Racial group stratification has been retained in national health surveillance in South Africa to reflect a social complexity not fully captured by education, income, etc. and to monitor progress toward reduction of health disparities.

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Table 1: Descriptive statistics adjusted for sample design

	Full Sample	Female	Male	p-value
Sample size	13,043	8,073	5,753	
Dependent variables				
Diagnosed with tuberculosis in past year	0.42	0.39	0.47	0.48
Diagnosed with tuberculosis ever	2.37	1.97	2.94	< 0.01
Individual-level demographic risk factors				
Sex		58.94	41.06	
Age in years				
15 - 29	39.1	36.5	43.0	< 0.01
30 - 44	27.6	28.1	26.9	0.15
45 and over	33.3	35.4	30.2	< 0.01
Race group				
African	75.5	76.1	74.5	0.04
Coloured	10.7	10.2	11.5	0.01
White	10.0	9.8	10.3	0.42
Asian	3.8	3.9	3.7	0.49
Individual-level SES risk factors				
Years of education (mean)	7.8	7.5	8.1	< 0.01
Worked for payment in past 12 months	35.8	29.1	45.4	< 0.01
Ever worked in a mine	4.3	< 0.01	10.2	< 0.01
Ever worked in a gold mine	2.8	< 0.01	6.7	< 0.01
Individual-level socio-behavioral risk factors				
Urban residence	61.9	61.2	62.8	0.11
Ever smoked 100 cigarettes or more	26.4	15.4	42.1	< 0.01
Ever drunk alcohol	39.1	25.7	58.3	< 0.01
CAGE score greater than one	17.1	9.8	27.4	< 0.01
BMI below 18.5	8.6	5.7	12.9	< 0.01
Household-level risk factors				
Number of adults per bedroom (mean)	1.50	1.49	1.52	0.03
Meals missed due to lack of funds	48.5	49.2	47.4	0.05
Asset score quintile (mean)	3.26	3.24	3.31	< 0.01
Community-level risk factors				
Headcount poverty rate (mean)	17.3	17.7	16.7	< 0.01
Unemployment rate (mean)	38.3	39.3	36.9	< 0.01
Gini coefficient (mean)	0.67	0.66	0.65	< 0.01
Robin Hood index (mean)	47.8	46.4	45.8	< 0.01

Community-level values are those assigned to each individual. All data adjusted for sampling weights at the cluster level. All figures proportions unless otherwise indicated. P-value is that of a χ^2 adjusted Wald test for equality.

Table 2: Bivariate relationships between tuberculosis outcomes and individual- and household-level explanatory variables

	Recent tuberculosis		Lifetime tuberculosis	
	Odds ratio	95% CI	Odds ratio	95% CI
Individual-level demographic risk factors				
Female	0.82	[0.49-1.39]	0.66	[0.51-0.86]
Age				
15-29 years	1.00	(reference)	1.00	(reference)
30-44 years	1.19	[0.58-2.47]	2.78	[1.90-4.06]
>44 years	1.23	[0.65-2.30]	3.38	[2.39-4.79]
Race group				
African	1.00	(reference)	1.00	(reference)
Coloured	1.00	[0.43-2.33]	1.72	[1.22-2.46]
White / Asian	0.12	[0.02-0.84]	0.46	[0.25-0.86]
Individual-level SES risk factors				
Years of education	0.90	[0.86-0.94]	0.89	[0.87-0.92]
Worked for payment in past 12 months	0.59	[0.34-1.04]	0.69	[0.51-0.87]
Ever worked in a mine	1.55	[0.61-3.92]	2.32	[1.55-3.47]
Ever worked in a gold mine	2.40	[0.94-6.10]	2.73	[1.81-4.13]
Individual-level socio-behavioral risk factors				
Urban Residence	0.61	[0.34-1.09]	0.67	[0.50-0.89]
Ever smoked 100 cigarettes or more	2.28	[1.30-4.00]	2.42	[1.89-3.08]
Ever drunk alcohol	1.72	[0.99-2.97]	2.44	[1.88-3.17]
CAGE score greater than one	3.09	[1.74-5.48]	3.59	[2.79-4.61]
BMI below 18.5	4.71	[2.63-8.43]	3.05	[2.30-4.04]
Household-level risk factors				
Number of adults per bedroom	1.27	[1.03-1.55]	1.05	[0.93-1.18]
Meals missed due to lack of funds	2.44	[1.31-4.54]	2.15	[1.60-2.90]
Asset score quintile				
1 (poorest)	1.90	[0.87-4.09]	2.37	[1.63-3.44]
2	0.97	[0.44-2.12]	1.36	[0.86-2.15]
3	1.00	(reference)	1.00	(reference)
4	0.76	[0.31-1.87]	1.10	[0.73-1.67]
5 (wealthiest)	0.15	[0.03-0.69]	0.67	[0.41-1.07]

