Social Science & Medicine 69 (2009) 1674–1680

Contents lists available at ScienceDirect

Social Science & Medicine

journal homepage: www.elsevier.com/locate/socscimed

Tuberculosis and syndemics: Implications for Pacific health in New Zealand☆

Judith Littleton*, Julie Park

Department of Anthropology, University of Auckland, Auckland, New Zealand

ARTICLE INFO

Article history:
Available online 27 September 2009

Keywords:
Syndemics
Tuberculosis
Diabetes
Pacific
Populations
New Zealand
Review
Policy
Social determinants

ABSTRACT

Syndemics have been conceived of as a way of approaching the multiple levels of causation and linkage between two or more health conditions and their socio-political environment. Our aim in this paper is to use the established literature on syndemic relationships to examine possible interactions involving tuberculosis. In particular, we explore the linkages between tuberculosis and diabetes mellitus which, we argue, is of particular relevance to Pacific populations resident in New Zealand. Reviewing current literature, we identify multiple synergies between these two diseases whereby their mutual presence has an amplified negative effect. Both conditions interact with other practices and aspects of the broader political economic context such as smoking, housing, and nutrition. A syndemic approach to Pacific health is argued as an effective way to address research, policy and prevention questions.

Introduction

In 2003, Singer and Clair pointed to the need for studies that "seek to determine the sets of health and social conditions likely to give rise to syndemics in that they tend to emerge under health conditions that foster the occurrence of multiple epidemics in a population (2003: 434)." In this paper we analyse the known and probable syndemic interactions around the focal point of tuberculosis (TB) and the applicability of a syndemic approach to a study of TB among Pacific peoples resident in New Zealand. In particular, we focus on synergistic relations relevant to Pacific populations e.g., TB and Type 2 diabetes. This noxious combination characterizes the history of health of many indigenous peoples beyond the Pacific.

While TB rates in New Zealand are relatively low, there are major disparities by ethnicity (Das, Baker, & Calder, 2006) with disproportionately high rates among elderly Pacific peoples, increasing incidence among young Pacific adults, and active transmission to children and infants (Voss et al., 2006). Given the significant clustering of health problems in this population, it is argued here that there are reasonable grounds for examining potential linkages between TB and other conditions affecting Pacific peoples in New Zealand.

What is a syndemic?

The term syndemic refers to 'a set of interactive and mutually enhancing epidemics involving disease interactions at the biological level that develop and are sustained in a community or population because of harmful social conditions and injurious social connections' (Singer & Clair, 2003: 429), for example, the TB-HIV/AIDS-malnutrition syndemic occurring in parts of Africa (van Lettow, Fawzi, & Semba, 2003) or the syndemic of whooping cough, tuberculosis and influenza that affected indigenous Canadian populations in 1927 (Herring & Sattenspiel, 2007). The term was first used to describe the three way interaction between substance abuse, violence and HIV/AIDS among inner city poor in the US (Singer, 1994) and "syndemic" has proved to be one of those useful conceptual frameworks which contribute to understanding and improving global public health (Nichter, 2008). It has been taken up by the influential Centers for Disease Control (Syndemics Prevention Network (SPN), 2005) as well as many health social science researchers. The concept includes not just the afflictions but the forces that cluster those afflictions in persons, places and/or times. Recognition of a syndemic is thus the result of a theoretical orientation that emphasizes interactions and does not consider "diseases as discrete, boundable entities… but more fully conceives of disease both in terms of its interrelationships with

☆ This work has been funded by the University of Auckland Faculty of Arts Research Fund and the New Zealand Health Research Council. We would like to thank our research assistants and summer scholars involved in various aspects of this research: Laura McLachlan, Gina McFarlane and Christina Johnston. We are also grateful to the editor and reviewers for their comments on this manuscript.

* Corresponding author. Department of Anthropology, University of Auckland, Private Mail Bag 92019, Auckland, New Zealand. Tel.: +64 93737559; fax: +64 93737441.
E-mail address: j.littleton@auckland.ac.nz (J. Littleton).

0277-9536/$ – see front matter © 2009 Elsevier Ltd. All rights reserved.
doi:10.1016/j.socscimed.2009.08.042

noxious social conditions and social relationships, and as one form of expression of social suffering (Singer & Clair, 2003: 434)." One goal of a syndemic orientation is to understand the multi-level, interactive conditions that lead to community health (Rock, Buntain, Hatfield, & Hallgrimsson, 2009). It acknowledges that the burden of disease in most societies is borne by those who are the most marginal and over time this burden becomes still more concentrated due to the additive effects of intergenerational deprivation.

Embedded in a syndemic orientation as used by anthropologists is the framework often called critical medical anthropology, exemplified by the work of Singer himself, Baer and many others including Farmer. In 2000 Farmer addressed the questions of why, despite effective therapies, TB remains as a leading cause of death in young adults worldwide (Farmer, 2000). Using the TB life-stories of three young adults from Haiti, Peru and the US, he demonstrated how intra-national political economic conditions, international forces such as World Bank structural adjustment programmes and everyday practices of class, race and gender-based discrimination, alongside the characteristics of health care provision (or lack of provision) and individual, family and community circumstances help to explain the persistence of TB. Thus while a syndemic orientation directs our attention to physiological interactions between diseases it also alerts us to high level interactions between the causes of causes incorporated in Singer's 2009 definition: "the concentration and deleterious interaction of two or more diseases or other health conditions in a population, especially as a consequence of social inequity and the unjust exercise of power" (Singer, 2009a: 996).

Herring and Sattenspiel (2007) used this concept to explore the relationship between infectious diseases and social and economic conditions in northern Aboriginal populations in Canada. They pointed out that while synergism between two or more pathogens has long been recognized, "social influences are equally important in determining risks of disease transmission" and that therefore a syndemic perspective unites a "long recognized biological phenomenon with an explicit social, economic, and political viewpoint, facilitating a broader discussion of cocirculating pathogens within varying social contexts" (Herring & Sattenspiel, 2007: 194). This treatment used as its core an explicit and classical ecological model of the web between pathogens, human host and environment. However, following Krieger (2001), we would re-theorise the "human host" as an active agent rather than a passive host.

