Migrants and tuberculosis: analysing epidemiological data with ethnography

Abstract

Objective: Media portrayals of tuberculosis (TB) in New Zealand are of immigrants who enter the country with active disease and pose a threat to inhabitants, which fosters a popular perception that border control is the best and only response to disease control. This paper reviews both New Zealand and international data on TB rates, causes and transmission among migrant populations to elucidate the precise nature of the link between immigration and TB rates.

Methods: Recent information from scholarly journals on immigration and TB was reviewed. Surveillance data from New Zealand and comparable information from other low-incidence countries were reviewed.

Conclusions and Implications: The importation of active TB is only a minor part of the total TB burden. While effective border control is essential, equally, if not more important, are the circumstances that promote the reactivation of latent TB infection in migrant communities, including migrants' experiences in transit and after arrival, structural conditions, and personal characteristics. For sound prevention strategies, attention needs to be paid to the existence of transnational communities and the conditions for migrants, rather than placing a singular focus on place of birth.

Key words: Medical anthropology, New Zealand, transnationalism, migrant health.

(*Aust N Z J Public Health.* 2008; 32:142-9)
doi:10.1111/j.1753-6405.2008.00191.x

Judith Littleton, Julie Park
Anthropology Department, University of Auckland, New Zealand

Craig Thornley
Auckland Regional Public Health Service, New Zealand

Anneka Anderson
Anthropology Department, University of Auckland, New Zealand

Jody Lawrence
School of Geography and Environmental Sciences, University of Auckland, New Zealand

A popular perception exists that the main source of active tuberculosis (TB) in New Zealand is new arrivals, and that turning them away at the borders is the best response to limit the incidence of TB in New Zealand.[1-3] Publicity, such as that provoked by a New Zealand Member of Parliament in mid-2005 about the deportation from New Zealand of a Korean visitor with a strain of TB that was resistant to virtually all anti-tubercular medication, reinforces this perception.[4]

In this paper we argue for a subtle shift in focus. We acknowledge that people with latent tuberculosis infection do come into New Zealand and urge that prevention strategies pay attention to the complex contexts in which active tuberculosis develops among migrants. We argue that progression from latent to active TB is not inevitable but is contingent upon circumstances in the country of origin, experiences in transit and after arrival in the new country, structural conditions and personal characteristics. This complexity contributes to ecologies that promote or prevent the transmission of TB infection (TBI) and the development of TB disease (TBD) in overseas-born people. In order to understand the high rates of tuberculosis disease among immigrants, the epidemiological data need to be contextualised ethnographically, i.e. in the light of studies of the everyday life experiences of migrants.[5] In doing this, we examine global and New Zealand epidemiological data in the context of a multidisciplinary ethnographic study of tuberculosis among diverse populations in Auckland, New Zealand.[6]

TB among overseas-born residents

In many Western countries, a high and increasing proportion of TB cases derive from foreign-born people (see Table 1). The data sources vary by country but show a consistent trend. In New Zealand, this proportion has increased from 48% to 76%, mirroring the changes in rates of immigration.[3]

In low-prevalence countries, TB rates among foreign-born people exceed rates among the native-born by 10 to 20 times (see Table 2).[7] Rates also vary from area

Submitted: December 2006 **Revision requested:** March 2007 **Accepted:** February 2008

Correspondence to:
Dr Judith Littleton, Anthropology Department, University of Auckland, Private Mail Bag 921019, Auckland, New Zealand. Fax: +64 9 373 7441; e-mail: j.littleton@auckland.ac.nz

to area. For example, in the United States in 2003, foreign-born cases accounted for 80% in Massachusetts, but only 12% in Arkansas.[8] In Australia in 2002, 86% of cases in New South Wales were foreign-born, but only 30% in the Northern Territory.[9] New Zealand is no exception. The incidence of TB (2000-04) varied by District Health Board from 1.9 to 20.9 per 100,000, and the proportion of overseas-born cases also varied (see Figure 1). This means that health workers in different regions within countries may have very different expectations and experiences when it comes to diagnosing TB.