Table 3: Bivariate relationships between tuberculosis outcomes and community-level explanatory variables

	Recent tuberculosis		Lifetime tuberculosis	
	Odds ratio	95% CI	Odds ratio	95% CI
Headcount poverty quintile:				
1 (poorest)	1.28	[0.62-2.62]	1.34	[0.89-2.02]
2	0.64	[0.25-1.66]	0.94	[0.60-1.46]
3	1.00	(reference)	1.00	(reference)
4	0.39	[0.16-0.94]	0.66	[0.41-1.08]
5 (richest)	0.74	[0.28-1.96]	0.97	[0.60-1.56]
Unemployment rate quintile:				
1 (highest)	0.80	[0.39-1.67]	1.00	[0.67-1.49]
2	0.41	[0.17-0.98]	0.76	[0.46-1.25]
3	1.00	(reference)	1.00	(reference)
4	0.30	[0.12-0.75]	0.41	[0.25-0.67]
5 (lowest)	0.37	[0.14-0.97]	0.63	[0.40-0.99]
Gini coefficient quintile:				
1 (most unequal)	1.43	[0.53-3.84]	2.60	[1.53-4.42]
2	0.54	[0.19-1.54]	2.16	[1.20-3.90]
3	1.00	(reference)	1.00	(reference)
4	1.03	[0.39-2.80]	1.54	[0.90-2.63]
5 (least unequal)	0.45	[0.16-1.28]	1.20	[0.72-2.01]
Robin Hood index quintile:				
1 (most unequal)	2.27	[0.81-6.39]	1.50	[0.96-2.33]
2	2.46	[0.85-7.18]	1.42	[0.84-2.39]
3	1.00	(reference)	1.00	(reference)
4	1.35	[0.49-3.73]	0.59	[0.36-0.89]
5 (least unequal)	0.99	[0.35-2.77]	0.73	[0.49-1.10]

Table 4: Multivariate relationships between tuberculosis and explanatory variables

