

CHAPTER 8

Syndemics in Global Health

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INTRODUCTION

In 1997, the Institute of Medicine (IOM) helped advance the growing recognition of health as an intertwined global phenomenon by noting that:

The health needs of diverse countries are converging as the factors that affect health increasingly transcend national borders. Among those factors are the globalization of the economy, demographic change, and the rapidly rising costs of health care in all countries. In a world where nations and economies are increasingly interdependent, ill health in any population affects all peoples, rich and poor.

Ten years later, the IOM (2008) reported that an eager new generation of philanthropists, students, scientists, private industry leaders, and citizens offer the promise of potential solutions to global health. The health social sciences, including medical anthropology, thus face the challenge of identifying specific ways it can contribute to the emerging global health agenda. An important step in this process is developing concepts “that may serve as useful lenses to bring issues in need of investigation into better focus” (Nichter 2008:157). One of the concepts Nichter recommends for this purpose is syndemics, which has been defined as 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 2009b). Syndemics is a useful concept for global health initiatives because of its sensitivity to “environments of risk and agents promoting risk, not just groups at risk and risky behaviors” (Nichter (2008:157) and its ability to focus attention on three fundamental

A Companion to Medical Anthropology, First Edition. Edited by Merrill Singer and Pamela I. Erickson.

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intersections in the making of health: synergistic disease interactions that increase the burden of disease beyond mere comorbidity, interspecies interactions that lead to emergent diseases, and health and society interactions that support the clustering of multiple diseases in vulnerable populations (Singer and Clair 2003). Syndemics thus offers a pathway for transcending shortcomings of existing health interventions and prevention initiatives in disparity populations (Paluzzi and Farmer 2005). In this chapter we examine existing and emerging applications of the syndemics model in global health research, past and present.

OVERVIEW OF SYNDEMICS

The syndemics approach to understanding disease is rooted in medical anthropology, especially the theoretical framework known as critical medical anthropology. The idea emerged as a response to early research in this field which tended to be narrowly focused on micro-level explanations of health related beliefs and practices and their interface with local ecological conditions, wider cultural configurations, or human psychological factors. The subsequent generation of medical anthropologists, influenced by social movements and broader cultural realignments in the 1960s/1970s, argued that the prevailing theoretical models tended to ignore crosscutting political economic influences on health and on human decision-making and action in the health arena.

The alternative understanding created in response to these limitations – critical medical anthropology – drew attention to the vertical linkages that connect a social group or behavior (or health configuration) to larger political and economic systems and to the arrangement of social relationships they help produce and reproduce over time (Singer 1995). The goal of critical medical anthropology has been not to dismiss the contributions of microanalyses of illness and healing or ecologically influenced accounts of health, but rather: to emphasize the relevance of culture to issues of power, control, and resistance associated with health, illness, and healing, and to rethink nature itself in light of historic anthropogenic (intended and unintended) restructurings of the environment. Further, the critical approach stressed that while human groups engage and are impacted by the environment, they do not simply adapt to it; rather, in thought and in deed they remake it, although never with unlimited ability nor without unintended and unforeseen consequences.

The syndemic orientation was forged as part of this rethinking, and in no small measure in direct response to the global challenges to health and society of the emergent HIV/AIDS pandemic. Specifically, this way of understanding disease recognizes the fundamental biosocial nature of health and that constellations of diseases and other health conditions interact synergistically, in consequential ways. This interaction involves multiple biological and psychosocial channels and mechanisms, including biochemical changes in the immune system, damage to cellular repair processes, and psychopathological effects on behavior. Further, the syndemics orientation emphasizes the ways that social conditions shape disease processes, often through broader environmental mediation. Consequently, the syndemics approach examines both the emergence and nature of “disease concentrations” (i.e., multiple coterminous diseases and disorders affecting individuals and groups) and “disease interactions” (i.e., the

ways in which the presence of one disease or disorder enhances the health consequences of other diseases and disorders). More particularly, this perspective aims to identify specific pathways through which diseases and other health conditions work together inside individual bodies and within populations and significantly multiply thereby the overall health burden of the afflicted. Human social environments, including prevailing structures of social relationship, such as institutionalized inequality and injustice, as well as socio-genic environmental conditions contribute enormously to disease clustering, disease spread and interaction, and the interlocking of diseases and other health conditions into health-threatening syndemic complexes.

SYNDEMIC RESEARCH

Syndemic theory extends the work of health researchers who long ago recognized the crucial importance of disease interaction in social context. In an important series of publications on health in New York City, for example, Rodrick and Deborah Wallace (Wallace 1988, 1990; Wallace and Wallace 1998) drew attention to the “synergism of plagues” produced by public policies deliberately designed to restrict municipal services in low income neighborhoods so as to pressure residents to move away and free up land for economic development. The tragic result was a mass movement of people to new (also poor) areas, overcrowding, and an unraveling of community relationships and social support structures, as well as a set of closely linked epidemics, including tuberculosis, measles, substance abuse, AIDS, low-weight births, and street violence. Separating these health issues – and thereby overlooking the ways in which they are intimately connected and mutually enhancing, and ignoring the underlying social and biological processes involved in their development and clustering – distorts “on-the-ground” and “in-the-body” realities and fails to address the nature of the relationship between the health of physical and social bodies.