Active TBD in overseas-born people represents four possible scenarios: people with active TBD at arrival that is not detected or reported to authorities; people with active TBD at the time of arrival, but who are known to the health and immigration authorities and treated before and/or after arrival; people who acquire the disease in their destination country after immigration; and people with existing TBI that reactivates at some time following arrival. We discuss these possibilities in the next section of the paper.

Is active TB imported?

As the brief account of the Korean woman visitor suggests, press coverage in New Zealand tends to imply that the first scenario is the biggest risk: people travel to New Zealand with undetected active disease and may infect locals. Analysis of newspaper reporting of TB issues within New Zealand's three largest newspapers between 2002 and 2005 revealed that particular migrant groups, such as foreign students and asylum seekers, were implicated as the 'cause' of the TB 'problem' in New Zealand.[3]

How accurate is this? There are two kinds of indicators of the extent of imported active TB. The first is immediate post-migration health checks; the second is the time lapse between migration and notification.

Table 1: Increases in the percentage of cases of TB among foreign and locally born people in English-speaking countries over time.

Country	Year and source	Cases overseas-born (%)
New Zealand	2005[58]	76.3
	2000[1]	55.6
	1995[1]	47.5
Australia	2003[59]	82.7
	1994[59]	66.4
Canada	2002[60]	67
	1997[61]	64
	1994[61]	57
	1980[62]	35
United Kingdom	2002[63]	67
	1988[63]	45
United States	200[38]	54
	2000[8]	46
	1997[8]	39
	1993[8]	29

Studies of post-migration health checks

One Western Australian study found that of 1,344 immigrants on TB-related health undertakings (i.e. a group suspected of imported active disease on the basis of pre-migration health checks), only 0.5% developed active TB in the three years after arrival.[10] This was in addition to the 5.1% active cases that were detected and treated before arrival.

A San Francisco study with a similar design found that 6.9% (51) had active TB. However, 25% had already been diagnosed in their country of origin and had begun treatment.[11] In both of these groups, rates were low despite all being possibly active cases.

Failure to diagnose before arrival and/or non-reporting cannot be attributed solely to fraud: TB, particularly non-cavity disease, is difficult to diagnose through chest X-ray, and diagnostic facilities in countries of origin are often not optimal. In the Western Australian study, three cases were extra-pulmonary (i.e., not considered infectious, and also more difficult to diagnose), and the other four were pre-symptomatic. In the San Francisco study, only three cases were both smear- and culture-positive.[11]

In New Zealand, as elsewhere, the possibility of active, infectious cases being undiagnosed is largely restricted to those entering the country for short time periods, where screening is not required (as in the case of the Korean woman), those arriving in very early stages of the disease, or those whose disease is missed by screening tests. A study of 900 asylum seekers who were not subject to mandatory health checks detected 0.6% active disease.[12] This is a group for which high rates of active TB could be expected, and its rates were comparable to international rates of high-risk groups.[10,11]

Time between arrival and diagnosis

The second source of data on active TB coming into the country is the period of time between arrival and diagnosis. These data have been collected from multiple sources, hence the time intervals vary by country so are not strictly comparable, although the trends are consistent. Notification within one year of arrival is potentially an indication of imported TBD.[13] Rates of TB diagnosis are high in the first year after arrival, but rates in the second year are often similar or, in some cases, even higher. This pattern of time of residence and notification has been noted to vary by place of origin.[14-16] Zuber et al. noted that about half of the foreign-born people who developed active TB did so more than five years after arrival, and this was common in all of the low-prevalence countries (see Table 3).[17]

In New Zealand, the proportion of those who develop TBD in their first year after arrival in relation to all overseas-born people who eventually develop TBD has been decreasing to just over 20% in 2001.[1] The other 80% of notifications in the overseas-born population are therefore likely to be either reactivations of TBI or recently acquired new infections, as are some of those notified in the first year. If the New Zealand notification data are divided into migrants from high-incidence and low-incidence countries,

a similar pattern can be seen to that in Australia. In New Zealand, about 25% of notifications among migrants from high-incidence countries were in the first year, and 7% in the first two months.[18]

