‘ETHNOGRAPHY PLUS’ IN TUBERCULOSIS RESEARCH

Julie Park & Judith Littleton

ABSTRACT

We argue that in some contexts we anthropologists need to go beyond ethnography through being involved in multidisciplinary approaches to research problems and through engaging in a more public anthropology in our ethnographic and other writing. This argument is based on our experiences in coming to an understanding of ‘immigrant TB’ in the context of a social sciences research project about tuberculosis in New Zealand. This paper unpacks some of the complexities around relatively high rates of tuberculosis in overseas-born persons in New Zealand, and suggests that the conditions of settlement, including poverty and discrimination, combine with life history to produce the observed epidemiological patterns. A corollary is that while control of tuberculosis at the border is important, support for migrants, ensuring their social participation, access to health care and their rights to freedom from discrimination are also key.

INTRODUCTION

Our paper derives from an oral presentation at the provocatively titled ‘Beyond Ethnography’ Conference. We argue that, though pivotal, ethnography is not enough in some contexts. Ethnography is still central to social and cultural anthropology, even in the twenty-first century, even in urban areas, even when research is focussed on a topic, not a ‘people’. Nothing can replace that advantage conferred by ethnographic research relationships (James, Hockey and Dawson 1997:13): the compounding interactions between the access to the drama and banality of the processes of everyday life that is the stuff of the theories we develop; and the take on these processes occasioned by our theories (Fitzgerald and Park 2003). But we have found in our recent work a need to go beyond ethnography via two different paths; others will take different directions.
In ‘The political ecology of Tuberculosis in New Zealand’ project, we have found it necessary to tack between ethnography as fieldwork and other forms of data and analyses to focus our questions and to try to answer them. We need history, geography, media analysis, and we need to go beyond social and cultural studies to draw on epidemiology, medical research and the results of various laboratory sciences to be able to make optimum use of the ethnography and to know where to focus our ethnographic research and writing. This is our first movement beyond ethnography. In our second movement, we want to be able to use our ethnographic and other research to construct a more public anthropology. We certainly want to be able to write accessible ethnography. By doing so, we also want to be able to disseminate our work strategically, to make interventions in public debates and political processes. We intend the phrase ‘ethnography plus’ to be understood as both these ways of going beyond ethnography.

In using this phrase we are not suggesting that this is a new trend, nor are we attempting to coin a neologism. Unlike the proponents of the development called ‘critical ethnography’, we believe that all ethnography has the potential ‘to address processes of unfairness or injustice within a particular lived domain’ (Madison 2005: 5, italics in original), and very often does so. Instead, we are suggesting that in some cases of complex, intertwined human phenomena, if we stick with ‘ethnography through thick and thin’, to use Marcus’s (1998) title-phrase, draw on anthropology’s long tradition of inter- and multi-disciplinary work (Helman 2006) and heed the calls for a more public anthropology which have gained momentum in recent years, we can tell better stories more effectively.

A brief account of the introductory remarks during our oral presentation at this Conference illustrates these two aspects. In my (Park’s) prefatory remarks, I noted that a lot of what I do in my life, including in anthropology, seems to be in response to an internal dialogue I hold with ‘Winston Peters’. This is not the living Winston Peters—who at the time of writing was the New Zealand Foreign Minister, a person whom I have never met—but a construction of my own: ‘Winston Peters’, derived from my reading of news media, appreciation of political cartoons, and our project’s analysis of TB and the media (Lawrence, Kearns, et al. n.d.) This ‘Winston Peters’ is the person who strikes terror into the hearts of our Muslim friends with his reported remarks; the person who told us that refugees and asylum-seekers were bringing HIV, TB, rubella and infectious skin diseases into New Zealand (Berry and Watkins 2002: 2); the person who appeals to frightened Kiwis with his slurs on people who are not ‘Us’, invoking the type of national managerial fantasies that Hage (1998: 179)
analysed for Australia: who should ‘we’ let in, and how should ‘we’ govern their lives?