The term syndemic was first used to label a tripartite health condition of the inner city poor called SAVA, a product of the complex interactions between substance abuse, violence and AIDS (Singer 1996). From the syndemic perspective, AIDS, drug use, and violence in particular social contexts are so entwined, and each is so significantly shaped by the presence of the other two, that it is wrong to conceive of them as distinct “things in the world.” In SAVA, all three disease/health related components interact. Thus there are ways in which drug use interacts directly with AIDS (e.g., in promoting HIV transmission, disease progression, AIDS-related mortality, and the development of AIDS-defining illnesses), and both conditions are worsened as a result (Cook et al. 2008). Similarly, violence and AIDS interact in mutually accelerating ways, such as when the risk of clinical progression of HIV is enhanced by domestic violence that prevents access to health care or the ability to adhere to HIV treatment (e.g., Lichtenstein 2006). Drugs and violence also help to propel each other along their injurious paths (Duke et al. 2006). In addition to these interactions, the actual expression of a SAVA syndemic is shaped by the local social context, including both the population being affected and the configuration of social conditions they endure (Singer 2006). It is thus more accurate to recognize the existence of multiple SAVA syndemics, each driven by its own arrangement of populations, social conditions, and structural relationships (Gonzalez-Guardia 2008).

Stall and co-workers (2003, 2008) have examined SAVA in populations of men who have sex with men (MSM) using a household telephone survey of almost 3,000 men in New York City, Chicago, Los Angeles, and San Francisco. They found that the SAVA syndemic among MSM is rooted in childhood sexual abuse which contributes to the development of depression in adulthood, and subsequent entrance into abusive adult relationships, use of multiple drugs, and high levels of HIV risk and infection. Further, being the victim of homophobic attacks contributes to serious health problems among adult gay men. These psychosocial factors interact and are mutually reinforcing.

Building on the work of Stall and co-workers, Mustanski and colleagues (2007) studied an ethnically diverse community-based sample of 310 young (16–24 year old), self-identified MSM in Chicago. Their analysis of various psychosocial, behavioral and health variables (e.g., regular binge drinking, experiencing partner violence, sexual assault, psychological distress, sexual risk taking and HIV status) led them to conclude: “that the number of psychosocial health problems additively increase risk for HIV among urban YMSM. For example, each problem increased the odds of an HIV positive status by 42% and also increased the odds of sexual health risk behaviors. Multivariate analyses indicate that substance use and being the victim of violence show the strongest relationship to sexual health and HIV risk” (Mustanski et al. 2007:44). This syndemic of psychosocial risk factors underscores the multiple health disparities, higher morbidity, and barriers to care faced by gay and lesbian individuals (Mustanski et al. (2007:39).

Building on the Wallaces’ work in New York, Freudenberg and co-workers (2006) point out that public policy decisions in New York City in the mid-1970s were driven by a cost-cutting mission intended, ostensibly, to save money for the city. Instead, these decisions contributed to deteriorating living conditions, a 20% rise in the number of poor people in the city (despite an overall population decrease of 10%), and a significant rise in the health burden of the poor. Tuberculosis rates, for example, rose despite a previous century of declining infection. At the same time, AIDS cases became more frequent, particularly among drug users. Homicide rates accelerated and climbed continuously through 1990. In short, neither was public health prioritized as a social good nor were the significant interactions that occur between health and social issues a basis for making public policy. The fallout from cost-cutting was enormous, fiscally and in terms of socially structured suffering, and sparked a costly tripartite syndemic of HIV, TB, and violence.

An explicit syndemic perspective informs several other studies, such as tobacco-related syndemics (Marshall 2005; Nichter 2008), a sexually transmitted diseases and HIV/AIDS syndemic in disparity populations (Singer et al. 2006), the role of syndemics in the health of inner city populations (Mercado et al. 2007; Singer 1994), syndemic interaction among diabetes, obesity, and periodontal disease (Hein and Small 2007), the HIV and food insecurity syndemic in Southern Africa (Singer 2008), the role of tuberculosis in syndemics (Mavridis 2008), infectious disease syndemics in Canadian Aboriginal populations (Herring and Sattenspiel 2007; Young and Herring 2004), and syndemics and the diseases of global warming (Baer and Singer 2003; Singer 2009a). These and other studies prompted the Centers for Disease Control and Prevention to incorporate the concept into public health prevention initiatives.