Refugees, migrants and others coming to New Zealand for six months or more (formerly two years or more) all undergo TB screening as part of their health checks, and some refugees begin treatment before arrival. Of the 1,722 cases of active TB notified in the Auckland region between 1997 and 2005, 121 (7.1%) had been initially identified through community-based non-refugee immigration screening (i.e. screening as part of an immigration permit), and 39 (3.2%) were identified as part of other screening (conducted as part of refugee screening, or health care student or worker screening).[19] This combined figure of 10.3% is consistent with the 11% detected through immigrant screening in a specific study of children.[20]

Data from the only refugee reception centre in New Zealand, which is situated in Auckland (population 1.3 million), suggest that in Auckland around a quarter of the notifications in the first year are among refugees.[21] Because all refugees have a six-week stay in the resettlement centre where they undergo a variety of health checks, including Mantoux tests, chest X-rays, and sputum and culture tests if indicated, existing TBI and TBD is particularly likely to be identified and treatment or prophylaxis begun.

Part of New Zealand's humanitarian commitment to accepting 750 quota refugees each year is the creation of 75 places within the quota for those with high medical needs and/or disability in contrast to many other countries (e.g. United States).[22] Hence, as long as refugees come from areas with endemic TB or spend time in high-prevalence situations such as refugee camps, and are sick, stressed and poor both before and after arrival, TBI and TBD are to be expected and systems need to be in place to deal with them. However, even in this most vulnerable of groups, between 40% and 75% of TB cases are likely to be due to the activation of existing infection or new infections, rather than people arriving with active disease based on the time between arrival and notification.

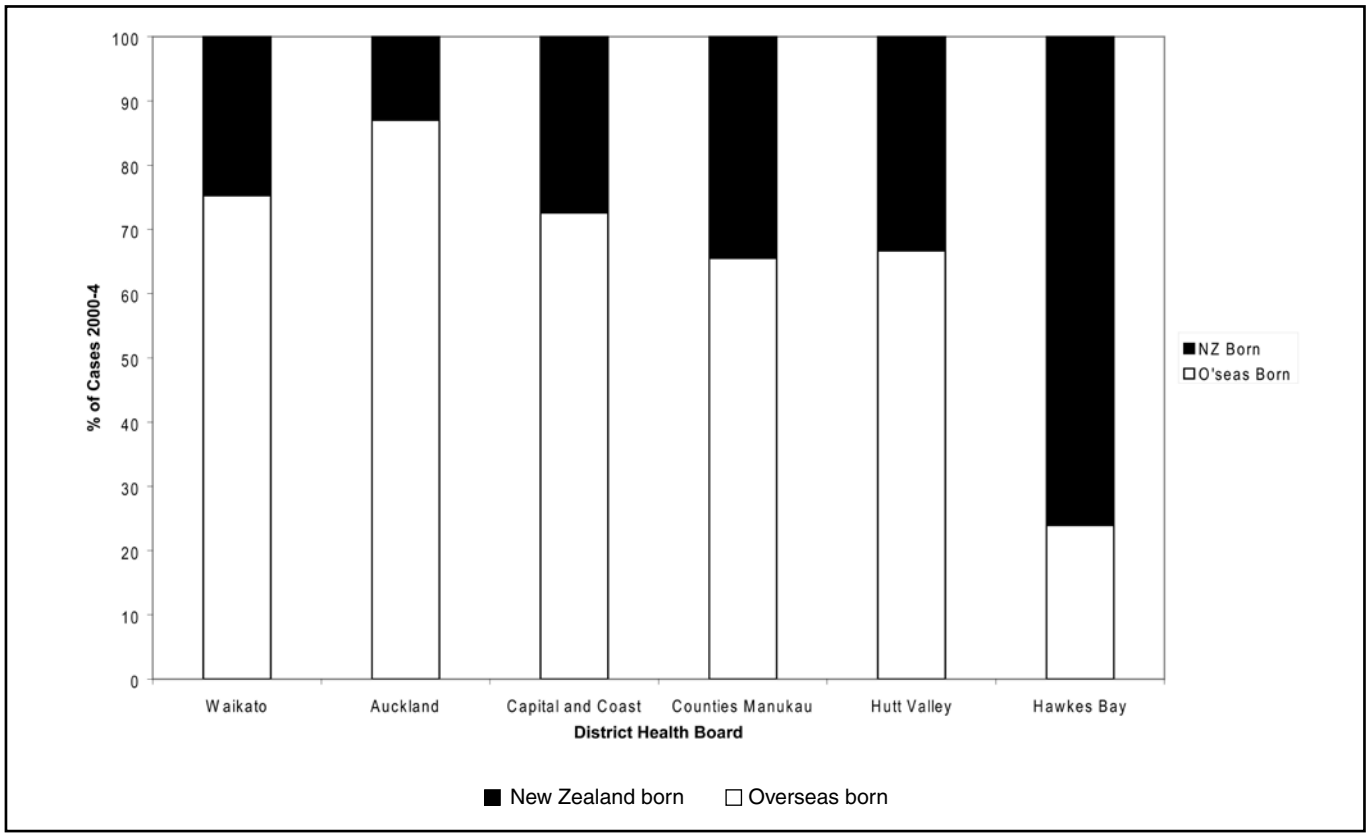
The international picture for Western countries, into which New Zealand fits, is therefore of a relatively small number of documented people travelling across borders with active disease. These people are likely to be escaping traumatic situations, and in New Zealand and other Western countries effective systems of screening and care are already in place.

