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Forced removals embodied as tuberculosis

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ABSTRACT

South Africa has one of the worst tuberculosis burdens in the world. Several ecological forces have contributed to this, including high HIV prevalence; failing TB control strategies; crowded, poorly ventilated indoor environments—including the complex web of political and economic interests which produce them; the development of racial capitalism; and mining and migration. In the following study, we measure CO_2 levels in public transport to investigate the role extended commutes from peri-urban settlements to urban sites of work—a direct result of forced removals—potentially play in propagating the TB epidemic in Cape Town, South Africa.

> "My friends … Circumstances … render it impossible that you can flourish in the midst of a civilized community. You have but one remedy within your reach. And that is, to remove to the West … And the sooner you do this, the sooner you will commence your career of improvement and prosperity." —Andrew Jackson (7th President of the United States), *To the Cherokee Tribe of Indians East of the Mississippi* (1835)

1. Introduction

With a yearly tuberculosis (TB) incidence of 834 cases per 100,000 people, South Africa has one of the worst TB burdens in the world (World Health Organization, 2015a). The number of TB cases reported in Cape Town alone (population 3.4 million) is more than three times the number of all TB cases reported in the United States, a country of more than 300 million people (Wood et al., 2011a). Furthermore, the rate of acquiring TB infection in Cape Town is similar to that which existed in early 20th century Europe, prior to the advent of chemotherapy (Vynnycky and Fine, 1999; Wood et al., 2010). Several ecological forces have contributed to this, including high HIV prevalence (World Health Organization, 2009a); failing TB control strategies (Wood et al., 2011b); crowded, poorly ventilated indoor environments (Chapman and Dyerly, 1964)—including the complex web of political and economic interests which produce them (Farmer, 2000, 2005); the development of racial capitalism (Packard, 1989); and mining and migration (Stuckler et al., 2011).

One underexplored determinant has been forced removals, that is, the policies and often violent processes involved in the massive, state-sponsored displacement of people (almost all of them black) from one area to another in South Africa (Platzky and Walker, 1985). Starting well before *apartheid* with the Public Health Act of 1897, the Native Reserve Location Act of 1902, and the Native Urban Areas Act of 1923—and consolidated in the Group Areas Acts of the 1950's and 60's—forced removals uprooted millions of individuals from both developed urban and rural areas to underdeveloped and poorly resourced peri-urban and rural areas resulting in widespread poverty, disease, and starvation (Desmond, 1971; Platzky and Walker, 1985; Surplus People Project, 1983).

As Rebecca Saunders writes, forced removals instantiate "a contrived geography that … forces thousands to travel as far as two

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hundred miles a day to and from work and to expend a quarter of their earnings on bus fares" (Saunders, 2003). In Cape Town, for example, hundreds of thousands of Africans, Coloreds, and Indians were forcibly relocated over a fifty-year period from the city limits to the Cape Flats (Fig. 1) (Platzky and Waler, 1985; South African History Online (SAHO), 2015).

For a paper presented at the 1982 University of Cape Town Medical Students' Conference entitled *Consumption and Underconsumption: The Effects of Population Resettlement on the Spread of Tuberculosis*, Saul Dubow wrote,

> Clearly, we have to accept as a first premise that TB is a social disease born out of and aggravated by poverty, malnutrition, overcrowding and stress. But if we are to make progress in our understanding of TB it is not sufficient merely to describe these social conditions. We have to explain them (Dubow, 1982).

The myriad ways in which social, political, and economic forces become embodied as pathology are often difficult to trace given the dearth of formal methods in mainstream epidemiology and other biomedical models of disease causality; however, over the past thirty years, social theorists have begun to offer critical analyses of epidemic disease which interrogate how features of disease distribution are obscured by dominant analytic frameworks (Farmer, 1996; Fassin, 2002; Richardson et al., 2016; Scheper-Hughes and Lock, 1986; Scheper-Hughes, 1990).

In his seminal genealogy of the TB epidemic in South Africa, Randall Packard traced the effects of social policies and capital interests that coalesced to drive TB incidence among marginalized black populations. He concludes in the Epilogue, "In effect the municipal authorities, who for decades pushed the sick beyond the city limits and thereby sowed the seeds of TB infection in the surrounding black peri-urban and rural areas, are now having to reap the harvest of TB cases their exclusionary practices have produced" (Packard, 1989).