Different authors give different weight to the effect of synergism. While it is at the core of work by Herring and Sattenspiel (2007) and Homer and Milstein (2004), Ventura and Mehra's (2004) analysis of the "heart failure syndemic" in Latin America focused more on the clustering of risk factors than on a specific synergism between them, resulting in a proposal to tackle the separate medical risk factors first before turning their attention to the social conditions (Ventura & Mehra, 2004: 388). This can be contrasted to Stall et al. (2003) who pointed out the need to combine services to avoid focus upon a single condition and to Homer and Milstein (2004) who emphasised the prior importance of addressing social conditions. The "heart failure" syndemic gives precedence to a single condition rather than explicitly detailing the pathways that make for meaningful programmes which could tackle multiple suffering.

Links between and concentration of health-related conditions occur for many reasons and amplifying synergies may occur at multiple levels. Freudenberg, Fahs, Galea, and Greenberg (2006) considered tuberculosis, HIV and homicide a syndemic in the context of the 1975 fiscal crisis in New York City. Their analysis suggested that:

> Although each of the three epidemics had its own dynamics, city, state and federal decisions about drug treatment, primary health care, and housing worsened all three, and the policy-driven deterioration in living conditions expanded the size of the population most vulnerable to these health problems (Freudenberg et al., 2006: 429).

While the epidemics are tied by shared underlying social determinants, their analysis highlighted direct and amplifying relationships between the three conditions which is at the core of a syndemics as opposed to a social determinants of health approach, which also typically focuses on multiple factors but not necessarily on their multi-level interactive pathways.

Identifying syndemic conditions

The identification of clustering and co-morbidity is the first step in identifying syndemic conditions (Singer & Clair, 2003). However, the importance of a syndemic perspective is that it refers not just to the co-occurrence but forces attention to the consequences of those biological interactions. The list of the modes of interaction has been broadening over time (e.g., see Singer et al., 2006). But in all of these cases of biological interaction social conditions have a determinant importance. As they write "syndemics are not merely co-occurring epidemics in populations … They also involve the interaction of diseases or other adverse health conditions" (Singer & Clair, 2003: 429).

A syndemic may not be simply the result of contemporary co-occurrence of conditions but of the co-occurrence of conditions within individuals and populations over the space of an individual's life course or intergenerationally. For example, Noymer's work suggests that exposure to TB early in life enhanced the risk of dying from the 1918 influenza epidemic later in life (Noymer, 2008). Another historic study of the 1918 epidemic also showed that such excess mortality may be very unevenly spread due to heterogeneity between households in the one community (Herring & Sattenspiel, 2007). This has been described by Schell (1997) in his work on lead exposure. He demonstrated how culture distributes stressors and buffering mechanisms unevenly across society and over generations, which he referred to as "culture as risk-focusing."

Bartlett (2007) has eloquently spelt out how, regardless of biology, failure to examine phenomena as linked can be disadvantageous to health care efforts. As he wrote in relation to TB and HIV (without reference to the term syndemic):

> experts in TB and experts in HIV infection live in different worlds, obtain grants from different sources, write for different journals, and go to different meetings. This great divide applies to clinical care, research, and training; it is lessened by the overlap between the two diseases but not as much as it should be. (Bartlett, 2007: S125)

Despite the fact that HIV and TB are now widely recognized as a syndemic, the institutions and professional worlds were formed long before a syndemic orientation was adopted (see Singer, 2009b).

Singer and Clair (2003) have pointed to the need for analyses of the health and social conditions that foster syndemic interactions. There is also a need for basic research concerning how syndemics emerge and function. Stall et al. (2003) suggested that identifying populations (indigenous groups, new migrants) that may be at similar risk is a lead to preventing syndemics. But there is also a need to realize that syndemics are the result of specifically local biologies – diseases will not necessarily operate in the same ways or be understood or identified in the same way in every context

since they are produced by local biological and social conditions (Lock, 1993).

Numerous syndemics have been identified (SPN, 2005) and tuberculosis (TB) has been implicated in several. As a chronic condition influenced by the host's immune status, TB infection and disease both facilitates, and is facilitated by, other health conditions and flourishes in noxious social conditions.

Synergisms between TB and HIV/AIDS

The best reported on and most substantiated synergism is between TB and HIV/AIDS. The links between TB and HIV/AIDS have been noted for several decades, with an increased incidence of TB being highly correlated with AIDS prevalence and HIV infection recognized as an important factor influencing the conversion of latent tuberculosis infection (LTBI) to active TB disease (TBD) in many situations (Albalak et al., 2007), but not all. Described as a "synergy from hell" (Bartlett, 2007:S124), the interaction between TB and HIV/AIDS occurs at the population level (in areas of poverty), at the institutional level (as a failure of public health services) and at the cellular level (Raviglione, Narain, & Kochi, 1992; Zhang, Nakata, Weiden, & Rom, 1995). More recent work has added to an understanding of the multiple linkages not just directly between HIV and TB (see McShane, 2005) but implicating other conditions, for example, malnutrition (van Lettow et al., 2003).

Many epidemiological studies (e.g., Albalak et al., 2007; Rose, Sinka, Watson, Mortimer, & Charlett, 2002) provide essential statistical and demographic information and analysis which substantiate this relationship at a national level and demonstrate geographic and population level differences in risk, and trends over time. Such studies tend to focus on TB and HIV control as the means to reduce co-infection.

More ecologically focused work concentrates on the underlying mutual causality for both infections. For example, Antunes and Waldman (2001) consider the statistical association of TB with other population characteristics including HIV, household crowding, SES and a history of migration in São Paulo, Brazil. Taking a more ecologically oriented approach they find both household crowding in terms of people per bedroom and smallness of the house to be associated with TB risk. However they do not develop a fully-fledged ecological model, but analyse the individual contributions to TB mortality of each separate association. Wallace and Wallace (2003) similarly analysed at a neighbourhood level the relationship between declining public services, in particular the New York fire services, and the epidemics of TB and HIV during the 1980s. The recent emergence of MDR-TB and of XDR-TB increase the threat of convergence with the HIV epidemic (Bartlett, 2007; Wells et al., 2007).

