Diabetes in Aboriginal populations

I am concerned that Philip Hall's description of the health status of aboriginal people in North America before their first contact with Europeans[1] might, albeit unintentionally, reinforce the stereotype of a robust, essentially disease-free "noble savage" practising good nutrition supplemented with plenty of fresh air and exercise. The only infectious disease predating European contact that Hall alludes to is syphillis; he lists tuberculosis among the post-contact diseases. In fact, *M. tuberculosis* acid-fast bacilli have apparently been found in the mummified remains of a young Peruvian boy whose death is estimated to have occurred approximately 800 years before the arrival of Columbus.[2] Tubercular-type bony lesions have also been documented in skeletal remains discovered in a pre-contact ossuary in southern Ontario.[2] Moreover, the traditional food supply was neither pristine nor ideal. Examination of coprolites provides evidence of meat-borne parasitic infections that could at worst infect the lungs and central nervous system or at best the alimentary tract. The maize diet produced circular dental caries similar to lesions found today in children from developing countries, a consequence of malnutrition caused by chronic diarrhea.[2]

I agree that the prevalence of diabetes is influenced by both geographic and genetic variables, but I would add socioeconomic variables to the list. A recent study of prescription drug utilization among aboriginal people in Canada shows higher rates of use of certain analgesic and anxiolytic drugs than among the general Canadian population; these are not unlike the differences in rates of diabetes between the 2 groups.[3] However, drug utilization rates among aboriginal people are strikingly similar to the rates within the portion of the nonaboriginal population whose socioeconomic characteristics are similar to those of the aboriginal population. These characteristics include poverty, unemployment, lower education and substandard housing. Furthermore, although aboriginal Canadians with diabetes may die from the disease at a rate 2 to 4 times higher than that among other Canadians with diabetes, overall mortality rates among aboriginal Canadians are also significantly higher. We must consider factors beyond genetics and lifestyle when we explain differences in health status.

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[The author responds:]

I agree with most of John Anderson's assertions, especially the last sentence, and if my writing reinforced anyone's stereotypes that was unintended. My purpose was to provide a partial explanation of diabetes as a novel epidemic in North American aboriginal groups and to argue that the epidemic of type 2 diabetes world wide has its main roots in genetic predisposition, dietary change, obesity and sedentarism.[1,2] I am sure that *CMAJ* readers would realize that the disease is also correlated intimately with socioeconomic variables.

Anderson may be correct that I should not have included tuberculosis in my list of infections unknown to "New World" tribes before European contact; in addition to the remains he mentioned, tubercular DNA was extracted from various human bones from Chiribaya Alta in southern coastal Peru. However, some researchers still consider that the presence of tuberculosis in the Americas before Columbus is unproven.[3]

I chose to refer to syphilis partly because, aside from tobacco, it was the only New World export that had a devastating effect in Europe. There are as many bittersweet ironies about the chronicles of tuberculosis and syphilis as there are about diabetes. The 2 infections have been linked, and confused, for centuries. In 1527 Jacques de Béthencourt provided a remarkably accurate description of the pox, stressing its polymorphic character, that it could cause osseous deformity and cavitation, and that it shared some characteristics of consumption. The answer to one of *M. tuberculosis*' enduring mysteries — how and when it migrated globally[4] — may remain forever obscured by the organism's antiquity. It is curious that syphilis and tuberculosis penetrated European populations thoroughly, whereas native North Americans – judging from history and the examination of human remains — were more resistant to both infections. While 1 in 4 16th-century Europeans were dying from tuberculosis, their Mesoamerican contemporaries were dying in large numbers from the more direct results of contact with the conquistadores. Although the Peruvian boy mentioned by Dr. Anderson took his last tubercular breath around 700 AD, his culture expired when Francisco Pizarro's troops executed Atahualpa, the last Incan Emperor, in 1532.