An even larger literature examines the negative health consequences of comorbid disease interactions without an explicit syndemics perspective (e.g., Alisjahbana et al. 2006; Abu-Raddad et al. 2006; Cain et al. 2007; Ponce-De-Leon et al. 2004; Sethi 2002). Exemplary research by Tashiro et al. (1987) shows that staphylococci bacteria secrete enzymes that activate the infectivity of influenza viruses in animal models. Virus–bacteria interaction has been reported to be a major cause of severe influenza pneumonia in humans that works in both directions, with bacteria enhancing influenza virulence and the influenza virus promoting the adherence of bacteria to host cells. Under social conditions that promote the transmission and clustering of these two pathogens, deadly global syndemics can ensue, as occurred in 1918 (Singer 2009b).

The term *syndemic*, however, is not synonymous with comorbid or equivalent medical terms (e.g., co-infection, dual-diagnosis). Patients in Hong Kong dually infected with SARS and human metapneumovirus (HMPV) were found to be no sicker than patients only infected with SARS (Lee et al. 2007). This suggests that comorbid SARS and HMPV either do not interact or that interaction does not lead to excess disease burden. SARS interacts with other diseases, however, such as diabetes or cardiopulmonary disease, resulting in poorer outcomes (Chen et al. 2005).

To date, the syndemics literature has stressed human/environment and human/human relationships. More recently, a focus on human/animal relationships in syndemic formation has begun to emerge.

ANIMAL–HUMAN CONNECTIONS IN SYNDEMICS

Even though anthropologists argue that people’s treatment of and ways of talking about animals represent cultural mirrors (Mullin 1999), medical anthropologists have tended to neglect the importance of animal health and ethnoveterinary practices to human health (but see McCorkle et al. 1996; Nyamanga et al. 2008; Rock and Babinec 2008; Barta 2008; Engel and Engel 2008). The history of anthropological work on animal–human connections, for instance, often refers to Evans-Pritchard’s (1960) research with Nuer pastoralists (e.g., Shanklin 1985), but not to *Witchcraft, Oracles and Magic among the Azande* (Evans-Pritchard 1976 [1937]). The Azande study remains “a monument in the history of the anthropology of medicine” (Cambrosio et al. 2000:6), but scant attention has been paid to animal–human connections in Zande beliefs and practical responses to disease specifically, or in health and disease generally.

When teaching this classic study, anthropologists often tell their students that colonial authorities dispatched Evans-Pritchard to investigate the local – seemingly irrational – responses to *trypanosomiasis*, also known as sleeping sickness. These responses included witchcraft accusations and a refusal to move to places where the tsetse flies that spread the infection were sparse. Yet, we suspect, very few anthropologists emphasize that sleeping sickness is a zoonotic disease that afflicts, for example, cattle and donkeys, as well as people. While “the oracles provide[d] an endless supply of ‘witches,’ thus perpetuating the Zande inclination to consult witch-catching oracles when someone falls sick or suffers misfortune” (Cambrosio et al. 2000:6), contact between infected livestock, wildlife, insect, and people ensured a continuous supply of human cases of sleeping sickness who were identified as victims of witchcraft. Thus attention was

directed away from flies, animals, colonial authorities, and local hierarchies, and towards specific individuals. The result was a generalized state of high anxiety in which most sicknesses and deaths were viewed as instances of malevolence (Evans-Pritchard 1976 [1937]:5). *Trypanosomiasis*, moreover, involves sleep disturbances and “various psychiatric and mental symptoms such as anxiety, lassitude and indifference, agitation, irritability, mania, sexual hyperactivity, suicidal tendencies, and hallucinations” (Kennedy 2008:118). Underlying and helping to animate this “synergistic brew” between animal health and human health was inequality within and between human populations, between colonial and local populations, and within the Zande population.

In syndemic fashion, inequality remains fundamental to *trypanosomiasis* morbidity and mortality, for *trypanosomiasis* “continues to pose a major threat to 60 million people in 36 countries in sub-Saharan Africa” (Kennedy 2008:118), where HIV is rife and can interact deleteriously with it (Lloyd-Smith et al. 2008). In addition, people living with both HIV and *trypanosomiasis* may be at elevated risk for drug treatment failure (Pepin et al. 1992). As Kennedy (2008:118) observes, “All four main drugs used for human African trypanosomiasis are toxic, and melarsoprol, the only drug that is effective for both types of central nervous system disease, is so toxic that it kills 5% of patients who receive it,” but Western governments, donor agencies and the pharmaceutical industry have shown little interest in developing new drug therapies.

If disrupting “man/tsetse fly contact” is essential for controlling sleeping sickness (Kennedy 2008:124–125), then treating cattle and other infected animals may prove to be a cost-effective and ethical avenue for improving human health (Zinsstag et al. 2007). Animal husbandry practices can help prevent and control *trypanosomiasis*, reducing the likelihood of human cases and improving the productivity of draught animals used in agriculture and for transportation (Mattioli et al. 1994).