Association between TB and tobacco

While TB and HIV/AIDS is the most widely recognized synergism other conditions have been implicated in syndemic interactions with TB. The association between tobacco and tuberculosis has been securely established and even though the effect may be quite small, because of the great prevalence of tobacco smoking, the overall impact may be substantial (Bates et al., 2007; Chiang, Slama, & Enarson, 2007; Lin, Ezzati, & Murray, 2007; Pai et al., 2007; Slama et al., 2007). A number of possible pathways are under consideration including the impact of smoking on host immunity (Wang et al., 2007). In Wang et al.'s study ever-smokers were less likely to complete TB treatment due to their older age, higher rates of co-existing disease (most frequently diabetes mellitus), higher rates of treatment related hepatotoxicity, as well as their more difficult economic and transport circumstances (Wang et al., 2007). Lin et al. (2007) also suggest that the links between TB risk and passive smoking and biomass fuel combustion should be further explored. In addition, smoking may increase the risk of contracting other conditions such as HIV infection (Pai et al., 2007), and diabetes (Willi, Bodenmann, Ghali, Faris, & Cornuz, 2007). The co-existence of multiple diseases with smoking points to the possibility of syndemic linkages.

Synergisms between TB and diabetes

Of particular concern in relation to Pacific peoples given disproportionately high rates of both conditions is the potential synergism between TB and diabetes mellitus (DM). The evidence for this link exists at both the epidemiological and physiological level and is summarized below.

The epidemiology

A link between TB and DM (both Type 1 and 2) has long been hypothesized. A comprehensive review by Root (1934) summarized a series of autopsy studies undertaken from the late 1800s on. As he stated "During the latter half of the nineteenth century the diabetic patient appeared doomed to die of pulmonary tuberculosis if he succeeded in escaping coma." (Root, 1934: 1). However, such data are difficult to interpret given the differences in age distribution between tuberculosis (at this time a disease of younger adults) and diabetes (primarily older adults), as well as differences in diagnosis, secular trends in mortality etc. In disaggregating all of the potential confounders, Root concluded that "active tuberculosis occurred in diabetics at autopsy between two and three times as frequently as expected." The link was also seen in children with diabetes (now known as Type 1 diabetes): a Massachusetts study of school children (Pope cited in Root, 1934: 7) showed that diabetic children had 13 times the prevalence of pulmonary TB as non-diabetic children, 16 times among adolescents (15–19 years). Further confirmation of elevated TB rates in diabetic patients was found in a radiographic study and among the death data despite falling TB mortality rates at the time. What Root was not sure about was the nature of the link. His data suggested that the presence of DM created a favourable environment for TB not vice versa but he could not differentiate between whether DM created a favourable environment for the TB bacillus to survive or whether it was the lowered resistance associated with DM that prevented an effective immune response to TB. He did, however, rule out possible causes such as linked inheritance of genetic susceptibility to tuberculosis and diabetes, exceptional circumstances, or social conditions as sole determinants of the relationship.

The ancient recognition of an association between TB and DM (Restrepo, 2007) had almost dropped from notice by the second half of the 20th century because of the successful treatment of DM and later, of TB. While Root's observations referred mainly to Type 1 DM, the association remains and its recognition is re-emerging with the global pandemics of Type 2 DM (90–95% of all DM) and TB, as testified in editorial comment in leading medical journals (Dixon, 2007; Restrepo, 2007).

Systematic reviews of studies exploring whether diabetes (Types 1 and 2) was linked with an increased risk of TB (not vice versa) found that the two diseases are associated across populations (Jeon & Murray, 2008; Stevenson et al., 2007). There was only one exception to this, a study by Dyck et al. (2007) which found a link among Saskatchewan women less than 60 years old but not a corresponding risk for males. Meta-analysis of cohort studies revealed that "compared with people who do not have diabetes, people with diabetes have an approximately three-fold risk of developing active TB. Higher increases in risk were seen

among younger people, in populations with high background TB incidence and in non-North American populations" (Jeon & Murray, 2008: 1098).

The extent of risk is heterogeneous across the studies with ethnicity and age being two significant factors. In a Californian study Pablos-Méndez, Blustein, and Knirsch (1997) found that the risk of diabetes associated with TB increased among foreign-born patients, or among patients who lived in areas with many foreign-born people. Particularly notable in this study was that among middle aged Hispanics (25–54) the risk of TB attributable to diabetes was similar to that attributable to HIV infection (25.5%). Kim, Hong, Lew, Yang, and Lee (1995) and Ponce-De-Leon et al. (2004) found higher risks in younger or middle aged groups rather than the old.

Physiological mechanisms

Stevenson et al. (2007) highlight a series of possible mechanisms underlying the association between diabetes and TB including:

1. TB possibly contributing to DM through chronic inflammatory effects predisposing people to DM,
2. DM facilitating reactivation of TB infection due to multiple effects on the immune system response including impairment of macrophage and neutrophil functions and reduction of the Th1 cytokine response,
3. mutual underlying causation (e.g., disorders of Vitamin D metabolism are linked to both TB and DM) and contributing factors (e.g., exposure to smoke), and
4. treatment complications between the two conditions resulting in a longer period of infectivity and more difficult glucose control (e.g., renal insufficiency, drug effects).

[In electronic version: see web supplementary data]

These interactions operate at multiple levels and not simply in the direction of DM creating susceptibility for TB. However, some linkages remain controversial because (as pointed out by Root, 1934) it is difficult to determine which condition came first. Clinically the interaction is associated with increased infectiousness of TBD, increased severity, a possible increase in atypical symptoms, longer and more difficult treatment, and worse outcomes (Restrepo et al., 2007; Stevenson et al., 2007). These potentially create circumstances for increased likelihood of transmission of TB.