How can [the Prime Minister] have confidence in a Minister of Immigration and a Minister of Health who have allowed to come into this country, and now to clog up the wards in North Shore and Auckland hospitals, hundreds of people with Third World diseases including tuberculosis cases where the wards are busting to overflowing—and 70 percent are immigrants—and how can that be a responsible way of defending the health of the New Zealand people? (Winston Peters, NZ Parliament, 2 Sept 2003 ‘Questions for Oral Answer’)

As I revealed my on-going, hitherto silent, dialogue to the conference audience, I was halted by hoots of laughter and shouts: ‘What do you mean you have an internal dialogue, I shout at the TV/radio!’ ‘Winston Peters’ was revealed as part of an anthropological collective imagination. Simultaneously, the constructed nature of ‘the field’ (Amit 2000) was revealed. Park’s academic ‘home
ground’, the local social anthropology association meeting, has now been incorporated into ‘the field’ for our TB study, as we use this incident as part of our ethnographic narrative. Such transformations are not new (Dyck 2000) and they continually problematise ethnography and the field whether ethnography takes us ‘away’ or brings us ‘home’. The field is not a place or a set of relations that pre-date our ethnographic efforts but is constructed by our field work.

Amit goes on to note that despite ethnography’s emphasis on access to people’s everyday lives through participant observation, this access has often been limited in many areas of lived experience.

The ethnographic ‘field’ therefore, has always been as much characterized by absences as by presences and hence necessitated a variety of corresponding methods – interviews, archival documents, census data, artefacts, media materials and more – to explore processes not immediately or appropriately accessible through participant observation (Amit 2000: 12).

In a context like ours, a study of tuberculosis, ‘the field’ is very fluid. Yet we think it useful to define as ‘beyond ethnography’ the biomedical information, the epidemiology and the DNA studies on which we draw to construct our narrative of enquiry. We recognise that there may be epistemological differences in the bases of these studies and our own. Some of the authors on whose work we draw do appear to assume that the statistics and other data that they deal with have an independent existence. Others, (e.g. Das, Baker, Vengopal et al. 2006) go to considerable pains to detail how their data are constructed and the problems that are inherent in the concept of a TB incidence rate by ethnicity, for example. While a detailed analysis along the lines that Mol (1998; 2002a; 2002b) has so brilliantly provided for atherosclerosis would illuminate the details of the daily enactments that go into making a singular ‘tuberculosis’ and could lay bare the diverse ‘tuberculoses’ created and experienced by epidemiologists, lab scientists, health workers, patients and their families or communities, this will have to wait to future publications where we present our ethnographic study. Yet many of the complexities of TB are well known to health workers and researchers who deal with TB on a daily basis. There are at least as many TBs as there are patients, and each patient, health worker, clinician, public health doctor or public health nurse does TB differently. Nonetheless, as Mol (2002a) shows for atherosclerosis, these diverse enactments of TB are made to cohere through the processes of notifications, classifications, filing of forms and notes, epidemiological analysis, DNA studies, X-rays and such like. In contrast with those people who experience TB in person, some
politicians and many members of the public do not comprehend these complexities. Instead they call for that single and natural entity ‘TB’ to be ‘stopped at the border’.

As noted above, this reliance on what we have defined as data from beyond ethnography is our first movement towards ethnography plus. The second is towards a more public anthropology. Providing an alternative perspective, such as in this paper and related publications, so that the intended audiences of public figures like Winston Peters have a basis from which to critically analyse the xenophobia that pronouncements like those quoted above incite, is, we suggest, an example of a public anthropology. This paper is an attempt to bring these shouts that my colleagues owned to–voiced or unvoiced—from cars, living rooms, kitchens and the conference, into the public arena. We start with an introduction to the TB project as a whole and to some key features of tuberculosis before moving to illustrate both aspects of ‘ethnography plus’ through the example of so-called ‘Immigrant TB’.

THE TB RESEARCH PROJECT

Our project comprised two historical studies and five contemporary ethnographic studies. One of the historical studies was New Zealand-wide, and discussed the social history of TB between the years 1930–1970. The other focussed on TB among Maori in the Rotorua district in the interwar years. The groups or categories chosen for ethnographic study were those in which either because of group size or because of a relatively high rate of TB we were likely to find several people undergoing treatment at the time of the study or shortly before. The groups / categories identified were Maori, Pakeha, Pacific, African and Asian within the greater Auckland area (Rodney to Franklin Counties). These ethnic labels and our defining of our units of study in ethnic terms are quite problematic. In addition, although we have often resorted to the term ‘community’ as a label of convenience, this is not necessarily a sociological description. Within each category there may be no or several self-defined communities or groups. Maori participants all identified as Maori but did not belong to a single iwi or hapu or rohe (district). ‘Pakeha’ had identified as such or as ‘European New Zealander’, but at least one person in that study group identified as Maori in most other contexts. The ‘Pacific’ study included participants from several different island backgrounds within Polynesia, both island and New Zealand born people of sometimes diverse heritage, and included a small research component in Samoa, in an attempt to capture the transnational nature of many Pacific communities. The ‘African group’, was focussed on a specific national and language category, the majority of whom had come to
New Zealand as refugees. The ‘Asian’ study included participants from three specific but internally diverse ethnic groups within the category ‘Asian.’ The study as a whole was funded by the Health Research Council of New Zealand, which provided research expenses and six graduate scholarships, and the University of Auckland Research Committee. Bright Futures awarded a PhD scholarship for Lawrence and BRCSS provided a Masters scholarship for Finn. The project received ethics approval from Auckland Ethics Committee X.