Even though medical anthropologists have paid insufficient attention to zoonotic diseases, recognition of the importance of human/animal relationships is growing because many public health problems have their ultimate origins in animal bodies. An estimated 60% of all emerging and re-emerging infectious diseases are zoonotic (Jones et al. 2008), and the socio-economic conditions that drive the distribution of diseases in human populations spill over to animal populations (Jones et al. 2008; Rock et al., 2009). For example, epidemics of tuberculosis in industrializing cities of the 19th century involved both *Mycobacterium tuberculosis* and *Mycobacterium bovis* (Jones 2004). *M. bovis* is often called bovine tuberculosis but can infect nearly all mammals, including people. While infected animals or people can transmit *M. bovis* to each other, people mainly contract it by consuming infected animal-sourced foods, particularly unpasteurized milk and raw or undercooked meat (Ayele et al. 2004).

Worldwide, at least 3% of all pulmonary cases and 9% of all non-pulmonary cases of tuberculosis in people involve *M. bovis* (Ayele et al. 2004). While this pathogen disproportionately affects low-income countries, it also disproportionately affects low-income inhabitants of wealthier countries. In San Diego, for example, researchers found that 1/24 adult human patients with TB-related respiratory disease and 11/24 with non-respiratory disease related to TB were infected with *M. bovis*, and that the infected population was 80% Hispanic (Dankner et al. 1993). Overall, 45% of pediatric human tuberculosis cases in San Diego between 1994 and 2005 involved *M. bovis*, 6% of the adult cases involved *M. bovis*, and 25% of them were also living with HIV (Rodwell et al. 2008:913). Compared with those with *M. tuberculosis*, people with

M. bovis were more than two and half times more likely to die in spite of treatment (Rodwell et al. 2008).

M. bovis is thought to be less virulent for humans than *M. tuberculosis*. In combination with HIV infection, however, *M. bovis* could pose a serious syndemic threat. If the differential virulence between the two pathogens stems from greater susceptibility of human host defenses to *M. bovis*, immunosuppression induced by HIV co-infection could enhance the contagiousness of *M. bovis* (Grange et al. 1994:1565). Indeed, European studies have confirmed human-to-human transmission of *M. bovis* among hospitalized patients with HIV (Guerrero et al. 1997; Samper et al. 1997). Human cases of *M. bovis*, furthermore, cannot be treated with standard drug therapy because *M. bovis* is almost universally resistant to pyrazinamide (PZA), the main antituberculosis drug (Rodwell et al. 2008:910). A strain of *M. bovis* that claimed the lives of at least 20 HIV+ patients in European hospitals has proved resistant not only to PZA, but to 11 different antituberculosis drugs (Guerrero et al. 1997; Samper et al. 1997).

People's reactions to policies put in place to control animal diseases can affect their health, and these perceptions are influenced by power differentials. Canada, for example, is one of many countries to adopt a "test-and-slaughter policy" for *M. bovis* in cattle and other animals that serve as human food, yet wildlife populations are exempt from this policy. Elk infected with *M. bovis* have been sources of infection for cattle that graze near a national park, within which hunting is prohibited (Brook and McLachlan 2006). Cattle farmers living near the park risk losing their entire herd should one of their animals test positive for *M. bovis*. While compensation is offered, farmers understandably fear bureaucratic delays, becoming embroiled in negotiations about compensation, stigma, and lower prices for their cattle or land. Successful measures to control animal-to-human disease transmission, in other words, can lead to mental health concerns such as depression in susceptible individuals and to mistrust and feelings of powerlessness across entire communities. Mistrust and powerlessness are not diseases, but human beings are sensitive to power relations and to the ability to exercise control over their lives such that mortality rates vary along these lines (Nguyen and Peschard 2004).

While animal-sourced foods can transmit infections to humans, they also have beneficial effects by preventing and controlling human diseases through nutritional pathways. The quality of nutrition influences the life expectancy of people with infectious diseases like *M. bovis*, *M. tuberculosis*, HIV/AIDS, or some combination of these. Veterinary expertise has helped reduce the effects of infectious diseases in human populations (Hardy 2002; Jones 2004), not least by improving food safety, yet animal health is largely absent from accounts of public health's history. Animals infected with *M. bovis* and other zoonotic diseases, moreover, are less productive, which affects people's livelihoods and food supply.

Disease interactions, zoonotic or otherwise, are not always deleterious in the sense of a syndemic; they may have protective effects. Such phenomena are referred to as counter-syndemics (Singer 2009a,b). *Trypanosome cruzi* (the protozoa responsible for Chagas disease, the most endemic zoonosis in Latin America), for example, inhibits HIV replication in human placenta, and so might reduce the likelihood of mother-to-child transmission of HIV (Dolcini et al. 2008). The Chagas case also illustrates how a single infection can have both counter-syndemic and syndemic-promoting effects. The co-occurrence of Chagas disease and rheumatic heart disease (caused by

Streptococcus pyogenes infection) among poor, rural children in Latin America enhances the chances that they will die in early adulthood from congestive heart failure because of the lack of access to health care and treatment of their childhood infections (Cubillos-Garzon et al. 2004).