As Restrepo (2007) points out, although at the level of the individual the risk of TB associated with diabetes is less than the risk of TB associated with AIDS, at the population level, because of the high prevalence of diabetes in some populations, the effect on public health can be marked. Recent studies from around the world have indicated between 10 and 30% of patients with TB have possible DM (Alisjahbana, van Crevel, & Sahiratmadja, 2006; Kim et al., 1995; Mugusi, Swai, Alberti, & McLarty, 1990). One estimate of TB with HIV based on multiple case registries is that 40% of TB cases in the US have HIV-1 (Zhang et al., 1995). Those populations with high prevalence of all three diseases present a great challenge, particularly when disease transmission into new previously unexposed populations is facilitated by these interactions, for example the entry of TB into HIV-infected populations (Wallace & Wallace, 2003), or TB infection to children (Voss et al., 2006).

The Texan/Mexican relationship: a parallel to the Pacific?

Many of the studies of the relationship between TB and DM have relied upon epidemiological, clinical or experimental data. One region where the broader relationship between TB, DM and ecological conditions has been analysed is southern US, specifically Texas, and southern Mexico. Restrepo and colleagues have examined records in the Texas border region (both Texan and Mexican sides) and confirmed the positive association between DM and TB. They point out that this link is particularly strong in areas or groups where the incidence of both are very high (Perez, Brown, & Restrepo, 2006; Restrepo et al., 2007). Independently DM and TB are both reported in populations with low socio-economic status. The odds ratio of TB risk for patients with diabetes was found by Perez et al. (2006) to be 1.5 times for non-border regions of Texas compared to two times in the border region. The variation in risk did not hold for Hispanic patients who had over twice the risk of TB regardless of place of residence. In other words, the syndemic interaction of TB and diabetes is likely to be more salient in some populations than others regardless of the biological interconnection between the two conditions. Using some of the same data but extending the study to northeastern Mexican data, Restrepo et al. (2007) confirm the elevated risk of TB among people self reporting with DM but point out a situation that is reminiscent of the TB/HIV interaction in New York: the association with DM creates a different risk group for TB. Rather than the typical risk group for TB in the region (males, history of incarceration or homelessness), TB-DM cases are more likely Hispanic females, more than 40 years old. DM is introducing a new subset of people to TB disease and more effectively because of the longer and more severe infectivity associated with TB-DM. In southern Mexico the same elevated risk of TB for people with DM has been identified (Ponce-De-Leon et al., 2004). Using molecular epidemiology they have managed to confirm that the heightened risk is due to both reactivation of latent TB infection as well as recent transmission.

What characterizes these populations apart from their health profile is their relative poverty and the underlying social conditions that create risk for infectious and chronic disease. While these authors point to the international complications of a link between TB and DM, particularly in countries with high rates of both conditions, they also point to an aspect of syndemic theory – that understanding what makes a population at risk of a syndemic may allow predictability. In this respect our attention has been drawn to another population experiencing relatively elevated rates of TB and epidemic rates of DM: Pacific people resident in New Zealand.

TB: a syndemic orientation in the Pacific

The vast majority of Pacific people resident in New Zealand derive from the islands of Polynesia. At the last census (2006) they comprised nearly 7% (265,974) of the New Zealand population of approximately 4.15 million (Statistics NZ, 2006). Although nearly 20 distinct language and culture groups are represented, approximately half are Samoan. After a period of high migration from approximately 1950 to 1970, most Pacific people in New Zealand are now New Zealand born and two-thirds live in the Auckland area. In the contemporary setting, there is a great deal of travel between the various islands and New Zealand and migration especially from the islands to New Zealand still continues. Pacific peoples in New Zealand tend to live in more impoverished areas, earn lower incomes, are less likely to be employed and generally have worse health statistics than the population as a whole. Nonetheless, segments of the population show high achievement in education, the arts, sports and business (Ministry of Health [MoH], 2007).

In this section we review the available information about tuberculosis and Pacific people in New Zealand and follow this with an overview of those social conditions, other diseases and practices that international research suggests interact syndemically with tuberculosis. The reason for using a syndemic orientation to

examine TB among Pacific peoples (apart from the apparently disproportionate rates) is that two preconditions are observable. First, there is evidence of mutual causality between TB and other conditions (e.g., household crowding, Baker, Das, Vengopal, & Howden-Chapman, 2008), and second, there is biomedical evidence of reciprocal and interdependent effects between TB and other conditions, in particular diabetes.

Current TB rates in New Zealand are partly the result of historical experiences which have occurred in diverse locations as well as in New Zealand. A molecular typing study of TB in New Zealand between 2003 and 2008 (Sexton, Perera, & Pandey, 2008) found the average annual notification rate of TBD for Pacific peoples was 26.2 per 100,000 during these five years, around three times the average national rate.

Pacific communities in NZ are not experiencing a decline in TB notification rates and some cohorts have increasing incidence (Das et al., 2006). In some cases, reported rates in NZ are higher than reported rates in the home islands (Ng Shiu, Park, & Kearns, 2008). There is evidence of active transmission of TB among some Pacific groups in NZ despite its preventable nature (Sexton et al., 2008). Public health bodies have faced significant barriers to contact tracing and ensuring effective treatment, with people's mobility and stigma being cited as major difficulties (Calder et al., 2000). While high rates of TBD among Pacific elders today is partly a reflection of the historical pattern of tuberculosis in the Pacific and also among those who migrated to New Zealand in the 1960s and 1970s, active transmission to children is a marker of current living conditions and life situations.

Given the high rates of diabetes among Pacific populations and the high rates of transmission of LTBI and conversion to TBD in certain circumstances, we hypothesise that TB and diabetes are part of a syndemic affecting particular Pacific groups.

Why might a syndemic perspective be useful?

The current epidemiological profile of Pacific peoples, particularly in New Zealand, highlights the potential for synergism between TB, DM, Vitamin D deficiency, tobacco smoking, crowding and poverty. It is possible to rule out HIV as a major syndemic condition because the total numbers of New Zealand Pacific men and women diagnosed with HIV between 1996 and 2007 is small, (39 men and 21women) (AIDS Epidemiology Group, 2008) although it is significant elsewhere in the Pacific.