The studies of Maori, Pakeha and Pacific TB, and the historical Maori case study were completed by Masters students: Alison Searle (2004, Searle, Park and Littleton 2007) from Anthropology; Moana Oh (2006) from Political Studies; Roannie Ng Shiu (2006) from Geography; and Catherine Finn (2007) from Anthropology, respectively. Three PhD students were also central to the project: Jody Lawrence from Geography (in press), on the African study; Anneka Anderson (in press) from Anthropology on the Asian research, and Debbie Dunsford (in press), from History, completing the New Zealand-wide historical study. Catherine Finn and Christina Bava both completed summer studentships, funded by the Faculty of Arts, doing bibliographical and archival research on sources for Maori and island Pacific TB respectively. The gender of our research students is a result of receiving scholarship applications from women only.

Each research student developed her own precise research questions and theoretical analysis, derived from her disciplinary background, and worked out her own specific research methods. All the community group studies, however, had a basis in ethnographic interviewing and participant observation in so far as that was possible (community participation as well as accompanying patients to clinic visits), and an emphasis on spatial relationships. Linda Bryder (History) and Robin Kearns (Geography) were co-investigators on the project. Heather Worth attempted a long-distance involvement from her Sydney base, Ron King, a GIS consultant, assisted with spatial modelling, and a network of community and public health advisors, especially Craig Thornley and Jill Miller, both from Auckland Regional Public Health Service, provided important assistance with advice and liaison.

Our guiding theoretical framework was drawn from political ecology. This approach required us to constantly tack between the many dimensions relevant to understanding TB. These include the minutiae of everyday life, including material conditions and livelihoods, social relationships and networks, peoples’ aspirations, values, understandings and experience, and their exercise of agency. Interactions between this personal level and health systems and policy,
Ecological models that do not prioritise any particular set of ‘determinants’ or any ‘level of analysis’ but rather build bridges and explore the multiple and recursive pathways between what are heuristically defined as different levels have gained ground in recent years. In 2006 the US Institute of Medicine of the National Academies published a significant report, *Genes, Behaviour, and the Social Environment: Moving Beyond the Nature/Nurture Debate*, in which they promoted an ecological model with a focus on the three major domains mentioned in the title. Quoting from an earlier report they proposed:

An ecological model assumes that health and well-being are affected by interaction among multiple determinants including biology, behavior, and the environment. Interaction unfolds over the life course of individuals, families, and communities, and evidence is emerging that societal-level factors are critical to understanding and improving the health of the public (IOM 2006:18).

They go on to point out that despite these complex interactions the vast majority of US health research resources are applied to biomedical research.

Political ecology builds from this type of framework and further theorises that ‘the environment’, sometimes assumed to be natural, is also the result of human interventions which are themselves the outcome of complex, globalising, historical, political processes (Baer 1996; Mayer 1996; Harthorn 1998; Vayda and Walters, 1999). In anthropology, the approach is used in a broad range of contexts, and it has found a particular use in medical anthropology (e.g., deWalt 1998, Hvalkof and Escobar 1998, Harper 2002). Along with the emphasis on historical political economy and ecological analysis with multiple and recursive pathways, anthropological usage usually (but not always) emphasises a critical approach to concepts of culture and society, analyses the differential agency of the actors involved, and especially the dynamic relationships, ironies,
inversions and inequalities that occur in these complex entanglements. In our project we see political ecology as a general guiding framework, something to aspire to in our accounts of what is going on, that orients us to what to look for, and prevents simplistic, premature closure.