Reactivation or new infections in overseas-born people

However, despite low rates of active disease upon or shortly after arrival, some groups of overseas-born people have higher rates of disease post-settlement. There are two possible sources for this: activation of latent tuberculosis infection or newly acquired infections.

In terms of these two possibilities, molecular epidemiology is the strongest indicator and suggests that reactivation is the main contributor in overseas-born cases. A study in San Francisco typifies this pattern.[23] Most TB cultures from foreign-born people

Figure 1: Distribution of tubercular disease, 2000-04, by selected District Health Boards according to place of birth, based on ESR data.



contain unique DNA sequences that fail to cluster. The prevalence of clustering is taken as evidence of recent transmission. Recently acquired infections will be shared, whereas old infections that have been reactivated will have unique sequences due to the length of time and geographic distance from the source of infection to its final activation.[24]

In the San Francisco study, all 252 foreign-born cases were initial cases, suggesting reactivation not new infection as the mechanism.[23] Only two were linked to a secondary case. A similar pattern was found in New York City, where Tornieporth et al. calculated that 16% of the foreign-born isolates clustered, compared with 42% of the US-born isolates, suggesting more frequent recent transmission among the US-born.[25] All of the foreign-born clusters were identical to clusters of US origin, and there was an increased likelihood of clustering the longer the period of residence in the United States. So, while 84% of TB among the foreign-born in New York City was from reactivation of infection, those with new infections acquired it from strains already circulating among US TB patients and generally from groups with similar risk factors, such as homelessness or multidrug-resistant TB. In other words, it appears that activation of latent infection creates the majority of TBD among many foreign-born, but the living conditions of some foreign-born people may make them susceptible to locally circulating infection.

Zuber et al. studied the records of all TB cases notified in the United States between 1986 and 1994.[24] Their work showed that persons from countries with high TB-prevalence rates have higher TB rates up to 20 years after arrival in the United States. This study also supports the view that TB in foreign-born, longer-term residents is due mainly to activation of TBI, a finding replicated elsewhere.[26,27]

While DNA studies in New Zealand have not yet been undertaken for a sufficient number of years to produce data comparable with the US studies, it is unlikely that New Zealand would show a different trend.

Are immigrants with TB a threat to locals? Are locals with TB a threat to immigrants?

Several studies point to the continuing low rates of TB among locally born populations, despite high rates among the overseas-born, as evidence of a lack of transmission.[16,28] Das et al. also demonstrate this in New Zealand, arguing that while Pakeha New Zealanders over 40 years of age continued to experience a decline in TB incidence in the period 2000-04, the absolute numbers of TB cases among migrants from high-incidence countries rose.[18] Some outbreaks do occur but tend to point to transmission between linked households and within close communities (e.g. church choir) rather than beyond. Institutional contact (school, prison) is one type of outbreak that may involve multiple ethnicities. This pattern is mirrored elsewhere. In New South Wales, for example, one study of TB points to a small amount of transmission among migrants to household members and not beyond that.[29]