Diabetes is a much more potent potential interactor in this population. The epidemic of DM in NZ is a concern for the population as a whole, and among Pacific peoples there is a prevalence about three times higher than for the total population (MoH, 2008a: 26). In addition, Type 2 DM seems to have an earlier onset in Pacific peoples. In the adult population, Pacific women have self-reported, age-standardised diabetes prevalence rates of 6.2% (25–44 years) and 20.1% (45–65 years). This compares with rates of 1.4% and 6.2% for the comparable national age groups of women (MoH, 2005: 8). Sundborn et al. (2007) estimate a total DM prevalence rate of more than 23 per 100 for Pacific people aged 35 and over.

As noted above, Vitamin D deficiency appears to be implicated in both tuberculosis and diabetes. Recent research in New Zealand indicates vitamin D insufficiency in the population at large, but particularly among Pacific Islands and Maori people. The 1997 adult nutrition survey found the mean blood levels of 25-hydroxyvitamin D in New Zealanders of 15 years and over was 50 nmol/L, which was 20 nmol/L lower than US residents in similar latitudes. Maori levels were lower at 42 nmol/L and Pacific peoples were a concerning 37 nmol/L (Rockell, Skeaff, Williams, & Green, 2006; Scragg et al., 1995). The two major associations of Vitamin D levels in a New Zealand-wide sample were ethnicity and season.

The links between TB and indoor air pollution, particularly by tobacco smoke, as well as between TB and smoking and diabetes and smoking are now securely established and relevant for Pacific populations. One in five (19.9%) New Zealanders in the 2006/2007 Health Survey were smokers, most smoking at least once a day. There were no gender differences in age-adjusted smoking prevalence or frequency. While there are likely to be major differences within the "Pacific" grouping (Rasanathan & Tukuitonga, 2007), smoking rates in 2006 were 24% for NZ Pacific men, and 29% for NZ Pacific women. This compares with a "European/other" prevalence rate of 18.6%. Young Pacific people in New Zealand have reported smoking rates of 10.2% for males and 14.5% for females of 14 and 15 years (MoH, 2007). Current smoking is three times higher in the most deprived areas, compared with the least deprived, after age adjustment (MoH, 2008a: 64). In the context of household crowding and socio-economic deprivation (Baker et al., 2008), exposure to second hand smoke in the home is significantly higher for Pacific children and non-smokers.

Syndemic interactions play out over a life time and across the generations. Drawing on a large birth cohort study of Pacific Islands families in Auckland New Zealand, Carter, Percival, Paterson, and Williams (2006) found that mothers who smoked in the final three months of pregnancy have a much greater and dose-responsive risk of having a low birth weight or small for gestational age baby. They note that because Pacific infants tend to weigh more than infants from other ethnic groups in New Zealand, some of the effects of smoking on birth weight may be masked. Adverse effects of low birth weight are well documented and include increased risk of childhood obesity, Type 2 DM, and cardiovascular disease. The implications for tobacco use over a person's life time by parents, others living in the household and the individual, for both TB and DM, seem to be worthy of close investigation in a syndemic framework.

Finally socio-economic status via routes such as household crowding and food insecurity provide background conditions for the transmission of TB and development of DM. Household crowding has been demonstrated to be associated with a higher incidence of TB (Baker et al., 2008). According to the NZ Ministry of Health, 27% of Pacific peoples (cf. 8% of the national population) live in severe hardship, and 15% live in significant hardship. In addition, Pacific people are less likely to own their own homes (26% cf. 55% of the national population) and are more likely to live in crowded housing than the population at large (MoH, 2007). Food security and the ability to provide a balanced diet is a struggle for many Pacific families (MoH, 2008b: 19–20).

So where is the syndemic?

If the state of Pacific health in New Zealand can be theorized as a result of a syndemic in which TB and diabetes are implicated, why is the syndemic effect not widely recognized? Taking TB and DM as our focus, we suggest that there are several reasons for this, to do with difficulties of diagnosis as well as the division of responsibilities for the two conditions.

A retrospective review of 600 death certificates for 1999 in Christchurch (Chen, Florkowski, Dever, & Beaven, 2004) found that documented DM was "under-reported on more than 50% of death certificates and not compensated by NZHIS [New Zealand Health Information System] coding". The authors report that only 7.8% instead of 43.8% were recorded as having had diabetes. There is no reason for suspecting that the situation in Christchurch is any different from elsewhere in New Zealand. When this under-recording for diabetes on death certificates is coupled with the results of a review of cases of tuberculosis found at autopsy the possibility of co-morbidity beyond that suggested by routinely

collected health statistics presents itself. Lum and Koelmeyer (2005) reviewed 13,866 cases in the Auckland Coronial Autopsy Service between 1994 and 2004 and found that of the 30 cases of tuberculosis 70% were undiagnosed in life. Especially notable in this Auckland study was that 14 cases (47%) were "Polynesian". Over half of cases of all ethnicities were over 65 years of age. Given the under-recognition of both conditions it is quite explicable that the linkage may not be apparent.

Furthermore, in their international survey Jeon and Murray (2008) suggest there may be negative confounding factors in the relationship between TB and DM such as improved nutrition and TB control as well as age. The relationships between the two conditions are more easily discernible amongst the younger age group while the impact among the elderly may be confounded or masked by the widespread nature of impaired glucose metabolism and differential mortality (Jeon & Murray, 2008).

We hypothesise that there may also be a structural reason for a lack of recognition. In the TB control guidelines, chronic renal insufficiency (a result of diabetes) is identified as a potential risk for TB as are the difficulties associated with treating both TB and DM at one time. Our own ethnographic research has revealed that Public Health Nurses and Doctors routinely treating TB patients are well aware of high levels of diabetes in the clinical setting. However, in the Ministry of Health TB is managed under the Public Health (infectious diseases) Division while DM is managed not only in a different division (Clinical /Personal Health) but as a non-communicable, chronic disease. As Bartlett (2007) points out in relation to TB and HIV, the divide between clinical care, research and training in conditions can lead to a failure to examine phenomena as linked.