TB AND MIGRATION

Early in our study, we came across statistics showing a large disparity in rates of TB in different ethnic categories, with ‘Other’ showing the highest rates. Pakeha have fewer than two cases per 100,000, Maori around 11 to 12, people whose families originated in the island Pacific around 35, and ‘Others’ around 75, with the figures varying slightly from year to year (Ministry of Health 2002; Sneyd and Baker 2003). Indeed, it was this and the practical necessity to consult with ‘community’ leaders and reduce the number of languages in which the research was conducted (to nine) that led us to focus on ‘ethnic/language’ groupings in designing our research, despite the potential pitfall of reinscribing ethnicity or culture as causal for TB. Of course, these tuberculosis rates are an artefact of the statistical classification process. The people who inhabit the ‘Other’ category in New Zealand ethnic statistics are particularly varied and include many people born overseas. The proportion of overseas-born notifications has been increasing and in 2005 was 76.3% of all notifications, but this varies greatly from area to area within the country (ESR 2005; Das, Baker, Venguopal et al. 2006).

It is easy to jump to the conclusion that TB is being brought into New Zealand by recent arrivals. Winston Peters did so, before he was Minister of Foreign Affairs, when he appealed to certain voters by pronouncing that our immigration and especially our refugee policy needed to be reformed to save the rest of ‘Us’ from infection or worse. TB and ‘other third world diseases’ had to be stopped at the border by stopping those people who bring them in (Berry and Watkins 2002: 2)

The problem with the ‘Winston theory’ is that it accords with neither international nor New Zealand epidemiological data. In a nutshell while in New Zealand and elsewhere a proportion of new notifications of TB disease are of people in their first year after arrival and therefore many of this group might justifiably be thought to have active tuberculosis disease when they crossed the border, the majority of notifications in overseas-born people occur after the first year of settlement, suggesting they did not have active disease when they first came (Das, Baker, Venguopal, et al. 2006). In addition the incidence among New Zealand born has been static or declining (Das, Baker and Calder
This information comes not from ethnography but from carefully collected and checked notification data which has been analysed by public health researchers and biostatisticians for many different countries, including New Zealand. Such data provides a framework for us to situate our ethnography to ask and at least partly answer: so what is going on?

WHAT IS TUBERCULOSIS?

Our next step was to understand some basic biomedical information about tuberculosis, and for this we drew on some of the many public talks and helpful explanations given by Craig Thornley, one of the key advisors for this project, as well as on the written material provided by the Public Health service and the Ministry of Health, and texts on TB, such as Reichman and Hersfield (2000). After Robert Koch discovered the tuberculosis bacillus in 1882, person-to-person transmission via the inhalation of germ-containing droplets which have been expelled by a person who has active tuberculosis disease in their lungs or respiratory tract became accepted in biomedicine as the main cause of the wide variety of health problems labelled the tuberculosis complex. This germ theory replaced – but perhaps not entirely – ideas of hereditary weakness, moral transgression or plain dirtiness as competing theories of causation.

As noted above, TB is far from being a single or simple entity. *Mycobacterium tuberculosis* and *bovis* are both part of the complex, indicating that not all transmission is human to human. Depending partly on where the bacillus is situated in the body: lungs, organs, bone, or several places at once, persons enact active TB variously and experience diverse bodily and life effects. In contrast to those who develop active tuberculosis, many people who have been infected with the bacillus experience no symptoms or effects at all, indicating that although infection is necessary to the development of tuberculosis disease, it is not a sufficient cause. The life effects of TB are intimately connected with a person’s life course and their ecological context.

A person who inhales the infectious droplets may resist infection because their immune defenses may neutralise the bacillus, or they may become infected but the infection may remain dormant for many years or forever (TBI), or they may become infected and go on to develop TB disease (TBD) themselves, often within four to 12 weeks from contact. When people develop TB in areas other than the lungs and respiratory system they cannot infect others as they do not produce bacillus-laced droplets. However, people who develop pulmonary disease are infectious to others until the bacillus is eliminated from the droplets they produce, usually by a few weeks of treatment with an array of
anti-tuberculosis drugs. Our ethnographic research discovered, however, that distinctions between infectious and non-infectious TB, or even between TB and other infectious diseases, such as HIV or leprosy, were not made in some communities.