In Chin et al.'s San Francisco study, only 2% (2) of the clusters involved transmission from a person born overseas to a person born in the United States.[23] This finding is confirmed by DeRiemer and co-workers.[11] Overseas and locally born populations had markedly different epidemiologies, with much higher transmission rates among local populations. Borgdorff et al.'s mathematical modelling indicates that there is considerable international variation in local and overseas-born transmission patterns.[30,31] People who have lived for long periods in countries where TB incidence is high are likely to be more resistant to new infection than people not so exposed, but are more vulnerable to reactivation, hence the prolonged mirroring of rates from their place of origin.[23,32] However, some scholars suspect that exogenous reinfection may be under-estimated.[33,34]

Reactivation of tuberculosis infection is still poorly understood, although it is known that conditions compromising host immune response are always implicated.[1,35] Immigrants' own experiences demonstrate one set of reasons why activation occurs. One African refugee participant said:

> *Sometimes I'm thinking too much, for example I had the TB so I believed that TB attacked me because I was thinking too much I was worrying about my family back home a lot sometimes I go to the toilet and I just sitting on the toilet but I was thinking of my family. I have got a big family and I can't afford to fix their problems and I can't afford to send them whatever they want so I was thinking, thinking, thinking a lot.*[36]

Ho's study of Chinese migrants makes a similar point.[37] Poor nutrition, crowding, lack of fresh air, other illnesses, hardship and stress were all identified as part of the process of contracting TB.

Table 2: Current percentage of cases among foreign-born people, and rates of TB among foreign-born compared with locally born people.

Country	Year	TB cases foreign-born %	Rate of TB per 100,000	
			Foreign-born	Locally born
New Zealand[7]	2000-04	64.0	32.30	3.91
Australia[59]	2003	82.7	19.9	1.1
Canada[60]	2002	67.0	19.4	1.9
United Kingdom[63]	2002	67.0	89.7	4.1
United States[a,8]	2004	54.0	22.8	2.6

Notes:
(a) Not including 0.5% of unknown origin.

In New Zealand, many 'Asian' migrants also face difficult settlement conditions, particularly in securing employment and income.[38] As one young male immigrant in his early 20s declared:

> *"Here, even getting a job in a gas station is difficult. In India I would never have done that [worked in a gas station] as I studied engineering to get a job and get paid for it. Here all expectations and no job, you need money to survive and this causes stress. If you can't get a job and don't have money you can't survive". (Young Indian male migrant in his early 20s) (p72)*[39]

Other settlement issues in New Zealand include racial discrimination, language barriers and lack of social support.[40,41] Studies elsewhere have highlighted these issues along with unemployment as significant predictor variables for tuberculosis.[13,32]

There are occasional instructive exceptions to the trend for activation of TBI as the main source of infection. A study of Mexicans in San Francisco indicated that this was one foreign-born group implicated in a San Francisco epidemic of TB – as recipients of infection.[42] Nine of the 43 cases (21%) of active TB in the Mexicans studied had been transmitted by US-born people, whereas only one Mexican had infected (2) US-born people. Neither their Mexican ethnicity nor their birthplace explained their susceptibility to new infections. Migration and settlement, often with undocumented status, had exposed them to the same conditions that promote the transmission of infection and the development of the disease in locally born populations – namely homelessness, poverty, HIV positive status, and drug and alcohol abuse.

In the Auckland region, different migrant populations vary in the extent to which TBD developed following migration represents reactivation of TBI or disease acquired through local transmission. In 2003, 51.7% of TBD cases among Pacific people occurred in people with a history of recent exposure to one or more cases with infectious disease within New Zealand; in contrast, only 6.8% of Asian cases had similar exposure histories.[43] Young Pacific people are experiencing increasing rates of TB.[18,20] This suggests that Pacific peoples (who may be born in New Zealand or in the Pacific Islands) are vulnerable to new local infections as well as to reactivations. This can be attributed to their socio-economic conditions, including uncertain residency status, other diseases and conditions, such as diabetes, that lower resistance to TB and complicate treatment, as well as to historically high TB rates. Thus, while those born overseas and locally usually have separate epidemiologies of TB, in certain situations these connect to the detriment of both.