While the relative risk of TBD from DM is less than that from HIV, the widespread prevalence of DM makes the linkage particularly salient (Stevenson et al., 2007). Jeon and Murray (2008: 1098) calculate that the relative risk of developing TBD among diabetics is approximately three times greater than among a person without DM. This means that approximately 67% of active TB cases in a diabetic population can be attributed to DM (the attributable risk fraction, Fig. 1). It is then possible to calculate the population attributable risk fraction which is the proportion of disease (TBD) in the total population attributable to exposure (in this case to DM). Jeon and Murray (2008) calculate that in Mexico where 6% of the population has DM, 11% of cases of TBD among the entire population are the result of diabetes. Using the same formula, we estimate that in New Zealand where diabetes is estimated to affect 4% of the population, 7% of all TBD can be attributed to DM. This is a relatively small population attributable risk fraction. However, among Pacific Island adults aged 35 or more in New Zealand the DM rate is 23.5% (Sundborn et al., 2007) implying that in this age group 31% of the TBD cases in this group may be due to DM. The calculation reflects only part of the synergy between TB and DM (that from DM to TBD) it does not measure the effects of that interaction (e.g., longer infectivity, atypical symptoms, altered risk group, Restrepo et al., 2007) for the individual patient, those in the household and for health services.

Our concern is the clustering of TB in the Pacific population of NZ (and in the Pacific) and its possible relationship to other health problems, as well as living conditions (e.g., household crowding) and diminished life chances (e.g., discrimination, stigma). We see elucidation of how and why patterns of TB are distributed among different Pacific groups and how TB clusters with other conditions, particularly DM, as essential preconditions for effective interventions.

Attributable risk fraction

[the proportion of disease (TBD) in the exposed population (those with TBD and DM) that can be attributed to exposure to diabetes, DM)]:

$$\frac{(RR-1)}{RR}\times 100\% = \frac{3-1}{3}\times 100\% = 67\%$$

where RR = relative risk (i.e. RR=3 for overall strength of association between TBD and DM, Jeon and Murray 2008).

Population attributable risk fraction

[the proportion of disease (TBD) in the total population that can be attributed to DM exposure]:

$$\left\{\frac{P(E)\times RR-1)}{P(E)\times RR-1+1}\right\}\times 100\% = \left\{\frac{0.04\times(3-1))}{0.04\times(3-1)+1}\right\}\times 100\% = 7\%$$

where P(E) = prevalence of exposure (e.g. 4% diabetes prevalence in New Zealand).

Fig. 1. Calculation of population attributable risk of TB disease (TBD) among diabetics (derived from Jeon & Murray, 2008).

Discussion

Syndemic relationships multiply risk. TB and diabetes provide a prime example. While higher TB rates among Pacific Islanders are partly a historical colonial legacy, their persistence and increase may be driven in part by high rates of diabetes, particularly among elders, which not only increases the risk of active TBD but is associated with longer infectivity and harder-to-diagnose symptoms. TB among the elderly is linked to active transmission to the young (Voss et al., 2006) so that syndemic relationships between TB and DM in adults amplify risks for children, perpetuating disadvantage.

Perspectives that recognize syndemic relationships are necessary in studying the health of Pacific peoples given the extent of clustering of disease and other social conditions. They offer a way of assessing and developing inter-sectoral approaches to health and disease prevention and to understanding the way risks are concentrated and buffered over time in particular people and communities. "A syndemic approach to prevention, meanwhile, focuses on connections among health-related problems, considers those connections when developing health policies, and aligns with forces for social change" (Rock et al., 2009: 991). TB and DM among Pacific peoples in New Zealand are affected by the organisation and priorities of the public health sector and by the policies of many other ministries, local governments, NGOs and communities, including the home island polities, and by the relationships between New Zealand and the island nations. On the basis of this review, we argue that a syndemic approach to research, policy and practice in the field of Pacific health is overdue. A community-based health development approach seems most likely to be effective in promoting systematic awareness and prevention strategies, alongside policy and clinical attention to syndemic interactions.

Appendix. Supplementary data

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.socscimed.2009.08.042.

References

AIDS Epidemiology Group. (2008). *AIDS – New Zealand*. Issue 61. Dunedin: University of Otago Medical School.

Albalak, R., O'Brien, R. J., Kammerer, J. S., O'Brien, S. M., Marks, S. M., Castro, K. G., et al. (2007). Trends in tuberculosis/human immunodeficiency virus comorbidity, United States, 1993–2004. *Archives of Internal Medicine, 167*(22), 2443–2452.

Alisjahbana, B., van Crevel, R., & Sahiratmadja, E. (2006). Diabetes mellitus is strongly associated with tuberculosis in Indonesia. *The International Journal of Tuberculosis and Lung Disease, 10*, 696–700.

Antunes, J. L. F., & Waldman, E. A. (2001). The impact of AIDS, immigration and housing overcrowding on tuberculosis deaths in Sao Paulo, Brazil, 1994–1998. *Social Science & Medicine, 52*, 1071–1080.

Baker, M., Das, D., Vengopal, K., & Howden-Chapman, P. (2008). Tuberculosis associated with household crowding in a developed country. *Journal of Epidemiological and Community Health, 62*, 715–721.

Bartlett, J. (2007). Tuberculosis and HIV infection: partners in human tragedy. *Journal of Infectious Disease, 196*(Suppl. 1), S124–125.

Bates, N., Khalakdina, A., Pai, M., Chang, L., Lessa, F., & Smith, K. (2007). Risk of tuberculosis from exposure to tobacco smoke. *Archives of Internal Medicine, 167*, 335–342.

Calder, L., Hampton, L., Prentice, D., Reeve, M., Vaughan, A., Vaughan, R., et al. (2000). A school and community outbreak of tuberculosis in Auckland. *New Zealand Medical Journal, 113*(1105), 71–74.

Carter, S., Percival, T., Paterson, J., & Williams, M. (2006). Maternal smoking: risks related to maternal asthma and reduced birth weight in a Pacific Island birth cohort in New Zealand. *New Zealand Medical Journal, 119*, U2018.

Chen, F., Florkowski, C., Dever, M., & Beaven, D. (2004). Diabetes mellitus: an underrecognised health problem in New Zealand. *The New Zealand Medical Journal, 117*, U828.

