

Indigenous Peoples, tuberculosis research and changing ideas about race in the 1930s

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Before the 1930s, many medical personnel, administrators and others in colonial settler societies considered race the deciding factor in who did and did not get tuberculosis (TB). Racial thinking came in 2 varieties. Both were rooted in white supremacy and in the belief that white bodies and Aboriginal bodies were fundamentally different. Virgin soil theory claimed that Indigenous Peoples, over time, could acquire resistance. Indigenous Peoples, in the language of the early 20th century, were not yet “tubercularized.” On the other hand, there was a theory of racial susceptibility that consigned Indigenous Peoples to a position of perpetual peril; simply being an Aboriginal person meant one was inherently susceptible to TB.¹ The tuberculosis committee of the National Research Council of Canada concluded in 1926 that “it has long been known that Indians are far more susceptible to tuberculosis than are the White races of mankind.”² Confidence in one theory or another, however, was born not from robust research on the susceptibility of Indigenous Peoples but out of deeply held beliefs in the inferiority of their bodies. For decades, neither theory met with much challenge.

Dislodging racial explanations for TB meant overcoming the views of influential TB specialists whose work spanned the colonial world as well as aboriginal North America. R.G. Ferguson was a Canadian physician and public health official who spent most of his career studying TB among First Nations in Canada and whose authority loomed — and looms — large. He began his work in the mid-1920s and by the end of the decade he had become so well known that William Charles White (director of medical services for the U.S. Bureau of



A group of four Navajo adults and a child stand near a nurse who is holding and pointing to a chest radiograph.

Indian Affairs) wrote that Ferguson’s data were “so carefully laid out that I am sure it offers the best opportunity in the world today for studying the epidemiology of TB in man.”³ White hoped Ferguson’s work could be the basis for similar work in the United States. Ferguson practised among First Nations in Saskatchewan on the Qu’Appelle reserve — what he called the Qu’Appelle Research Area. To explore his interest in the problem of resistance, Ferguson compiled data from both historical and clinical sources. He and his team scoured libraries, dug into the records of

the Indian Department, wrote to old settlers and tracked the disease in real time on the Qu’Appelle Reserve. In 1929 he considered lack of resistance to be the primary cause of TB. “It appears,” he wrote, “to outshadow any effect of predisposing factors, such as food, housing, and sanitation, in survival and recession of the epidemic.”⁴ Well into the 1930s, he believed that race-based “inherent susceptibility” was the primary explanation for high rates of TB among First Nations in Canada between 1882 and 1926. He wrote that “To explain these indescribably fatal

epidemics of tuberculosis among primitive peoples in known environments requires the admission of increased susceptibility to the disease [among virgin soil populations].”^{5,6} Ferguson further believed that intermarriage with non-Indigenous people would strengthen resistance to TB in Indigenous Peoples. This kind of crude Darwinian thinking, regarding the “tubercularization” of Indigenous Peoples, was not uncommon.⁷

While Ferguson and others clung to virgin soil and racial susceptibility, research on TB among Indigenous Peoples was making such notions harder and harder to hold onto. In the early 1930s, as researchers fanned out across North America, colonial Tanganyika and elsewhere, the research showed TB as it was rather than as it was imagined to be. Of special interest was research on healed lesions — research possible only with the widespread availability of radiography.⁸ As early as the late 1920s, Ferguson began looking at the relation between healed lesions and resistance. And while he did not shake his belief in racial susceptibility, radiographic evidence convinced him that Indigenous Peoples were able to resist TB. Confusion over the concept of race itself further complicated racial explanations. For example, Charles Walton, in a 1935 article titled “Racial incidence of tuberculosis in Manitoba,” found that misdiagnosis and over-reporting (nearly half of the deaths among Indigenous Peoples that were attributed to TB were reported by non-physicians), as well as poor record keeping, meant it was impossible to get a clear picture of the incidence of TB in Manitoba. Further blurring the picture was an inability to determine who was and was not Indigenous based on the racist notion of “blood quantum.” In instances where Indigenous people were not obviously “full-blooded” — how that was determined went unsaid — there was no consistency in their categorization. As a result, when the Canadian government tried to assess the incidence of TB among Indigenous Peoples “nothing [was] accomplished but confusion.”⁹ Such admissions showed the bankruptcy of hard-held beliefs about either racial susceptibility or virgin soil theory.

