

# COMMENTARY: THE INVENTION OF ABORIGINAL DIABETES: THE ROLE OF THE THRIFTY GENE HYPOTHESIS IN CANADIAN HEALTH CARE PROVISION

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The purpose of this study was to analyze the extent to which the ‘thrifty gene hypothesis’ remains embedded within regimes of Canadian health care. The thrifty gene hypothesis, formulated by the American geneticist and travelling scientist James V. Neel in 1962, proposed that Indigenous peoples were genetically predisposed to Type 2 diabetes due to the foodways of their ancestors. The hypothesis was functionally racist and based on what biological anthropologists now call ‘the myth of forager food insecurity.’ Importantly, Neel reconsidered his own hypothesis in 1982 before he ultimately rejected it in 1999; nonetheless, in the mid-1990s, a team of Canadian scientists led by the endocrinologist Robert Hegele of Western University conducted a genetic study on the OjjiCree community of Sandy Lake First Nation in northern Ontario. Thereafter, Hegele told the academic world and news media that he had discovered a thrifty gene in Sandy Lake. Like Neel, Hegele later came to reject his own study in 2011. Nonetheless, the ‘thrifty gene hypothesis’ and Hegele’s Sandy Lake study continue to be cited, referenced, and reproduced in the current Clinical Guidelines of the Canadian Diabetes Association, as well as across state-related health literature more broadly. The purpose of this study, then, will be to apply the PHCRP to the thrifty gene hypothesis in a Canadian context. *Ethn Dis.* 2018;28(Suppl 1):247-252; doi:10.18865/ed.28.S1.247.

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## INTRODUCTION

The thrifty gene hypothesis is a failed evolutionary explanation for high rates of type 2 diabetes in Indigenous populations.<sup>1</sup> It was formulated in 1962 by the American geneticist James V. Neel, who proposed that the food procurement strategies of what he termed primitive hunter-gatherers left them chronically underfed.<sup>2</sup> Within Neel’s fanciful narrative framework, the constant selection pressure of food shortages shaped Indigenous bodies all over the world to be metabolically thrifty in terms of fat storage, glucose tolerance, and insulin regulation; however, under conditions of colonial modernity, Neel hypothesized, these thrifty genes became maladaptive and began contributing in a foundational way to high rates of type 2 diabetes in Indigenous communities. Put more bluntly, “Neel imagined Indigenous Peoples as camel-like

beasts with an inherited ability to over-eat during times of plenty.”<sup>3</sup>

Following his development of the thrifty gene hypothesis in 1962, Neel spent the rest of the decade travelling through Brazil, Venezuela, Costa Rica, and Panama collecting Indigenous blood, bone marrow, breast milk, saliva, urine, feces, and other biological materials in an effort to find evidence for the thrifty gene. Though he found some supporting evidence for differential metabolism on these research trips, Neel was ultimately not prepared to stand behind the hypothesis. In 1989, he wrote that “the data on which that (rather soft) hypothesis was based has now largely collapsed.”<sup>4</sup> Ten years later, shortly before he died, Neel insisted that there was “no support to the notion that high frequency of non-insulin dependent diabetes mellitus (NIDDM) in reservation Amerindians might be due simply to an ethnic predisposition.”<sup>5</sup> And yet, the thrifty gene hypothesis outlived its inventor.

In 1999, a team of Canadian scientists conducted a research project in Sandy Lake First Nation – an Indigenous community located in northern Ontario. In their pub-

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lished report,<sup>6</sup> the researchers did not explicitly cite the thrifty gene hypothesis or its creator, Neel; instead, it was one member of this team, the endocrinologist Robert Hegele, who appears to have popularized this study as a thrifty gene discovery. For example, on March 9, 1999, Hegele and his team held a press conference in London, Ontario to announce the findings of the Sandy Lake study.<sup>7</sup> At the time,

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Hegele told *The Globe and Mail* that “the finding certainly has all the earmarks of what a thrifty gene would be.”<sup>7</sup> Hegele also expressed that he felt “very gratified because there was a lot of doubt that this could be done, that a mutation could be

found.”<sup>7</sup> The finding was a newsworthy one for scientists around the world. The *Canadian Medical Association Journal* ran an article announcing, “Gene Defect Driving Diabetes Epidemic on Ontario Reserve.”<sup>8</sup> Whereas, the *British Medical Journal* reported that “a study conducted in a reservation in northern Ontario has identified a genetic mutation that seems to have allowed the Indians there to survive famines in the past but to have triggered diabetes when food became plentiful and their lives became sedentary.”<sup>9</sup> A Chinese news agency also found the study newsworthy, reporting in March of that year: “Canadian researchers have found that a thrifty gene, or genes, may account for the world’s third highest rate of diabetes in the Ojibway-Cree native reserve at Sandy Lake in Northern Ontario.”<sup>10</sup> In short, Hegele’s thrifty gene study put Canadian genomic science on the map, at a time when the human genome project was investing new life, enthusiasm, and funding dollars into the field.