Chiang, C. Y., Slama, K., & Enarson, D. A. (2007). Associations between tobacco and tuberculosis. *The International Journal of Tuberculosis and Lung Disease, 11*(3), 258–262.

Das, D., Baker, M., & Calder, L. (2006). Tuberculosis epidemiology in New Zealand: 1995–2004. *New Zealand Medical Journal, 119*(1243), U2249.

Dixon, B. (2007). Diabetes and tuberculosis: an unhealthy partnership. *The Lancet Infectious Diseases, 7*(7), 444.

Dyck, R., Klomp, H., Marciniuk, D., Tan, L., Stang, M., Ward, H., et al. (2007). The relationship between diabetes and tuberculosis in Saskatchewan. *Canadian Journal of Public Health, 98*(1), 55–59.

Farmer, P. E. (2000). The consumption of the poor: tuberculosis in the 21st century. *Ethnography, 1*(2), 183–216.

Freudenberg, N., Fahs, M., Galea, S., & Greenberg, A. (2006). The impact of New York City's 1975 fiscal crisis on the tuberculosis, HIV, and homicide syndemic. *American Journal of Public Health, 96*(3), 424–434.

Herring, D. A., & Sattenspiel, L. (2007). Social contexts, syndemics, and infectious disease in northern Aboriginal populations. *American Journal of Human Biology, 19*(2), 190–202.

Homer, J., & Milstein, B. (2004). Optimal decision making in a dynamic model of community health. In B. Milstein (Ed.), System sciences, 2004. *Proceedings of the 37th Annual Hawaii International Conference*, (p. 11).

Jeon, C., & Murray, M. (2008). Diabetes mellitus increases the risk of active tuberculosis: a systematic review of 13 observational studies. *PLoS Medicine, 5*(7), 1093–1101.

Kim, S. J., Hong, Y. P., Lew, W. J., Yang, S. C., & Lee, E. G. (1995). Incidence of pulmonary tuberculosis among diabetics. *Tubercle & Lung Disease, 76*(6), 529–533.

Krieger, N. (2001). Theories for social epidemiology in the 21st century: an ecosocial perspective. *International Journal of Epidemiology, 30*, 668–677.

van Lettow, M., Fawzi, W. W., & Semba, R. D. (2003). Triple trouble: the role of malnutrition in tuberculosis and human immunodeficiency virus co-infection. *Nutrition Reviews, 61*(3), 81–90.

Lin, H., Ezzati, M., & Murray, M. (2007). Tobacco smoke, indoor air pollution and tuberculosis: a systematic review and meta-analysis. *PLoS Medicine, 4*(1), 173–189.

Lock, M. (1993). *Encounters with aging: Mythologies of menopause in Japan and North America*. Berkeley: University of California Press.

Lum, D., & Koelmeyer, T. (2005). Tuberculosis in Auckland autopsies, revisited. *New Zealand Medical Journal, 118*(1211), U1356.

McShane, H. (2005). Co-infection with HIV and TB: double trouble. *International Journal of STD & AIDS, 16*(2), 95–101.

Ministry of Health. (2005). *The health of Pacific peoples*. Wellington: Ministry of Health.

Ministry of Health. (2007). *Pacific health factors affecting Pacific peoples' health*. Wellington: Ministry of Health.

Ministry of Health. (2008a). *A portrait of health: Key results of the 2006/07 New Zealand health survey*. Wellington: Ministry of Health.

Ministry of Health. (2008b). *Pacific child health: A paper for the Pacific health and disability action plan review*. Wellington: Ministry of Health.

Mugusi, F., Swai, A. B., Alberti, K. G., & McLarty, D. G. (1990). Increased prevalence of diabetes mellitus in patients with pulmonary tuberculosis in Tanzania. *Tubercle, 71*, 271–276.

Ng Shiu, R., Park, J., & Kearns, R. (2008). Placing the experience of Pacific peoples living with tuberculosis in Auckland, New Zealand. In J. Littleton, J. Park, A. Herring, & T. Farmer (Eds.), *Multiplying and dividing: Tuberculosis in Canada and Aotearoa New Zealand* (pp. 218–231). Auckland: RAL-e 3.

Nichter, M. (2008). *Global health: Why cultural perceptions, social representations, and biopolitics matter*. Tucson: University of Arizona Press.

Noymer, A. (2008). Discussion of Bradshaw, Smith, and Blanchard, a cohort study of tuberculosis and influenza mortality in the twentieth century. *Biodemography and Social Biology, 54*(2), 74–94.

Pablos-Méndez, A., Blustein, J., & Knirsch, C. A. (1997). The role of diabetes mellitus in the higher prevalence of tuberculosis among Hispanics. *American Journal of Public Health, 87*(4), 574–579.

Pai, M., Mohan, A., Dheda, K., Leung, C. C., Yew, W. W., Christopher, D. J., et al. (2007). Lethal interaction: the colliding epidemics of tobacco and tuberculosis. *Expert Review of Anti-Infective Therapy, 5*, 385–391.

Perez, A., Brown, H. S., 3rd, & Restrepo, B. I. (2006). Association between tuberculosis and diabetes in the Mexican border and non-border regions of Texas. *American Journal of Tropical Medicine & Hygiene, 74*(4), 604–611.

Ponce-De-Leon, A., Garcia-Garcia, M.d. L., Garcia-Sancho, M. C., Gomez-Perez, F. J., Valdespino-Gomez, J. L., Olaiz-Fernandez, G., et al. (2004). Tuberculosis and diabetes in southern Mexico. *Diabetes Care, 27*(7), 1584–1590.

Rasanathan, K., & Tukuitonga, C. F. (2007). Tobacco smoking prevalence in Pacific Island countries and territories: a review. *New Zealand Medical Journal, 120*, U1263.

Raviglione, M., Narain, J., & Kochi, A. (1992). HIV-associated tuberculosis in developing countries: clinical features, diagnosis and treatment. *Bulletin of the World Health Organisation, 70*(4), 515–527.