SYNDEMICS IN HISTORY: CASE STUDY I

Thus far we have underscored the utility of the syndemics concept for identifying pathways through which biological and social phenomena interact and capacitate each other in contemporary settings. The concept also offers a powerful framework for interpreting health in historical contexts. We illustrate this through a consideration of the circumstances that led to entwined epidemics and deteriorating life circumstances in northern Aboriginal communities in Canada. Apart from its ability to illuminate the complex processes through which the disease burden of communities increases through time, application of the syndemics concept allows scholars to speak to larger historical issues, such as the role of introduced pathogens in the demographic history of the Americas.

It is becoming increasingly clear from research on Aboriginal communities in Canada that epidemics of the early 20th century were not simply the result of exposure to new pathogens, but rather the expression of the long-term operation of a multifaceted set of interacting social and biological processes more appropriately interpreted as a syndemic (Herring and Sattenspiel 2007; Stephens 2008). These include: ecological changes associated with the importation of new plant and animal species (including pathogens) imposition of social, political, economic and religious structures by missionaries, traders, and government officials; ever more numerous settlers farming the lands of Aboriginal people; increasing isolation of Aboriginal people from centers of economic and political power; assimilationist policies; and social fragmentation that left Aboriginal groups on the margins of Canadian life (Waldram et al. 2006). In addition, the growth of endemic urban foci for density-dependent diseases, such as measles, and improvements in transportation efficiency allowed a wider spectrum of infectious diseases to travel over long distances (Hackett 1991, 2002), enmeshing northern Aboriginal communities in global disease networks through the confluence of disease pools (McNeill 1976:69 ff.).

At the same time, centuries of hunting and trapping fur for export had depleted animal resources in the Canadian north, eroding the economic foundation of most northern Aboriginal communities. In response, international businesses, such as the Hudson's Bay Company, closed fur trade posts, the hubs of the northern fur economy. Growing intrusions of the Canadian Government, including the system of native reserves, and progressively larger numbers of non-native participants in the northern economy, further undermined social and economic life in the north. Added to the demise of the fur economy, crowding of Aboriginal peoples into reserve housing, the growing concentration of children in government-funded residential schools, and the change from "a portable home within an ecological range, to housing in sedentary settlements" (Preston 1986:245), left many communities impoverished and ever more vulnerable to malnutrition and to diseases spread from elsewhere (Herring et al. 2003; Honigmann 1948; Tisdall and Robertson 1948; Waldram et al. 2006).

During the epidemiologic transition that accompanied this social and demographic transformation (Young 1988), tuberculosis emerged as a major health problem. A study of children in residential schools in the early 20th century, for instance, concluded that TB was the leading cause of death (Bryce 1907). By the 1930s, TB death rates among Aboriginal people in the western provinces of Canada were ten to twenty times higher than the rate for non-Aboriginal people (Stewart 1936). TB also increased susceptibility to other infections (Stone 1926). The relocation of Aboriginal people to reserves with minimal economic resources, and over-crowded houses and schools, allowed tubercular infection to spread rapidly, especially among children (Bryce 1907; Stoops 2008; Waldram et al. 2006). TB soon became a central element in the syndemic conditions that developed on many northern reserves.

The circumstances at the site known as the York Factory, a Cree community located on the western edge of Hudson Bay, illustrates how syndemic processes operated through endemic TB and two acute respiratory epidemics in 1927 (ACCA 1864–1929). From February to April, the York Factory was overwhelmed by a severe epidemic of influenza. The influenza death rate was highest among adults between the ages of 21 and 65, who accounted for 87% of the deaths (120 deaths per 1000). By mid-February, life in the community had ground to a halt; by March, coffin building was a major task. Not only was everyone sick, but the fur trade that year was a disaster. The tragic loss of such a large group of productive adults, which occurred at a vital point in the fur harvest cycle, led to an unusually poor harvest (HBCA 1794–1939), adding additional hardship by cutting the already wobbly economic legs out from under the community that year (Young and Herring 2004).

The following autumn, another virulent epidemic struck. Whooping cough (*Bordetella pertussis*), a highly contagious bacterial disease acquired by droplet infection, swept through the York Factory. The bacteria attach themselves to the cilia in the trachea where they produce exotoxins that kill the ciliated cells and induce inflammation and mucous production that compromise the lungs. The worst effects of whooping cough are felt by children under the age of six, especially among infants and two-year olds (Cherry 1999). Acquired immunity fades with age, however, making adults an important source of this strictly human disease.

The York Factory's autumn whooping cough epidemic erupted in September during another crucial time in the annual cycle when families were preparing for the fall fishery. The epidemic lasted until December (HBCA 1794–1939), taking a heavy toll among children under the age of six (237 deaths per 1000), the group normally affected (Young and Herring 2004; Herring 2008). Two teenage girls also died, indicative of the severity of the outbreak. While children were dying from whooping cough, adults were suffering from what was described as "very bad colds" (HBCA 1794–1939) and more than likely were the source of the whooping cough outbreak (Young and Herring 2004).