Like Neel before him, Hegele eventually developed doubts about his findings. In 2008, he wrote that “the modern revolution in molecular genetics and biology has focused our attention on the genetic component of disease, at the expense of the environmental component.”<sup>11</sup>

In 2011, Hegele told the *Globe and Mail* that “newer genetic data suggest it’s incorrect to pin the blame for type 2 diabetes on a single gene in any population” and that “the whole thrifty-gene idea seems to me not to capture the subtlety and complexity... of type 2 diabetes in

First Nations communities.”<sup>12</sup> Elsewhere, Hegele has explained that while “the thrifty gene hypothesis might have seemed like a good idea many years ago...current research suggests that in most cases a single mutation in a single gene is unlikely to predispose an entire group of people to a complex outcome like type 2 diabetes.”<sup>13</sup> To review, then, both Neel and Hegele have been clear that the thrifty gene hypothesis is a misrepresentative and problematic way to think about and approach the biosocial reality of nutrition-related diseases in Indigenous communities. The thrifty gene hypothesis has also been challenged by a series of other scientists for its lack of empirical evidence as well as problems with what has been called its ‘assumption of forager food insecurity.’<sup>14-16</sup> Notably, in his 2011 *Making the Mexican Diabetic*, Michael Montoya took the thrifty gene hypothesis to task for its role in geneticizing and racializing type 2 diabetes in Mexican Americans.<sup>17</sup> One would therefore assume that the thrifty gene hypothesis no longer meaningfully informs Canadian health care administration, state literature, or clinical guidelines given its critical treatment by genetic as well as social scientists. Unfortunately, this is not the case.

## THE PERSISTENCE OF THE THRIFTY GENE HYPOTHESIS IN CANADIAN HEALTH CARE

In 1999, the same year as Hegele’s claims on a thrifty gene discovery,

the Canadian government instituted the Aboriginal Diabetes Initiative (ADI), which had an original funding of \$58 million.<sup>18</sup> Further, Hegele and colleagues began work based on the thrifty gene data to develop an intervention strategy to prevent diabetes in younger Sandy Lake Oji-Cree.<sup>19</sup> Thus, it is accurate to claim that anti-Indigenous scientific racism, embedded in the thrifty gene hypothesis, was present at the very inceptions of Aboriginal diabetes as an interventionist public health framework in Canada. More troublingly, the Canadian Pediatric Society's (CPS) *Position Statement on Risk Reduction for Type 2 Diabetes in Aboriginal Children in Canada* (affirmed on February 28th, 2018) cites the 1999 study of Hegele et al.<sup>20,21</sup> Hegele's study is also cited in the *2018 Clinical Practical Guidelines of the Canadian Diabetes Association*, as well as in state literature more broadly.<sup>22</sup> For example, in 2011 (long after Hegele had already rejected his findings), Health Canada issued a report entitled *Diabetes in Canada*, in which the discussion on genetic risk factors suggests that the thrifty gene effect is associated with the increased rates of obesity and diabetes in the Aboriginal population.<sup>23</sup> In 2014, moreover, *Eatright Ontario* released a report on risk reduction for type 2 diabetes in Aboriginal people; this report contained the following passage:

"Traditionally, Aboriginal people lived off the land, which meant sometimes they had little food and at other times there was more than enough. This meant that the Aboriginal people had the genetics to

store fat easily to help provide energy when there was little food. Today Aboriginal people still have the genes to store fat easily but because of easy access to high calorie food and less active lifestyles, this is leading to overweight and obesity."<sup>24</sup>

Suffice it to say, then, that the thrifty gene hypothesis persists as a viable and state-sanctioned explanation for differential rates of type 2 diabetes in First Nations and Inuit communities.

### CRITICAL RACE THEORY (CRT) AND THE PUBLIC HEALTH CRITICAL RACE PRAXIS (PHCRP)

The history of the thrifty gene hypothesis and its curious afterlife in Canadian regimes of health care are powerful examples of the profound need to apply CRT and the PHCRP to public health research on Indigenous communities located in Canada. In discussing their PHCR praxis, Ford and Airhihenbuwa suggested a four-step schematic to help assist in isolating the problems and possibilities associated with doing anti-racist public health research.<sup>25</sup> The first step is to study racism's effect on health and, in order to do so, scholars "must conceptualize racism based on how it operates in the period of interest to the study."<sup>25</sup> In terms of scientific research on the genetic determinants of diabetes in Indigenous communities in Canada, this step requires White and other travelling scientists to commit to understanding the multiple ways in which structural violence