Restrepo, B. I. (2007). Convergence of the tuberculosis and diabetes epidemics: renewal of old acquaintances. *Clinical Infectious Diseases, 45*(4), 436–438.

Restrepo, B. I., Fisher-Hoch, S. P., Crespo, J. G., Whitney, E., Perez, A., Smith, B., et al. (2007). Type 2 diabetes and tuberculosis in a dynamic bi-national border population. *Epidemiology & Infection, 135*(3), 483–491.

Rock, M., Buntain, B., Hatfield, J., & Hallgrimsson, B. (2009). Animal–human connections, "one health," and the syndemic approach to prevention. *Social Science & Medicine, 68*, 991–995.

Rockell, J., Skeaff, C., Williams, S., & Green, T. (2006). Serum 25-hydroxyvitamin D concentrations of New Zealanders aged 15 years and older. *Osteoporosis International, 17*(9), 1382–1389.

Root, H. F. (1934). The association of diabetes and tuberculosis. *New England Journal of Medicine, 210*, 1–13.

Rose, A. M. C., Sinka, K., Watson, J. M., Mortimer, J. Y., & Charlett, A. (2002). An estimate of the contribution of HIV infection to the recent rise in tuberculosis in England and Wales. *Thorax, 57*, 442–445.

Schell, L. M. (1997). Culture as a stressor: a revised model of biocultural interaction. *American Journal of Physical Anthropology, 102*, 67–77.

Scragg, R., Holdaway, I., Singh, V., Metcalf, P., Baker, J., & Dryson, E. (1995). Serum 25-hydroxyvitamin D3 levels decreased in impaired glucose tolerance and diabetes mellitus. *Diabetes Research and Clinical Practice, 27*(3), 181–188.

Sexton, K., Perera, S., & Pandey, S. (2008). *Five years of molecular typing of* Mycobacterium tuberculosis *isolates in New Zealand, 2003 to 2007*. Wellington: Institute of Environmental Science and Research Limited.

Singer, M. (1994). AIDS and the health crisis of the U.S. urban poor; the perspective of critical medical anthropology. *Social Science & Medicine, 39*(7), 931–948.

Singer, M. (2009a). Doorways in nature: syndemics, zoonotics, and public health. A commentary on Rock, Buntain, Hatfield & Hallgrimsson. *Social Science & Medicine, 68*, 996–999.

Singer, M. (2009b). *Introducing syndemics: Toward a new biosocial synthesis in public health*. San Francisco, CA: Jossey-Boss.

Singer, M., & Clair, S. (2003). Syndemics and public health: reconceptualizing diease in bio-social context. *Medical Anthropology Quarterly, 17*(4), 423–441.

Singer, M. C., Erickson, P. I., Badiane, L., Diaz, R., Ortiz, D., Abraham, T., et al. (2006). Syndemics, sex and the city: understanding sexually transmitted diseases in social and cultural context. *Social Science & Medicine, 63*(8), 2010–2021.

Slama, K., Chiang, C., Enarson, D., Hassmiller, K., Fanning, A., Gupta, P., et al. (2007). Tobacco and tuberculosis: a qualitative systematic review and meta-analysis. *International Journal of Tuberculosis and Lung Disease, 11*(10), 1049–1061.

Stall, R., Mills, T. C., Williamson, J., Hart, T., Greenwood, G., Paul, J., et al. (2003). Association of co-occurring psychosocial health problems and increased vulnerability to HIV/AIDS among urban men who have sex with men. *American Journal of Public Health, 93*(6), 939–942.

Statistics NZ. (2006). *Tuvaluan people in New Zealand*. Wellington: Statistics NZ.

Stevenson, C., Forouhi, N., Roglic, G., Williams, B., Lauer, J., Dye, C., et al. (2007). Diabetes and tuberculosis: the impact of the diabetes epidemic on tuberculosis incidence. *BMC Public Health, 7*, 234–242.

Sundborn, G., Metcalf, P., Scragg, R., Schaaf, D., Dyall, L., Gentles, D., et al. (2007). Ethnic differences in the prevalence of new and known diabetes mellitus, impaired glucose tolerance, and impaired fasting glucose. Diabetes heart and health survey (DHAH) 2002–2003, Auckland New Zealand. *New Zealand Medical Journal, 120*(1257), U2607.

Syndemics Prevention Network, CDC. (2005). Syndemics overview. Centres for disease control. URL. www.cdc.gov/syndemics/overview.htm.

Ventura, H., & Mehra, M. (2004). The growing burden of health failure: the "Syndemic" is reaching Latin America. *American Heart Journal, 147*(3), 412–417.

Voss, L., Campbell, M., Tildesley, C., Hay, D., Vaughan, A., & Thornley, C. (2006). Paediatric tuberculosis in a Pacific Islands community in New Zealand. *Journal of Paediatrics & Child Health, 42*(3), 118–122.

Wallace, D., & Wallace, R. (2003). The recent tuberculosis epidemic in New York City: warning from the de-developing world. In M. Gandy, & A. Zumla (Eds.), *The return of the white plague* (pp. 125–146). London: Verso.

Wang, J.-Y., Hsueh, P.-R., Jan, I.-S., Lee, L.-N., Liaw, Y.-S., Yang, P.-C., et al. (2007). The effect of smoking on tuberculosis: different patterns and poorer outcomes. *The International Journal of Tuberculosis and Lung Disease, 11*, 143–149.

Wells, C. D., Cegielski, J. P., Nelson, L. J., Laserson, K. F., Holtz, T. H., Finlay, A., et al. (2007). HIV infection and multi-drug resistant tuberculosis—the perfect storm. *The Journal of Infectious Diseases, 196*, S86–S107.

Willi, C., Bodenmann, P., Ghali, W., Faris, P., & Cornuz, J. (2007). Active smoking and the risk of Type 2 diabetes: a systematic review and meta-analysis. *JAMA, 298*, 2654–2664.

Zhang, Y., Nakata, K., Weiden, M., & Rom, W. N. (1995). *Mycobacterium tuberculosis* enhances human immunodeficiency virus-I replication by transcriptional activation at the long terminal repeat. *Journal of Clinical Investigation, 95*, 2324–2331.