In 1927, therefore, we see three respiratory diseases, one endemic (tuberculosis) and two epidemic (influenza and whooping cough), acting together and sequentially to amplify each others' effects on the lungs of the people of the York Factory. The mortality profile that year is effectively described by these three causes: 32% of deaths are attributable to the spring influenza outbreak, 40% to the fall whooping cough outbreak, and another 7% to tuberculosis (Young and Herring 2004). As discussed earlier in this chapter, active tuberculosis can exacerbate influenza infection. Influenza,

in turn, enhances bacterial lung disease, impairs normal recovery mechanisms, and impairs the immune system. It can also induce latent TB to erupt. Whooping cough affects the respiratory tract, destroys the respiratory lining, and makes it necessary to cough to remove mucous. This can worsen existing lung disease in the form of tuberculosis. Adults already suffering with TB at the York Factory, in turn, may have been more likely to experience a bout of whooping cough.

Together, these diseases made it more difficult to make a living off the land under the already difficult circumstances of increased competition and resource depletion. Ultimately, the economy was not sustainable. By the 1950s, the York Factory had essentially been abandoned as residents progressively moved out of the area to find a better living in more prosperous southern locations. In short, a series of interacting epidemics were both enhanced by and contributed to the erosion of the local economy and were part of entwined historical, biological and economic processes. Operating synergistically over the long-term, these processes contributed to the eventual abandonment of the York Factory and led to its current status as a heritage site.

The story doesn't end there. Today TB rates in Canada are low (5.0/100,000) (Public Health Agency of Canada 2007) on the global scale and in comparison to the high rates characteristic of the early 20th century. Yet there are residual pockets where the disease flourishes and particular groups in which it continues to take a higher toll, notably among migrants from TB endemic areas and Canadian-born people of Aboriginal descent who, together, account for the largest proportion of contemporary TB cases. The clustering of cases among Aboriginal people becomes even clearer when foreign-born cases are eliminated and only native-born cases considered. In 2005, for example, the TB rate for the Aboriginal population was almost 30 times higher (26.8/100,000) than that for the non-Aboriginal population (0.9/100,000). Aboriginal communities at highest risk for TB remain those in remote locations, with poor housing and crowding, lacking adequate health care, or in the process of implementing new health transfer agreements (Health Canada 1999). In other words, the conditions that underlay the operation of syndemics in the 20th century persist into the 21st, yet the vast majority of studies fail to address the social and economic determinants of this disease's tenacity in Aboriginal populations (Farmer and Jacklin 2008), let alone its interaction with other conditions.

The second case study shifts focus from Canada to the Pacific Islands, but also addresses the role of TB in indigenous and migrant populations.

A CONTEMPORARY SYNDEMIC: CASE STUDY 2

In the study of health problems it at times becomes clear that the health condition being analyzed could be replaced by others and the same patterns and processes would be found (Farmer 1999). Alternatively, synergistic interaction may multiply misery, a process highlighted by the syndemic perspective. A shift in understanding from the former to the latter perspective is illustrated by a recent research project on tuberculosis (TB) in New Zealand.

Although a significant public health issue globally, TB in New Zealand has a relatively low national prevalence of 10 per 100,000 per annum, making it a minor problem. As is the case for Canada, closer examination reveals major disparities by

ethnicity, geography, class and history (Das et al. 2006). Interdisciplinary research on tuberculosis in Auckland, New Zealand, using a political ecology framework, elucidated highly variable patterns and experience of TB infection among six different groups: Maori, Pakeha (New Zealanders of European origin), Pacific Islanders, an African refugee group, and Asian immigrants (Park and Littleton 2007). Despite living in the same urban location and utilizing the same health care system, different ethnic groups experienced very different patterns of TB transmission and disease and had very different treatment experiences, termed “local ecologies” (Littleton and King 2008). Beyond TB, the study shows how analysis of an infectious disease can illuminate the experience of social and economic inequality. A diagnosis of TB places patients and their families into prolonged contact with administrative structures, creates social tensions, and highlights the importance of social support or its lack (Anderson 2007). Notably, another disease, Meningococcal B viral infection, shows a similar geographic and economic distribution (Crump et al. 2001). In other words, the study of either of these diseases would have yielded similar findings.

In the case of the Pacific Island group, however, a series of further puzzles materialized. In contrast to the other communities in the study, TB among this group has increased especially among the young (Das et al. 2006). Furthermore there is active transmission from adults to children and from local to foreign born individuals and vice versa, creating a significant future burden of ill health (Das et al. 2006; Littleton et al. 2008; Howie et al. 2005). In the other immigrant communities, TB is most commonly due to reactivation of preexisting infection, often acquired in the home country (Verver and Veen 2006) and in these groups transmission between foreign and locally born populations is small to non-existent (Littleton et al. 2008).

Mexicans living along the Mexican–USA border have a similar pattern of transmission to Pacific Islanders living in Auckland (Restrepo et al. 2007; Littleton et al. 2008). Other similarities occur: both populations are transnational with frequent border crossings, both have marginal economic positions and, at times, equivocal migration status, and, significantly, Mexicans, like Pacific Islanders, experience disproportionately high rates of TB and diabetes mellitus (DM) (Restrepo et al. 2007).