Paul Farmer provided another historically deep analysis of TB persistence in marginalized populations, tracing how poverty and structural violence both constrain individual agency and shape the physical environments that place people at risk (Farmer, 2001). Moreover, he has argued compellingly that public health programs which neglect discussions of racial injustice, political oppression, and health equity perpetuate this structural violence through analytic omission (Farmer et al., 2006). He thus promotes *biosocial* approaches to understanding and alleviating disease burdens

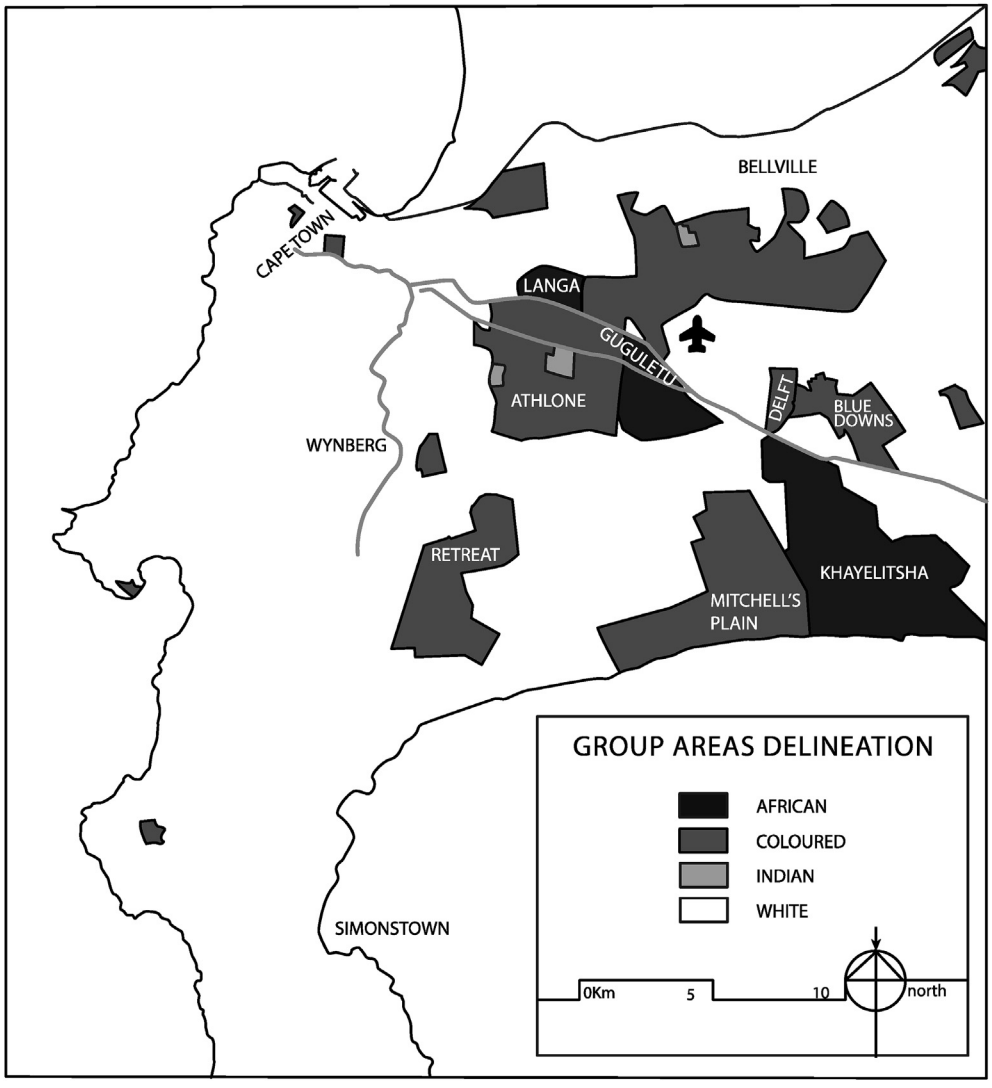

Fig. 1. Map of Group Areas Act delineations.

(Farmer, 2013; Ortblad et al., 2016).

Our research group has previously estimated the annual risk of TB infection attributable to public transit among daily commuters in Cape Town to be 3.5%–5.0% (Andrews et al., 2013). In the following paper, we present empirical data which illustrates the impact that extended commutes from peri-urban settlements to urban sites of work—a direct result of forced removals—potentially play in propagating the TB epidemic in Cape Town, South Africa.