Borders or transnational neighbourhoods

Other studies of Mexicans have also drawn lessons that are applicable in New Zealand. Wells and co-workers' research in US border States indicated that many Mexicans crossed the US-Mexico border frequently, and when in Mexico travelled widely, indicating that US TB control concentrated only in the border regions of Mexico was misplaced.[44] In New Zealand, frequent border crossing is common for many immigrants, including students studying in New Zealand who travel between school semesters back home, as depicted by a young Chinese man:[38]

> *"Normally [every] half a year I go back to China. Before leaving New Zealand I just ah finish renting, then when I come back [I] rent another place again … Yeah I go back to see my parents yeah". (p54)*[39]

The concept of transnational neighbourhoods, which may span many hundreds or thousands of miles with frequent visits back and forth and other forms of contact between them,[45] is more useful in planning TB prevention than the idea of borders and single crossings. Many other studies, especially in the United States, note that thousands of undocumented migrants arrive every year, along with authorised categories of visitors, business people and students who do not require health checks.[17,46] For these reasons, too, a fixation on borders – rather than on good-quality, accessible health care for all, regardless of immigration status – promotes TB infection.[47] Given the high global rates of TB disease and infection, isolating New Zealand from any contact with overseas infection is impossible.

It is also well to remember that in a nation where 'OE' (overseas experience) is a valued pursuit of young people and many not-so-young, and travel is a hobby or business necessity for many others, overseas strains of TB can easily be brought into New Zealand by the New Zealand-born and by returning long-term residents.

Obviously, screening and treating people who come to New Zealand for appreciable periods is an important component of TB control, but ameliorating the conditions that contribute to reactivation, or that place those born overseas in high-risk situations for new infection, need to be real foci for effort.

Table 3: Time between arrival in country and diagnosis with TB, note different intevals.

Country	Years between arrival and diagnosis (%)						
	<1	1-2	2-3	3-4	4-5	5-9.9	≥10
New Zealand 2001[1,a]	26.7		35.9 (1-5yrs)			38.4 (5+yrs)	
Australia 1997[15]	14.0	11.0	17.0	19.0	38.0		
Canada 2002[60]	12.4	14.9	7.0	4.8	3.2	18.8	38.6
UK 2002[63]	29.0 (0-2yrs)	10.4	8.4	6.1	13.0	33.0	
US 2003[8,b]	20.7		27.9 (1-5yrs)			14.9	36.4

Notes:
(a) Excluding 17% unknown.
(b) Excluding 13.7% unknown.

Ecologies of TB

In the Auckland region in 2003, the rate of TBD in the most deprived areas was six times higher than that in the least deprived neighbourhoods. This gradient was not confounded by ethnicity.[43] This Auckland trend is replicated in the whole of New Zealand.[48]

Whereas media articles often link higher rates of TB in the locally born to poverty, and recognise the implications of other health conditions for susceptibility, when it comes to overseas-born people TB is often attributed to unexamined foreignness or ethnicity.[3] Yet exactly the same situations that promote the development of disease in the locally born promote it in new citizens and residents and create a situation whereby migration and deprivation have a compounding effect.[49] Cantwell, Snider et al., commenting on different exposures to TB, say of ethnic differences in TB rates that they "may be caused by confounding factors, such as low socioeconomic status, crowding and homelessness".[50] In a later study, they focused particularly on race/ethnicity in the United States and its relationship with socio-economic status (SES).[51] They found that SES had a direct effect on susceptibility to TB through crowding as well as a more generalised effect at all levels of SES. They concluded that SES accounted for much of the difference previously attributed to race/ethnicity, a finding supported by a study in Cologne.[52]

Wallace and Wallace and Story and Citron are unequivocal about the negative effects of social disinvestment in their studies of epidemics of TB in New York and London, respectively.[53-55] They also point to the political and other disruptions that cause thousands of refugees and migrants to leave their homes and seek better life chances in new countries as graphically described by one African refugee:

> *I had to leave. We had to leave all the cars and the houses and everything. We had to take nothing even no food, nothing. Just walk. When we came to Kenya, you know, the refugee camp, my husband died there so life was getting really hard …* [36]

While case finding, contact tracing and preventive treatment for those with TBI at risk of developing TBD are clearly crucial parts of any anti-TB program, attention to the life chances of those at risk is equally important. As Barnes argues, based on his review of debates about multifactorial explanations of TB, *Mycobacterium tuberculosis* is a necessary but not sufficient cause of TB disease.[56] Eliminating it would indeed banish the disease, but, because of the extent of latent and hard-to-detect TB, and the difficulties of effective treatment in some cases, this is an unlikely scenario. Reducing the conditions in which TB is transmitted and the factors that promote the conversion from latent to active TB are just as important.