There is stong evidence that syphilis was brought to Europe on Columbus' caravels. Contemporary European authors agreed that the disease was new. For example, Gonzalo Fernandez de Oviedo y Valdes was at court when Hernando and Isabel received Columbus after his first voyage. After interviewing the sailors and their captives he considered it to be "certain that this malady — the bubas — comes from the Indes, where it is very common amongst the Indians."[5]

When Columbus died, crippled by gout and arthritis, in 1506, it is unlikely that he had any idea of the consequences of his ventures. Nonetheless, the world had been "changed, uprooted

and catapulted out of life-patterns that had endured for thousands of years."[6]

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A worthy Web site

I recently underwent a set of screening, then diagnostic, mammograms, followed by 2 ultrasounds and a biopsy. I found trying to gather reliable and comprehensive information about breast health issues by phone, in person and online to be an exercise in sleuthing and perseverance — until I came across the *CMAJ* Web site (www.cma.ca/cmaj), which is offered at no subscription cost. It provided a clear, concise and complete guide to all of the questions to which I needed answers in order to be informed at each stage of the screening and diagnosis process.

Thank you for providing an informative and helpful online resource to both health care practitioners and lay people.

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Anticoagulant prophylaxis against stroke

Jaime Caro and colleagues are to be congratulated for their study confirming the beneficial effect of anticoagulants to prevent stroke in atrial fibrillation.[1] In the same issue Stuart Connolly asks why so many eligible patients are not receiving anticoagulant therapy.[2] I would suggest the following possible reasons.

First, patients may be reluctant to go to a testing laboratory on a regular basis. They may also be concerned about the restrictions the use of blood thinners may impose on their lifestyle.

Second, there is the issue of informed consent. Using the results of the study by Caro and colleagues, a diligent physician might explain to a patient that the risk of stroke in individuals taking warfarin is 2.3 per 100 person-years as opposed to 6.7 per 100 person-years in the no-treatment group and that the hazard rate from bleeding is 3.4 per 100 person-years in the warfarin group versus 1.9 per 100 person-years in the no-treatment group. The patient might assume that taking warfarin would mean going from the frying pan into the fire.

The third reason is physician reluctance. Connolly makes no mention of the increased workload anticoagulant therapy places on the treating physician and his or her staff. Whenever a patient goes for a blood test, the international normalized ratio (INR) results are typically phoned into the physician's office. The physician must then modify the dose as required and notify the patient of any changes. This requires several phone calls and can be a major source of anxiety (and possible medicolegal liability) when, for whatever reason, the doctor's office is unable to reach the patient to make the required medication changes. Admittedly, in BC physicians do get paid the princely sum of $2.73 for providing this service.

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Reference-based pricing

We found the articles by Lutchmie Narine and colleagues[1] and Chantal Bourgault and associates[2] to be of particular interest, as we have been involved in the development of the Reference Drug Program in BC from its inception. The program has received both criticism and accolades since it was launched in the fall of 1995. Criticism has come primarily from the pharmaceutical industry, as any savings that governments achieve from the application of the policy are also reduced profits for the drug companies. Indeed, it has been stated that one gauge of any policy's effectiveness is the vigour of the industry response.[3] Accolades have come from those who recognize the importance of a sustainable drug program for the long term.

Narine and colleagues[1] attempt to draw correlations between the reference pricing policies in Europe and those in BC. Although there may be some similarities, there are significant differences. The primary focus of the policy in BC is the baseline prescribing habits of physicians. The policy is designed to ensure that the most cost-effective agent within a drug class is used initially. If there are particular patient circumstances that would justify the use of a more costly agent, such as an adverse reaction or lack of therapeutic effect, the alternative agent is funded fully. In addition, the Reference Drug Program in BC does not target generic equivalents as stated in the article, but rather it targets competing drugs in a class.

Bourgault and associates[2] review the utilization of a select group of angiotensin-converting-enzyme (ACE) inhibitors, as well as hospital admissions and physician visits. Although the authors speculate that there are therapeutic differences among the ACE inhibitors, they present little evidence to support this assertion.

We agree with the critical comments by editorialists Paul Grootendorst and Anne Holbrook.[4] There are many plausible explanations for the differences in health services utilization rates ob-