Epidemiologically TB and DM operate synergistically. Along the Texas border, it is estimated that the relative risk of TB disease in a Hispanic person with DM is two times the risk of a person without DM (Restrepo et al. 2007). In addition, the presence of DM changes the traditional epidemiological profile of a TB patient in this population from a young man with a history of incarceration to an older woman introducing a new subset of people to TB disease and opening up new transmission links.

Clinical and experimental studies demonstrate that the syndemic interaction between TB and DM operates in multiple ways (see Stevenson et al. 2007). Preexisting diabetes (DM) serves to increase the risk of TB disease through multiple effects on the immune system including increased bacterial loads among DM patients, lowered production of interferon- γ in diabetics (Martens et al. 2007), and depressed activation of alveolar macrophages (Wang et al. 1999). DM and TB also interact to make treatment of both more difficult, prolonged and with higher failure rates. In an Indonesian study by Alisjahbana et al. (2006), diabetic patients treated with rifampicin have lower plasma levels of the drug than non-diabetics on equivalent treatment. In reverse, active TB disease is associated with difficult glucose control among diabetics (Stevenson et al. 2007). It has also been hypothesized that TB predisposes people to DM through

amyloidosis of the pancreatic islet cells which impairs insulin production (Broxmeyer 2005). Finally, there is some evidence of mutual causation via routes such as disorders in Vitamin D metabolism (Chan 2000; Flores 2005; Ustianowski et al. 2005).

Given the evidence for a TB/DM syndemic affecting the Mexican population in the area of the Texas border the question arose: could Pacific Islanders also be affected by a similar syndemic? Stall et al. (2003) identified a need to discover populations that might be at similar risk of syndemic conditions and that is a starting point for planned future research.

At the same time, diseases do not necessarily operate in the same ways since they are produced by local biological and social conditions interacting within a global political economic context. The initial study on TB in Auckland identified the importance of history at the international, national, community, and generation levels (Dunsford 2008). For example, international relations, particularly as they translated into migration policies, had and continue to have a significant impact on requirements for TB testing of immigrants, particularly from different Pacific Island nations and the access to health care of different communities in Auckland (non-permanent residents do not have access to free treatment) (Ng Shui 2006). At the national level, some island nations (e.g., the Cook Islands) achieved very effective control over TB during the 1960s and 1970s thereby reducing the pool of currently infected people. At the community and generation levels, experiences of stigma affect the willingness of people to come forward for contact tracing and treatment (Ng Shui et al. 2008). These complexities, visible in the first study, will almost certainly be salient in the next. Indeed, the Ministry of Health ethnic grouping "Pacific peoples" hides diverse experiences. Some Pacific groups in New Zealand experience similar rates to their home countries, others have higher recorded rates in New Zealand than at home although such rates need to be treated cautiously given the small numbers involved (e.g. the rate of TB in Tuvalu in 2005 was 305 versus 629.3 among Tuvaluan born in New Zealand from 2000 to 2004; the comparable figures for the Cook Islands are 16 versus 34.2 (Das et al. 2006; World Health Organization 2007)). For this reason the planned research will work with two communities both in New Zealand and at home to capture and explain this diversity.

One of the barriers to health improvement in populations is the failure to examine linked phenomena. As Bartlett writes: "experts in TB and experts in HIV infection live in different worlds ... 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).

In relation to TB and DM in New Zealand this division occurs bureaucratically at the national and local level, while relationships between health and immigration status offer a further difficulty in coordinating effective policy. A syndemic approach to TB is envisaged as a way of assessing and developing inter-sectoral approaches to health while recognizing the transnational lives of research participants and the way risks are concentrated and "buffered" over time.

TB is a historical residue of previous exposure occurring within a new disease environment. Working with Pacific people requires a transnational perspective to explore the continual movement of people, capital, goods, and information across national borders. Such movement is a significant challenge to traditional approaches to health research and policy (Vertovec 1999; Messias 2002). Developing effective strategies to

reduce health disparities among transnational populations will require an understanding of: the relationships between migration status and health, life history (inter- and intragenerational), multiple health services, and political economic conditions in the sender and receiver communities. Attention to such factors is built into the syndemic perspective, which is one of the advantages of this approach.

In addition, the syndemic approach offers a means of moving beyond a focus on prevention strategies dealing with proximate causes by drawing attention to the processes that create clusters of disease and noxious living conditions for particular populations. As Stall et al. (2003:941) assert, the basic public health question regarding syndemics is “What are the best approaches to disrupting syndemics so that the health of vulnerable populations is enhanced?”

