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Diabetes in Canada's First Nations

The comprehensive article on diabetes in Canada's First Nations by Kue Young and colleagues describes a modern epidemic.[1] However, it seems that little has been said about the situation from a historical perspective other than by Chase,[2] to whose reflections Young and colleagues refer. Writing in 1937, Chase noted that "Indians are not subject to diabetes … not because they are all thin … some older Indian women are very fat."[2] If, as many maintain, genetic factors play a role, it is curious that the epidemic was not noted earlier.

In addition, the 2 most devastating complications of diabetes, retinopathy and nephropathy, appear to have been infrequent early in the 20th century. In a classic textbook of the pathology of diabetes published in 1938 it is difficult to locate any reference to these.[3] In a long chapter on diseases of the kidney in his 1945 edition of *Pathology of Internal Diseases*, Boyd devoted only a single paragraph to Kemmelsteil–Wilson nephrosclerosis.[4]

If the triad of elevated blood sugar, overeating and lack of exercise contributes to diabetes and its microvascular complications then why was such a cause-and-effect relationship not apparent prior to World War II? Perhaps the blame should not be on eating and exercise habits, but on the quality of "white man's food." Could there be nutritional deficiencies or toxic additives in modern food that are at least partly responsible for the increased frequency of the disease and its complications?

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Major tobacco companies have long recognized that tobacco use may cause hyperglycemia.[1,2] Current or lifetime smoking or use of smokeless tobacco is very likely to cause diabetes as well as many of the severe complications experienced by tobacco users who have diabetes.[3–5]

Approximately one-quarter of diabetes cases among Canada's First Nations could be attributed to smoking,

even assuming relative risks (RR) of smoking-related diabetes as low as 1.5 and a smoking prevalence among First Nations populations of 64% (1/4 = [(RR – 1) × prevalence]/[1 + (RR – 1) × prevalence]).[6] Given that tobacco use probably plays a leading role in causing type 2 diabetes and its complications, I hope that physicians, researchers, governments and tobacco packagers will soon better inform the public about tobacco's roles in diabetes.

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[One of the authors responds:]

William Panton raises an interesting issue regarding the rarity of historical reports of type 2 diabetes among Aboriginal people in Canada. The fact that "the epidemic was not noted earlier" does not preclude a genetic explanation for the susceptibility of Aboriginal people to this disease. Space limitations prevented us from more fully discussing the gene–environment interactions involved in the emergence of the diabetes epidemic in the past 3 decades.[1] James Neel, who first proposed the "thrifty genotype" theory, recently published an update,[2] which we cited in our review.[1] We also cited several dietary studies, but few dietary culprits have been consistently identified. Panton's comments about nutritional deficiencies or toxic additives in "white man's food" are purely speculative at this stage but perhaps warrant further study.

I appreciate the fact that Bruce Leistikow has pointed out references concerning the association between tobacco use and hyperglycemia. According to data from the First Nations and Inuit Regional Health Survey, current smokers are *less* likely to be diabetic (8.4%) than people who are not currently smokers (13.4%) (the latter group includes people who have never smoked and people who are former smokers).[3] A possible explanation for this finding is that people who are diagnosed with diabetes may make lifestyle changes that include stopping smoking. Because of the cross-sectional nature of the data it

is not possible to delineate the temporal sequence between the onset of smoking and the onset of diabetes. However, the potential link warrants further inquiry.

I would, however, caution against hoping that removing a single factor, be it dietary toxins or tobacco smoke, will vanquish the diabetes problem in Canada's Aboriginal population. Current evidence points to a very complex etiology and to date no magic bullets have been found.

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Reducing the rates of inappropriate labour induction

Susan Harris and colleagues have described a clinical quality improvement (CQI) initiative to reduce the rates of inappropriate induction of labour.[1] They claim that their initiative was associated with a sustained reduction in induction rates and recommend that "similar projects be undertaken at other institutions." We are unconvinced that their data support these conclusions. Specifically, the authors provide only descriptive data without statistical testing. We reanalysed the data using time-series regression models, which allow assessment of and adjustment for preintervention time trends.[2,3] Although our reanalysis has limited statistical power owing to the small number of data points, we found a decreasing trend in induction rates before the intervention (0.45% decrease per 6 months, $p = 0.10$) and no evidence of a continuing trend after the intervention (0.11% decrease, $p = 0.64$). However, there was evidence of an overall shift in pre- to post-intervention rates (absolute reduction of 2.6% in the 6 months following the intervention, $p = 0.06$). This could be due to a small intervention effect, although we are uncertain of its clinical significance. We invite the authors to consider conducting a more powerful time-series analysis by disaggregating their data into shorter intervals that still allow stable point estimates of performance.

The authors state that their CQI initiative was "very time-consuming," representing "a significant cost to the institution." Hospitals have limited resources to spend on quality improvement. There are substantial opportunity costs if hospitals adopt unproven methods. If we are to generate a robust evidence base for quality improvement activities, we should demand that quality improvement strategies be evaluated with the same scientific standards that are used to evaluate any clinical intervention. This paper fails to provide compelling evidence that CQI works or provides good value for money. Further evaluation is required before widespread adoption of CQI can be recommended.

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[Three of the authors respond:]

We appreciate the response to our paper[1] from Craig Ramsay and colleagues; we reached a conclusion similar to theirs when we initially analysed the data, recognizing that induction rates were already declining when we implemented the intervention. We did include statistical testing in early drafts of the paper but this information was omitted at the editors' request. Table 1 shows how logistic regression was used to calculate odds ratios for induction with the first time period (January–June 1994) as the reference category. The odds of having an induction in 1997 and 1998 were significantly less than in the reference time period; in contrast, the odds of having an induction in the time periods before 1997 did not differ from those in the reference time period.

We then created a logistic regression model with time periods 1, 2, … 10 entered as a continuous variable, which allowed us to compare the slope of the change in the induction rate for different groups of time periods. The slope of the change in induction rate

Table 1: Odds ratios expressing changes in the frequency of labour induction at British Columbia's Women's Hospital and Health Centre, 1994–1998

	Odds ratio (and 95% confidence intervals)	*p* value
Jan–Jun 1994*	1.00	
Jul–Dec 1994	1.12 (1.00–1.25)	0.040
Jan–Jun 1995	1.09 (0.98–1.22)	0.113
Jul–Dec 1995	1.03 (0.93–1.22)	0.576
Jan–Jun 1996	0.96 (0.86–1.07)	0.490
Jul–Dec 1996	0.93 (0.83–1.04)	0.219
Jan–Jun 1997	0.82 (0.73–0.92)	0.001
Jul–Dec 1997	0.76 (0.67–0.85)	0.000
Jan–Jun 1998	0.80 (0.71–0.90)	0.000
Jul–Dec 1998	0.78 (0.70–0.88)	0.000

*Reference category