overdetermines public health in Indigenous communities. Indigenous scholar Pam Palmater has offered the concept of "death by poverty in First Nations" as a way to conceptualize the multiple ways in which lack of access to affordable food, clean water, safe shelter, and health-care in some Indigenous communities has led to alarming yet predictable disparities in chronic disease morbidity and mortality on and off reservations.<sup>26</sup> By engaging with Indigenous scholarship, activism, and agitation against structural forms of racism and colonialism, public health researchers can seek to transgress disciplinary quarantines that have thus far prevented science-based studies on aboriginal diabetes from understanding or even commenting upon the structural determinism of community health in Indigenous communities. These structural determinisms result from the existence of grocery store monopolies and defunct federal food subsidy programs that ensure fresh and healthy food is largely unaffordable.<sup>27-29</sup> Simply put, understanding the prohibitively high cost of fresh food in northern Indigenous communities is a necessary step in judging the genetic components of nutrition-related diseases in those communities. If this step is not taken, the trauma of present-day structural violence and colonialism can be geneticized by researchers and unscientifically rendered a consequence of Indigenous biologies.

The second step of the PHCRP, which asks researchers to focus on the politics of knowledge production, is particularly important in

this Canadian story of the thrifty gene hypothesis that unveils the racialized and gendered politics of knowledge production with respect to aboriginal diabetes. The history of the thrifty gene hypothesis demonstrates that when White male scholars such as myself, as well as those who adopt their scholarship uncritically, theorize complex and multi-factorial diseases in Indigenous communities, our hypotheses

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are often privileged over and against community voices in the formulation of public health policy. Significantly, on January 18, 1988, five members of the Sandy Lake Band began a hunger strike at the Sioux Lookout Zone Hospital in northern Ontario as a way to press the federal

and provincial governments to address health care inequities in their community.<sup>30</sup> That the 1999 thrifty gene study of Hegele et al<sup>21</sup> is much more widely recognized and cited in state literature and clinical guidelines than the story of the Sandy Lake Hunger Strike speaks volumes about the need for all scientists to cultivate what Ford and Airhihenbuwa describe as a “deep awareness of one’s racial position.”<sup>25</sup> This is particularly important for White male scientists because of the privilege and power positions we occupy vis-à-vis the production of public health knowledge. As White male researchers, our mistakes can be longer-lasting and much more influential in shaping public health interventions than the rightful political actions of Indigenous communities.

In keeping with the suggestions of the third step of the PHCRP (that is, Conceptualization and Measurement), it seems prudent to point out that the thrifty gene study in Sandy Lake used “fixed racial categories to examine social determinants of health across diverse geographic regions” and thereby contributed to overestimating racial effects and underestimating regional ones.<sup>25</sup> For that reason, immediate action needs to be taken concerning the reproduction of the thrifty gene hypothesis in state literature, clinical guidelines, and scientific textbooks, as well as all scientific studies on Indigenous diabetes and metabolic diseases more generally.<sup>14,16,31</sup>

Action toward resolving these issues, which is the fourth step of the PHCRP, must include proactively challenging similar and ongoing ge-

netic studies into metabolic disease, death, and morbidity in Indigenous communities in Canada. For example, in January 2016, the Canadian Broadcasting Corporation reported on an ongoing study wherein high rates of infant mortality in northern Indigenous communities were being studied as the consequence of a gene-variant.<sup>32</sup> Thus, given the history of the thrifty gene hypothesis and the ongoing nature of these issues within a Canadian context, it is prudent to draw upon and deploy CRT and the PHCRP to aboriginal diabetes historically as well as moving forward. This would involve a recognition by non-Indigenous researchers that there are material realities and forms of structural analysis central to the understanding of chronic disease on the reservation that are simply not accessible to those outside of the reservation. Indeed, even Indigenous scientists who do not live on-reserve in the provincial or territorial norths of Canada may have a hard time understanding local realities of disease, health care, and what Palmater calls ‘death by poverty on First Nations.’

## CONCLUSION

It is clear that both the veracity and integrity of scientific research, as well as the quality of health care provision, would be greatly improved in Canada if more respect was shown for local and Indigenous knowledges of diabetes in public health research. Thus, despite the tendency for Canadian researchers to think overtly in terms

of race, the example of the thrifty gene hypothesis makes it clear that race and racialization both operate within Canadian research. Therefore, race and racialization need to be explicitly named and examined in research projects seeking to understand health disparities in Canada, and especially in the context of nutrition-related illness in First Nations and Inuit communities.

CONFLICT OF INTEREST

No conflicts of interest to report.

AUTHOR CONTRIBUTIONS

Research concept and design: Hay; Acquisition of data: Hay; Data analysis and interpretation: Hay; Manuscript draft: Hay; Administrative: Hay

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