SYNDEMICS, MEDICAL ANTHROPOLOGY, AND THE FUTURE OF GLOBAL HEALTH

Improving public health remains one of the great challenges of the 21st century. As the World Health Organization (2004:4–7) documents, “Almost 57 million people died in 2002, 10.5 million (or nearly 20%) of whom were children of less than 5 years of age... Of these child deaths, 98% occurred in developing countries. Across the world, children are at higher risk of dying if they are poor.” While impressive gains were made in adult health internationally in the last quarter of the 20th century, the subsequent slowdown in the rate of decline in adult mortality has been interpreted by the WHO as a clear warning that further reductions, especially in developing countries, will not be easily realized without changes in contemporary efforts and approaches.

Among the factors contributing to the problem are the forces of globalism that in ways large and small are reshaping the contexts of social life and health everywhere. The ranges of many infectious diseases have increased in part because of more rapid and widespread movement of goods and services worldwide, leading to the global diffusion of pathogens. Included in the global flow of commodities are antibiotics, contributing to their inappropriate use and the development of drug resistant strains of previously controllable infections. Further, as a consequence of resource wars and rural poverty, people move across national boundaries and into concentrated urban centers that facilitate disease onset and transmission. Moreover, as the proportion of elderly people grows in a world in which more than half of the elderly population is found in developing societies (Barrett, in press), we can expect the intersection between infectious and chronic diseases, such as TB and DM, to become an increasingly important health research issue. Understanding the risks that emerge in a changing world requires attention to the kinds of biosocial interactions that are shaping contemporary global health. The syndemic perspective offers one tool for enhancing the armature of public health for its daunting task.

The health of indigenous populations that are socially subordinated within the context of a dominant state structure and political economy is a case in point. In a recent review of the literature, Farmer and Jacklin (2008) point out that in addition to tuberculosis – which we have discussed at some length – other health conditions cluster disproportionately among Aboriginal people in Canada. These include diabetes,

cancer and renal failure – all of which, notably, are medical risk factors for TB. At the same time, Aboriginal communities suffer disproportionately from deleterious social conditions, including poverty, poor nutrition, limited access to or use of health care services, homelessness, overcrowding, alcoholism, and drug abuse. These social and health disparities are linked to the historical relationship of indigenous peoples to the wider society. Here we see the intersection of biological and social conditions that gives rise to syndemics, yet the concept has yet to be applied in any meaningful way by health care providers (Farmer and Jacklin 2008). The syndemics perspective asks researchers to consider how disease synergies have come to be channeled into vulnerable populations – and sustained among them – through social inequalities and systems of power. These kinds of relationships have not sprung up *de novo* but are rooted in historical systems, such as colonial policies and practices, whose effects persist today (Farmer 1999). The continued resonance of historical systems of power into contemporary social relations is exemplified by the excess burden of ill-health among many indigenous populations. We echo Farmer and Jacklin's (2008) call for more participatory and interdisciplinary research that integrates indigenous, biomedical and social science knowledge concerning TB, but in addition suggest that a syndemics perspective is a fruitful avenue for future public health research with disparity populations across the spectrum of human diseases and health conditions.

As suggested, such an approach leads to new frontiers of work for medical anthropologists, such as health problems that transcend the borders of human and non-human animal populations. A starting point in this instance is closer consideration of animal–human connections in our teaching and in our own research. The veterinary community is primed to consider the global reach of socio-economic forces on both animal and human health (Jones et al. 2008; Rock et al., *in press*; Waltner-Toews 2007; Zinsstag et al. 2006), and also the importance of communication and culture for effective practice (Kurtz and Adams 2009). To date, however, we have come across only a single published report on public health training of social scientists that has involved at least one veterinarian (Good 1992), while the 2008 World Veterinary Congress hosted a standing-room-only crowd for a full-day session on global health. We envision much stronger links between veterinarians and anthropologists, for example, among those working with disadvantaged populations (Obrist et al. 2007) and in regions increasingly affected by climate change (Baer and Singer 2003). Similar opportunities for multidisciplinary approaches in syndemics research may develop with medical historians, biologists, microbiologists, virologists, “wet-lab” epidemiologists, and a range of other health related disciplines confronting the rapidly changing world of global health, past and present.

On the intervention front, the syndemic perspective offers an approach for addressing existing fragmentation in prevention and treatment that is needed to address the multiple and entwined health consequences of social disparities (Walkup et al. 2008). Similarly, medical anthropology skills and training are well-suited to prevention-oriented research because of the subdiscipline's focus on health in social context. The “next wave” of contributions from medical anthropologists to public health should include designing, following, and evaluating efforts to attenuate the syndemic potential of various combinations of disease and other afflictions. Of importance in this regard is a needed focus on the various expressions of syndemic phenomena, including sequential epidemics (in which biosocial changes introduced by one epidemic

shapes subsequent epidemics in a population), various kinds of interacting epidemics, such as infectious/noninfectious disease syndemics, counter-syndemics, ecosyndemics (i.e., disease interactions that are mediated by environmental change, such as climate change), and supersyndemics (i.e., two or more independent syndemics that come together and significantly impact a population's health burden), as well as factors that account for the particular patterns of syndemic diffusion (and resistance to diffusion, such as resiliency) within and between populations (Stall et al. 2008).

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