2. Materials and methods

Re-circulated indoor air has long been recognized as a mechanism for infectious disease transmission (Wells, 1955). In addition, several studies have identified public transport as a risk factor for TB transmission (Edelson and Phypers, 2011; Feske et al., 2011; Horna-Campos et al., 2007). By measuring carbon dioxide (CO_2) levels in public transport, one can estimate probabilities of TB transmission using methods developed by Rudnick and Milton (Rudnick and Milton, 2003).

2.1. Data collection procedures

A portable carbon dioxide detection device with data logger (Fig. 2) was developed through collaboration between the Desmond Tutu HIV Centre (DTHC) and the University of Cape Town Electrical Engineering Department. The device provides a CO_2 measurement in parts per million (ppm) every 60 s, as well as global positioning system (GPS) location data. Incorporated into the device is a COZIR Ambient 0–1% transducer (Gas Sensing Solutions Ltd, Glasgow, United Kingdom, http://www.cozir.com/), GPS antenna, microcontroller device, Universal Serial Bus (USB) interface, and an independent power supply. The collected measurements are stored on flash memory and can be uploaded to a computer via USB. These devices have been used previously by the DTHC in studies examining carbon dioxide levels in indoor environments (Richardson et al., 2014; Wood et al., 2014).

A heterogeneous convenience sample of seven study participants was recruited from the Institute of Infectious Disease and Molecular Medicine at the University of Cape Town. We selected seven key 'resettlement' locations—Langa, Guguletu, Retreat, Mitchells Plain, Khayelitsha, Delft, and Blue Downs—and recruited staff members who commuted on public transport (bus or minibus taxi) to each location (Fig. 3). We provided each study participant with portable loggers to carry with them at all times during the one-week study period.

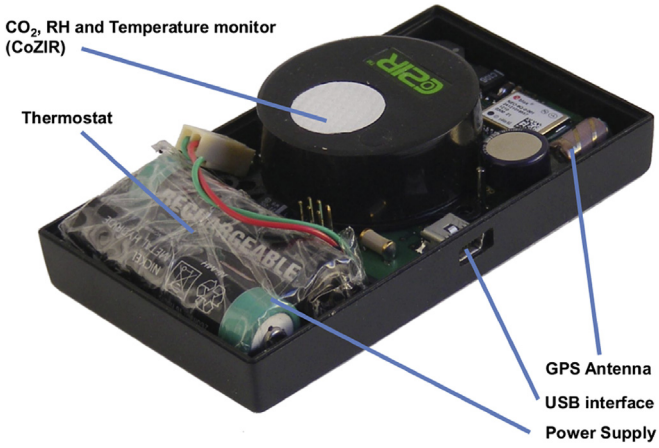

Fig. 2. Portable logger to measure CO_2 concentration, temperature, humidity and GPS coordinates.

2.2. Travel diary

We provided each of the study participants with a paper diary to record morning and evening commutes during a one-week period. The participants were given instructions to write down departure and arrival times of their daily commutes (both morning and evening), mode of transport (e.g., bus or taxi), number of co-riders present, and whether vehicles' windows were open or closed. The diaries were collected at the end of the study period and entered into the database. Missing information or entries were reviewed with the participants retrospectively.

2.3. Data analysis and equations used for estimation of transmission risk

GPS data from the portable logger were matched with routes reported in participant diaries. CO_2 measurements during participants' commutes were used to compute the average rebreathed amount of air per participant for those commutes where vehicles had the windows closed.

Following the work of previous studies (Wood et al., 2014), we first used Rudnick and Milton's equation (Rudnick and Milton, 2003)

$$f = \frac{C - C_o}{C_a} \quad (1)$$

to calculate the fraction of air that is exhaled breath (f). Here, C is the observed concentration of CO_2 in the vehicle, C_o is the concentration of CO_2 in outdoor air (400 ppm), and C_a is the concentration of CO_2 in exhaled air (for individuals at low levels of physical activity, C_a was estimated to be 38,000 ppm based on a CO_2 production rate of 0.30 L/minute and respiratory minute volume of 8.0 L/minute) (Feher, 2012; Morawska et al., 1995; Tans and Keeling, 2014).