As might be expected, given these complications, testing people for TB is complex. A positive Mantoux skin test may mean a person has got TB infection or disease, or it may mean the person has had a Bacillus Calmette Guerin (BCG) injection against TB in the past. A chest X-ray may help with screening for TB disease, but X-rays are quite difficult to interpret and do not detect infection only or non-pulmonary TB. Tests of other bodily fluids and attempts to grow cultures may be necessary to make a definitive judgement but there are difficulties with these too.

TB is only partially vaccine-preventable. The BCG vaccination is used in New Zealand only with new-borns from areas or groups with a high rate of TB infection. In some developed countries it is not used at all because of concerns about risks. BCG is now known to be a relatively ineffective form of long-term prevention. Instead, prevention of TB involves effective treatment of people with it, accurate and timely finding of contacts and treatment of those people as necessary, and removal of those circumstances that either promote the passing on of infection, such as crowded, poorly ventilated living or working conditions, or that assist the conversion of TBI to TBD.

Clearly, it was important for us to try and understand why it might be that people who arrived in New Zealand without active TB disease developed disease up to many years after arrival. In addition to infection with the bacillus, another likely contributor was stress and its relationship to immune function, part of the conceptual apparatus of biological anthropology and physiology. Many studies have shown that stress can alter immune function and hence may be detrimental to health (IOM 2006: 148). Anything that suppresses a person’s immune response may therefore assist the conversion from infection to disease (McDade 2005). Potential stressors might be difficulties of settlement in a new country where the environment is experienced as difficult and hostile (Ho 2003), co-infections such as HIV, or non-infectious conditions such as diabetes, or malnutrition.

We are fortunate in New Zealand that co-infection with HIV is low (Thomas and Ellis-Pegler 1997, 2006), as is multi-drug resistant TB (Cameron and Harrison 1997; Thomas and Ellis-Pegler 2006). Thus good prevention, timely treatment and healthy living and working conditions have the potential to reduce
the TB notification rate to a level lower than the current 10–11 per 100,000 (Sneyd and Baker 2003). Australia, for example, has approximately half the New Zealand rate (Harrison 2000: 236; WHO 2005: 235).

THE PUZZLE OF MIGRANT TB

Using the international literature and examining the logic of possible explanations for higher rates of TBD in overseas-born people we arrived at three possible explanations that could all make contributions to the rate. These are that TBD is

- not detected or reported at the time of migration
- acquired in the destination country after migration
- a result of activation of existing TBI.

We examine these possibilities in turn, summarising here the detailed research that we have carried out in the international literature and submitted for publication elsewhere (Littleton, Park, et al. n.d.).

There are two kinds of indicators of the extent of imported TBD

- post-migration health checks
- time between arrival and diagnosis.

Reviews of research using data drawn from post migration health checks in Western Australia (Pang, Harrison, et al. 2000) and San Francisco (DeRiemer, Chin, et al. 1998) suggest that TBD crossing borders makes only a small contribution to notification rates in the host country. Studies of time between arrival of migrants and diagnosis of TBD shows considerable variation depending on place of origin (Farah, Meyer, et al. 2005), however in low prevalence countries it is common for about half the cases of TBD in migrants to develop five years or more after arrival.

We did not find it easy to come to grips with the rather confusing array of New Zealand epidemiological studies that might help us frame our ethnographic studies. However, with the publication in 2005 and 2006 of several highly relevant papers, which we cite below, we were able to resolve most of our questions. The details are contained in Littleton, Park, et al. (n.d.). In New Zealand, the percentage of overseas-born people, who eventually develop TBD developing it in their first year of arrival is just over 20% (Ministry of Health 2002). The figure is 25% for those from high-incidence countries alone (Das, Baker, Vengopal, et al. 2006). Thus we can assume that at least 80% of TB disease in overseas-born people develops in New Zealand. Under its refugee...
quota agreement with the United Nations High Commission for Refugees (UNHCR), New Zealand accepts 750 refugees a year, including 10% with fragile health status. Thus refugees with TB are to be expected as part of the country’s humanitarian assistance and a screening and treatment system is in place for them. An average of 2% of refugees a year (or around 15) are diagnosed with TB and treated, while about one third are monitored or treated for possible TBI (McLeod and Reeve 2005). This humanitarian policy is likely to account for most of the minority of migrants who develop TB within two months of arrival, according to Das, Baker, Vengopal, et al. (2006:8), and who, because of this short time interval to diagnosis, almost certainly came into New Zealand with active disease. In all, screening (including refugee and migrant screening) accounts for 10-11% of annual notifications of active TB from all sources (Littleton, Park, et al. n.d.). Especially with the extension in 2005 of TB screening to all those coming to New Zealand for six months or longer (rather than two years or longer), it appears that quite effective screening is in place, for most groups. Yet despite this, overseas-born people undeniably have higher rates of TBD post settlement and most of these notifications occur after the first year of settlement, including around 30% after five years of settlement (Ministry of Health 2002, Chapter 1:10). This suggests that the disease develops after arrival in New Zealand. How does this happen?