	Recent tuberculosis		Lifetime tuberculosis		Recent tuberculosis		Lifetime tuberculosis	
	Odds ratio	95% CI	Odds ratio	95% CI	Odds ratio	95% CI	Odds ratio	95% CI
Demographic risk factors								
Age	1.00	(0.97 - 1.03)	1.02	(1.01 - 1.03)	1.00	(0.97 - 1.03)	1.02	(1.01 - 1.03)
Female	0.69	(0.47 - 1.04)	1.13	(0.86 - 1.50)	0.70	(0.48 - 1.02)	1.14	(0.86 - 1.52)
African	1.00	(reference)	1.00	(reference)	1.00	(reference)	1.00	(reference)
Coloured	1.12	(0.23 - 5.45)	2.04	(0.79 - 5.24)	1.09	(0.22 - 5.33)	2.16	(0.81 - 5.75)
White / Asian	0.42	(0.13 - 1.39)	0.93	(0.34 - 2.50)	0.42	(0.12 - 1.43)	0.96	(0.35 - 2.66)
Individual-level SES risk factors								
Years of Education	0.96	(0.90 - 1.01)	0.97	(0.93 - 1.00)	0.96	(0.90 - 1.01)	0.97	(0.93 - 1.00)
Worked for payment in past 12 months	0.75	(0.47 - 1.20)	0.72	(0.53 - 0.98)	0.76	(0.47 - 1.24)	0.73	(0.53 - 1.01)
Ever worked in a gold mine	1.47	(0.43 - 4.99)	1.28	(0.82 - 2.00)	1.44	(0.43 - 4.77)	1.25	(0.81 - 1.93)
Individual-level socio-behavioral risk factors								
Urban Residence	1.06	(0.49 - 2.32)	1.04	(0.71 - 1.50)	1.04	(0.48 - 2.23)	1.17	(0.82 - 1.67)
Ever smoked 100 cigarettes or more	2.20	(1.20 - 4.02)	1.62	(1.02 - 2.58)	2.15	(1.19 - 3.89)	1.64	(1.03 - 2.62)
CAGE score greater than one	1.97	(1.21 - 3.22)	2.52	(1.86 - 3.4)	2.01	(1.21 - 3.34)	2.53	(1.88 - 3.40)
BMI below 18.5	3.94	(1.68 - 9.23)	2.84	(1.91 - 4.22)	3.91	(1.71 - 8.93)	2.87	(1.95 - 4.23)
Household-level risk factors								
Number of adults per bedroom	1.11	(0.78 - 1.57)	0.96	(0.82 - 1.13)	1.12	(0.78 - 1.59)	0.98	(0.84 - 1.14)
Meals ever missed due to lack of funds	1.22	(0.58 - 2.55)	1.76	(0.92 - 3.38)	1.21	(0.55 - 2.67)	1.73	(0.91 - 3.32)
Asset score quintile:								
1 (poorest)	1.51	(0.87 - 2.60)	1.68	(1.10 - 2.55)	1.48	(0.77 - 2.82)	1.56	(1.04 - 2.34)
2	0.86	(0.43 - 1.72)	1.03	(0.73 - 1.45)	0.86	(0.42 - 1.76)	1.00	(0.71 - 1.41)
3	1.00	(reference)	1.00	(reference)	1.00	(reference)	1.00	(reference)
4	0.71	(0.36 - 1.41)	0.93	(0.56 - 1.53)	0.72	(0.35 - 1.48)	0.95	(0.57 - 1.57)
5 (wealthiest)	0.25	(0.13 - 0.51)	0.81	(0.48 - 1.39)	0.26	(0.13 - 0.54)	0.80	(0.48 - 1.32)
Community-level SES risk factors								
Gini coefficient quintile:								
1 (most unequal)					1.15	(0.41 - 3.23)	2.37	(1.59 - 3.53)
2					0.48	(0.08 - 2.87)	2.18	(1.42 - 3.34)
3					1.00	(reference)	1.00	(reference)
4					0.98	(0.15 - 6.48)	1.25	(0.89 - 1.75)
5 (least unequal)					0.85	(0.42 - 1.72)	1.57	(0.88 - 2.82)

Figure 1: Models of causal pathways between socioeconomic status and tuberculosis

Figure 1a: Hypothetical common pathways for SES causes

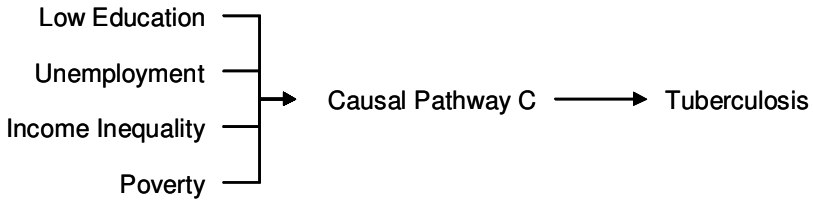

Figure 1b: Hypothetical multiple pathway for SES causes

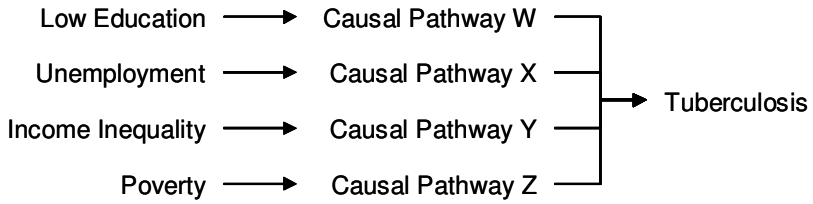

Figure 1c: Hypothesized multiple causal pathways for income inequality