Health care

In Auckland, 66% of TB cases are diagnosed when a patient makes a doctor's visit, rather than through TB screening or contact tracing.[43] Therefore, access to general health services is vital to TB care. Many migrants in New Zealand experience complex issues in accessing health care that can lead to delays in diagnosis (both patient and doctor delays). These include language barriers, lack of understanding of New Zealand's health care system and, particularly for TB, stigma surrounding the disease.

> *Researcher: How do you think people would react if you did tell them?*
>
> *Response: They will run away from me.*[36]

Many migrants who have limited English have to rely on others (children or church members) to act as translators for them when visiting health care professionals. Differences in health care systems between New Zealand and migrants' country of origin can often cause confusion or even suspicion, as highlighted by comments from a Korean immigrant in New Zealand:

> *Yeah, first time I thought that in New Zealand the health system wasn't comfortable, actually … Yes, yeah in Korea ah, normally we go to the big hospital, there is more staff, specialists for health problems. But in New Zealand you need go GP first, and the GP can be quite small, so I can't trust them. Yes, yes just one doctor in small you know clinic (p109).*[39]

In trying to access health care many migrants see multiple providers, which increases the likelihood of diagnostic delay.

Delayed diagnosis is not only dangerous for the patient, but in the case of infectious TB is also dangerous for the patient's family and friends. Even though public health services do not communicate patients' residency status to the Department of Immigration, fear of deportation may inhibit patients coming forward even when they know that they have TB. Uncertainty regarding whether they will have to pay for treatment, and its cost, may also contribute to delay. This combination of barriers presents very real difficulties for speedy diagnosis, effective treatment and comprehensive contact tracing. Media attention, like that provoked by an MP's question in Parliament around the deportation of the Korean woman visitor, creates a climate of fear regarding the immigration consequences of presenting for diagnosis and treatment. Any barrier, no matter how small (even a required 0800 telephone call), can have a major effect on access.[10]

Conclusion and Implications

Portrayals by the media and politicians of immigrants as the 'diseased other' who need to be stopped at the border lest they bring infections to which locals are vulnerable are not only inaccurate but also ineffective and counterproductive. Inaccurate because TB in overseas-born persons rarely appears to infect the locally born; ineffective because it will miss approximately 80% of the TB in overseas-born people; and counterproductive because it makes immigrants and visitors nervous about the security of their tenure and less likely to access health care, and it drives their contacts, so necessary for TB prevention, underground.

Public health needs to tread a delicate line between acknowledging and dealing with the realities of tuberculosis disease in some migrant groups and re-stigmatising these groups and driving tuberculosis underground. Mass media campaigns are clearly not the way to go. However, selective use of community language newspapers in conjunction with community health promotion initiatives as described by Miller[57] for the Indian community in

Auckland are much more likely to be effective, while avoiding unwanted publicity or re-imposing stereotypes.

Attention to TB at the border is an important component of an overall TB prevention strategy. However, contextualising the epidemiological data in people's everyday experiences suggests that just as crucial is a focus on the living conditions, life chances and access to affordable and appropriate health care and TB services for those people who, because of their life history, have been exposed to active TB in the past and who struggle in their present circumstances.

Acknowledgements

This research is part of The Political Ecology of TB project funded by the Health Research Council of New Zealand and the Auckland University Research Fund. We are grateful for the assistance of Auckland Regional Public Health staff, the Population and Health Group (Institute for Environmental Science and Research), Drs Baker and Das, Alison Searle, and our participants. We also acknowledge the constructive comments of two anonymous reviewers.

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