Again, logically, there are two possible explanations:

- activation of latent infection
- newly acquired infections

To help sort out these alternatives we needed to read in the field of molecular epidemiology. Particular strains of TB have their own genetic ‘finger-print’ which can be used in a DNA lab using polymerase chain reaction techniques (PCR) to see whether the disease is being caused by strains shared with the host country or by locally unusual or unique strains which are much more likely to have been imported: another aspect of different tuberculoses. Where a person has only recently developed the disease after a considerable period of residence, DNA sequences that are unlike those in the host community indicate reactivation of an infection that was contracted elsewhere. At present molecular studies are being carried out in New Zealand in order, for example, to define outbreaks, and a database is being built up but this local work is too recent to yet tell a tale about local or imported strains. International studies suggest that most of the higher rate in overseas-born people is due to re-activation of TBI in people who in their countries of origin or of long term residence were exposed to high levels of TBD (Antunes and Waldman 2001; Chin, de Reimer, et al. 1998; Small, Hopewell, et al. 1994; Tornieporth, Ptachewich, et
al. 1997; Zuber, McKenna, et al. 1997). It is likely that similar patterns will be found in New Zealand and we await publication of the New Zealand molecular studies with interest.

These international studies also show that migrant and local tuberculoses are different epidemiologically. For example, different age groups are affected. Most international studies also show that there is little cross-transmission between migrants and locals, a pattern also reported from a recent New Zealand study (Das, Baker and Calder 2006). Where cross-transmission does occur, it is at a low rate. Many cases of cross-transmission are, counter-intuitively, from locals to the overseas-born, in other words, the reverse of the ‘Winston’ theory. This takes place where the overseas-born are exposed to the same conditions that promote TB in any population: poverty, homelessness, crowding with other infected persons, HIV infection, other illnesses and discrimination (Ho 2003; Jasmer, Ponce de Leon, et al. 1997). A study in Montreal (Kulaga, Behr, et al. 2002) interpreted clustering of TB among Haitians as activation of infection and transmission within this community which suffered social deprivation. Some groups in New Zealand appear to show similar patterns. For example, contemporary TB among Cook Islanders in New Zealand is likely to follow the Haitian pattern as there has been little TB in the Cook Islands for over a decade (pers. comm. Dr Roro Daniel, Health Manager, Ministry of Health, Cook Islands, 6/11/2002; WHO 2006:23). However, TB was common both in NZ and the Cook Islands in earlier years when older New Zealand residents of Cook Islands origin were growing up. It is therefore likely that activation of long-term TB1 and transmission within the community is producing the higher rate for Cook Islands people in New Zealand. Calder and his colleagues have demonstrated a number of times that community transmission is very ‘effective’ in Pacific communities, (e.g., Calder, Hampton, et al. 2000), a figure born out by the high rates in Pacific children in the 0-9 age group, a rate which between 2000 and 2004 was twice as high as that in the Other category (Das, Baker and Calder 2006:Table 2).