Emerging caution regarding the weak explanatory power of blood quantum and

the resulting confusion over the racial identity of Indigenous Peoples, combined with a consensus on the importance of healed lesions, meant that racial explanations for high rates of TB among Indigenous Peoples — worldwide — became harder and harder to sustain by the end of the 1930s. In 1941, James Townsend (the director of health at the U.S. Bureau of Indian Affairs) wrote: “It is our belief that the Indian race, as a race, is no more susceptible to tuberculosis than the white race. In other words, given the same low economic level malnutrition, in some instances, and poor housing, the tuberculosis morbidity rate among whites would more closely approximate that of the Indian. In the thousands of x-rays that have been taken throughout Indian country, it has been found that the pulmonary pathology in the Indian’s chest is practically identical with that of the white tuberculosis subject. This picture shows a tendency to heal.” Townsend based his remarks on the work of Ferguson in Canada and others across the colonial world.

As the popularity of racial explanations waned, economic and social conditions emerged as explanations to fill the breach. In 1936, when Joseph Aronson began his work leading the U.S. Bureau of Indian Affairs’ tuberculosis control unit he found the racial susceptibility argument compelling. It was not until he had begun what he called the “trailblazing project” of TB surveying and vaccination that he even began “to question the validity of the claim that the Indian is peculiarly susceptible [to TB].” Four years later, after surveying thousands of Indigenous Peoples on 4 reservations and in southeastern Alaska, he and his colleagues concluded that social and economic conditions, or what they called “bionomic” factors, were responsible for the high incidence of TB in these communities. They wrote, “The establishment of slum areas in rural districts, rather than ... a peculiar racial susceptibility to the disease,” was responsible for such high rates of infection.¹⁰

Locating TB in social circumstances meant things could change. Acceptance that Indigenous people were not simply doomed to die was a monumental shift in

thinking. In 1936, a physician in Manitoba reported that “we have been accustomed to think of the Indian’s lack of resistance to tuberculosis as entirely racial.” But ideas were changing. Now, it had become clear, “there is no doubt that a great part of the Indian’s low resistance to tuberculosis is due to his abject poverty. Like most other social problems this one is less medical than economic and educational.”¹¹ By the late 1940s, TB researchers had reached an alternative consensus based on surveying and vaccination that involved tens of thousands of Indigenous Peoples — by the end of the 1940s, for example, more than 28 000 radiographs had been analyzed; 17 500 children were vaccinated in the spring of 1949 alone. The epidemiological research carried out in the 1930s and 1940s led Arthur Myers and Virginia Dustin to conclude in 1947 that “Tuberculosis is simply a contagious disease. Wherever it is permitted to exist, it spreads to all races of people. Ideal conditions for its spread have obtained among the so-called ‘primitive’ human races. This fact has been largely ignored while tuberculosis workers have ascribed the disease to such poorly understood factors as low resistance, high susceptibility and lack of immunity.”¹²

None of this is to suggest that because views on race began to change that the health of Indigenous Peoples suddenly improved. To the contrary. By the middle of the 20th century, a doctor on the Navajo reservation claimed that TB cases had doubled. And Fred T. Foard, director of health for the U.S. Bureau of Indian Affairs, called the health of Indigenous Peoples in the US a national disgrace in 1949.¹³ The work done in the 1930s overturned the common view that race was the main driver of TB susceptibility. What distinguished the work done in the 1930s was that scientists came to their conclusions through their extensive field research, whereas those who subscribed to virgin soil theory did so based on assumptions, anecdote and an inability to transcend the prevailing views on race. In much the same way, we now understand that TB is not solely explained by poverty. Indigenous Peoples acquire TB in far greater numbers than non-Indigenous people in Canada and around the world.

This suggests, as historians have been arguing over the last generation, that the ongoing effects of colonization and policies rooted in racism have powerful and lingering effects.¹⁴

Christian McMillen PhD

Corcoran Department of History and associate dean for Social Sciences, University of Virginia, Charlottesville, Va.

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