Next, we calculated the rebreathed fraction (f_o) of exhaled air from other riders using

$$f_o = f \times \frac{(n-1)}{n} \quad (2)$$

where (n) is the average number of people present in commuting vehicles as recorded in participant diaries. Finally, the average amount of air rebreathed from other riders per daily commute (R) was calculated from the product of f_o, the minute respiratory volume (p), and the average commute time in minutes (t):

$$R = f_o p t \quad (3)$$

3. Results

Table 1 lists the average amount of rebreathed CO_2 in litres for commutes with closed windows to and from the seven key 'resettlement' locations, including mode of transport, average recorded CO_2 in ppm, and average number of co-commuters.

4. Conclusions

This study answers Singer's call for "theoretical models in the anthropology of infection that link microlevel social patterns [commutes on crowded, poorly ventilated transport] with macrolevel social structures [institutionalized racism and forced

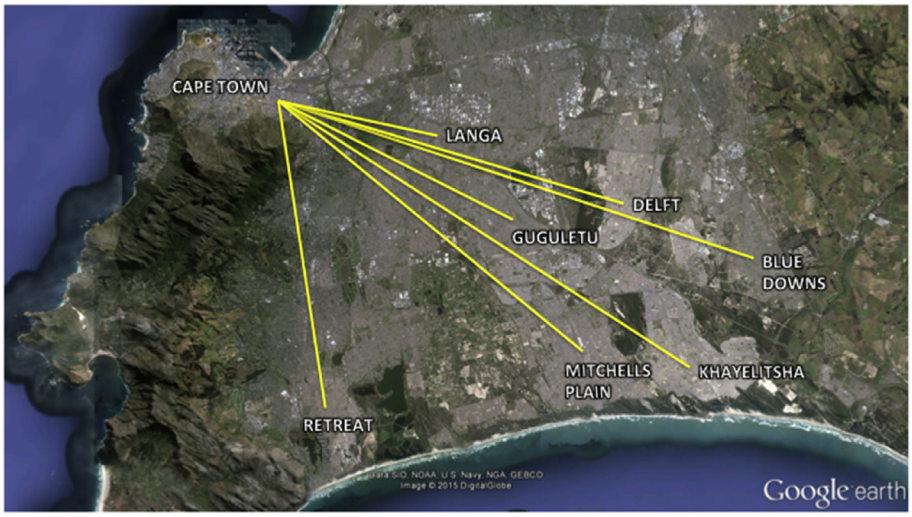

Fig. 3. Map of commuting destinations in the Cape Flats.

Table 1
Average amount of rebreathed CO_2 per daily commute for seven key 'resettlement' locations. 1000 ppm is the cutoff for indoor air quality promoted by the American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE, 2010).

Commuter	Destination	Mode of transport	Average CO_2 (ppm)	Average # of co-riders	Average time per commute (minutes)	Average amount rebreathed CO2 (litres)
1	Langa	Bus	4030	57	45	33.4
2	Guguletu	Taxi	1720	18	48	12.6
3	Retreat	Bus	1767	45	77	21.6
4	Mitchells Plain	Bus	3586	67	72	47.6
5	Khayelitsha	Taxi	5096	17	80	74.4
6	Delft	Taxi	2627	18	73	32.3
7	Blue Downs	Bus	2789	62	87	43.1

removals]" (Singer, 2015). It relies on a small convenience sample of commuters and is thus not powered to provide a true CO_2 average for populations that commute to Cape Town from the Cape Flats; however, our research group has previously reported on the role that public transportation plays in sustaining TB transmission in South Africa (Andrews et al., 2013). Instead, the current study aims to provide a quantitative ethnography of the link between forced removals from urban Cape Town over the past century and the current TB epidemic in 'resettlement' areas.

We have previously demonstrated a CO_2 cutoff of 1000 ppm in crowded indoor environments, above which threshold TB transmission—and thus propagation of the South African epidemic—becomes more likely (Richardson et al., 2014). That our study participants commuted in transport with CO_2 values well above this cutoff suggests these commutes represent a significant potential for TB transmission.

Using the equations developed by Rudnick and Milton, these high CO_2 levels can be integrated with social contact data to calculate the amount of air commuters are rebreathing from each other (Wood et al., 2014). Given the exceptionally high TB prevalence in South Africa (World Health Organization, 2015a), repeated daily commutes with the high amounts of rebreathed air we observed in our study likely result in an elevated transmission of small-particle airborne infections, including TB. We thus begin to appreciate how forced removals and *apartheid* urban planning—which have led to extended commutes in poorly ventilated public transport—become embodied as tuberculosis.