All the pointers therefore suggest that overseas-born people in New Zealand with TBD develop the disease mainly as a result of activation of pre-existing TB infection, acquired in former high-incidence living situations, and that there is also some intra-community transmission in New Zealand. This gives us a firm basis to direct our ethnographic enquiry in Auckland and our historical work in New Zealand. What is it about the political ecology of some groups in NZ that promotes conversion from TB1 to TBD and intra-group transmission of TBD?
Hence we are enquiring in our project into the ecologies of TB, including the life stories of migrants and transmigrants, their current living situations, their social networks, understandings of health and TB, interactions with the health services, and so on. We are interested in how these features interact over time with poverty, crowding and other less material noxious social conditions that all act and interact as stressors. Some people in our studies experience severe difficulties of acceptance within their own communities because of TB. For example, one young man was forced out of his flat when his flatmates learned he had TB, and an older woman explained to us how bad it was for a woman in her community to have TB because she could lose her children and her husband. These events occurred despite the individual no longer being infectious according to biomedical reasoning. The opprobrium associated with TB outlasted the infectious stage. Others conducted their lives with TB to avoid this anticipated stigmatisation, by covering their illness with a veil of secrecy. One student had not even told his parents back home, while others told friends that they had various sorts of other non-stigmatised illnesses. A ripple of fear was observed among both the ‘Asian’ and ‘African’ participants when deportations of visitors or students with TB received publicity. Stigma and secrecy has implications for diagnosis, treatment, recovery and infection of others, as well as for access to social support. A further toxic socio-political stressor experienced by particular groups is their continually being singled out as potential threats to ‘the New Zealand’ way of life and health: the ‘Winston Peters’ effect. This kind of marginalisation and inducement of fear, by adding extra stressors and increasing social isolation, is more likely to increase the rate of conversion of TBI to TBD, and to increase transmission rates, rather than to prevent the importation of TBD into New Zealand.

Conclusions

Better conditions here for the overseas-born have the potential to mitigate TBI and prevent the transmission and activation of TBD. We cannot make this argument on the basis of ethnography alone, we need history, epidemiology, molecular analysis and immunology. But on the basis of this research framed in terms of political ecology, we can speak to public questions about immigration and do ‘ethnography plus’.

On the positive side, our research has the possibility of creating greater understanding of the life circumstances of migrants, thus reducing the distance between them and the other New Zealanders who might, though ignorance and a lack of imagination, be influenced by crude appeals which forment fear and call up chauvinistic responses. It can be used to support policy and practices
that increase the well-being and social support of migrants, as well as their access to health care. On the negative side we can suggest that every threat against migrants to win political support from voters or for any other reason has the potential to promote TBD in the overseas-born, on the basis that the cumulative effect of always being cited as a social threat is a high level of sustained stress, and hence lowered immunity. Perhaps the promotion of Winston Peters to Foreign Minister has brought a period of calm for these vulnerable groups who for the most part, and certainly as much as those born in New Zealand, strive to become good citizens of our joint diverse nation.

NOTES


2. In his introduction to the second edition of *The Professional Stranger*, Michael Agar (1996: 26–29) discusses the sociological tradition of ‘critical ethnography’, with its strengths in situating ethnography in political economy and its attendant problems and conundrums such as the necessity to identify false consciousness.

3. See, for example, the American Anthropological Association’s ‘RACE Project’ (Overbey and Moses 2006), and the continuing interest in the pages of *Anthropology Today* from the Royal Anthropological Institute in Britain, in engaging in key questions of the day, following in the footsteps of anthropological ancestors, such as Margaret Mead and Edmund Leach.

4. We are indebted to an anonymous reviewer for referring us to Mol.

5. Recently published age standardised rates show that compared with Europeans, Maori, Pacific, and Others have rates 10.5, 22.3 and 36.5 times greater, respectively (Das, Baker and Calder 2006:1).

6. In 2000 the proportion of overseas-born cases was reported as 55.6% while in 2005 it was 76.3% (Littleton, Park, *et al.* n.d.). Between 2000 and 2004 the average proportion of overseas-born cases was calculated as 64.6% (Das, Baker, Vengopal, *et al.* 2006:1). Not surprisingly, cases are mainly from countries with a high incidence of tuberculosis disease.

7. However there is some confusion in the Ministry of Health documents. For example, in the *Guidelines for Tuberculosis Control in New Zealand 2003* (2002),
in Chapter 1, Figure 1.5, p.10 shows about 25% of notifications for overseas-born people were of people who had arrived within the last year, whereas in Chapter 7, p.5, it is claimed that the figure is 60%. The other data reviewed here, particularly Das, Baker, Vengopal, et al. (2006) suggest that this 60% refers to ‘within five years of arrival’.

8 Short term visitors may occasionally arrive in New Zealand with active TB. The screening and health services for some of the ‘family reunification’ category of migrants (i.e., those who to all intents and purposes are refugees but do not have a six week stay at Mangere reception centre), and for asylum seekers are not as comprehensive as those for UNHCR quota refugees.

REFERENCES


Littleton, J., J. Park, C. Thornley, A. Anderson, J. Lawrence n.d. ‘Do immigrants or does immigration give you tuberculosis’ (ms undergoing revisions, submitted to ANZJPH).