As Rene Dubos writes, tuberculosis can only "be conquered by broadening the scope of conventional medical philosophy" (Dubos and Dubos, 1952). There is growing consensus that biomedical solutions will be insufficient to tackle TB burdens in impoverished settings (Hargreaves et al., 2011), underscoring the imperative to take environmental (Nardell, 2016) and social (Farmer, 1997) determinants of TB transmission seriously. Regarding the former, a renewed focus on ventilation is called for (Richardson and Wood, 2014). The nascent Open Windows Campaign in the Western Cape, which stresses the "importance of opening windows in public transport so to create adequate ventilation and help prevent the spread of TB," is a promising strategy (Fig. 4) (Mbombo, 2015). Another example is the Butaro Hospital in Rwanda, which incorporates the recent revival—almost forgotten in the age of effective treatment and chemoprophylaxis—of architectural design principles for optimal natural ventilation in preventing communicable disease (Fig. 5) (World Health Organization, 2009b).

Such technical interventions, however, must not distract from the social inequities that continue to drive TB incidence. These can be addressed by income redistribution (Ferguson, 2015), urban regeneration (Hargreaves et al., 2011), a greater focus on care (Wilkinson and Kleinman, 2016), and most of all, working for social justice (Farmer, 1995). Examples of social justice include the South African Surplus People Project, which was originally founded to show the effects of forced removals on people and communities through research and to improve the socio-economic conditions of victims through advocacy; the work of Partners In Health, a medical nonprofit which has been instrumental in reducing the costs of TB therapy in places like Peru, Russia, and Rwanda, and in advocating for universal access to treatment for drug-resistant TB; and a recent decision by South Africa's High Court, which will allow a landmark class action suit seeking damages from gold mining companies for up to half a million miners who contracted silicosis and

Fig. 4. Open Windows Campaign sticker. Source: Western Cape Government.

tuberculosis as a result of their work (Mojapelo et al., 2016).

One final means of intervention merits discussion: a critical theory of emerging infectious disease. A 2014 article, "Tuberculosis Control in South Africa: Successes, Challenges and Recommendations", makes no mention of poverty, racism, or inequality as challenges or targets for intervention (Churchyard et al., 2014). In contrast, the World Health Organization's *End TB Strategy* (World Health Organization, 2015b) devotes significant attention to social protection (Siroka et al., 2016) and poverty alleviation (Rocha et al., 2011) as part of a broad strategy to contain TB. The fetishism of epidemiological figures in the former is an example of how 'global health' statistics (knowledge) (Adams, 2013) potentially bewitch the public into a one-dimensional (Marcuse, 1991), apolitical understanding of TB burdens—an understanding which exaggerates claims about the effectiveness of solving social ills solely through medicine (Dubos and Dubos, 1952) while simultaneously neglecting how power (e.g., institutional racism) integrates with disease dynamics (Mayer, 1996; Richardson and Polyakova, 2012). As Levins and Lewontin write, contemporary science represents an "alienated worldview [that] captures a particularly impoverished shadow of the actual relations among phenomena in the world …" (Levins and Lewontin, 1985). A critical theory of emerging infectious disease can help us pierce the "unthought categories of thought" (Bourdieu, 1990) that are supplied by professional education—a *déformation professionelle* (Carrel, 1935) indeed—and serve to frame both our understanding of disease causation and the means we employ to prevent and treat emerging infections. By this, we do not aim "to diminish the role of the biomedical sciences in the theory and practice of medicine but to supplement them with an equal application of the social sciences in order to provide both a more comprehensive understanding of disease and better care of the patient. The problem is not 'too much science,' but too narrow a view of the sciences relevant to medicine" (Eisenberg and Kleinman, 1981).

5. Summary

The failure to control TB in an era of effective prophylaxis and treatment suggests that the millions of dollars poured into chemical solutions (e.g., the Thibela study) (Churchyard et al., 2014) will continue to fail until environmental interventions are given equal priority (Yates et al., 2016) and, most importantly, the gross inequalities endured by black South Africans are met head-on.

Fig. 5. Butaro Hospital in Burera District, Rwanda. Source: Partners In Health.

6. Limitations

We did not ascertain whether co-commuters had active TB, but instead relied on probabilities given the exceptionally high TB prevalence in South Africa; however, we are currently working on a portable device to capture viable airborne TB bacilli (based on an aerosol sampling chamber (Wood et al., 2016) our group has developed).

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