

Preventing a Cardiovascular Disease Epidemic among Indigenous Populations through Lifestyle Changes

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ABSTRACT

Cardiovascular disease (CVD) is the driving force behind the discrepancy in life expectancy between indigenous and non-indigenous groups in many countries. Preceding CVD many indigenous groups exhibit a cluster of cardiometabolic risk factors, including overweight-obesity, diabetes, high cholesterol, and high blood pressure. In turn, modifiable lifestyle risk factors contribute to the development of this cluster of cardiometabolic conditions. Modifiable lifestyle risk factors include, but are not limited to, physical inactivity, poor nutrition, excessive alcohol consumption, and cigarette smoking. Notably, these metabolic and lifestyle risk factors are relatively simple to monitor and track. The current review will look at modifiable cardiometabolic (overweight-obesity, diabetes mellitus, high cholesterol, and high blood pressure) and lifestyle (physical inactivity, poor nutrition, risky alcohol behavior, and cigarette smoking) risk factors among indigenous populations from Australia (Aboriginal Australians and Torres Strait Islanders), New Zealand (Māori) and the United States (Native Americans). Discussion will focus on the causal relationship between modifiable lifestyle risk factors and cardiometabolic outcomes, as well as, simple measurements for tracking these risk factors.

Keywords: Heart disease, endothelial dysfunction, Maori, Aboriginal Australian, Native American.

INTRODUCTION

There are more than 370 million indigenous people in 70 countries worldwide.^[1] Indigenous people are not monolithic, there is a significant variation between and within these indigenous populations in terms of worldview, culture, political forces, education, socioeconomic status, living conditions, and familial factors. However, many indigenous groups do share a striking commonality: A discrepancy in life expectancy when compared to their non-indigenous kinsman — even in the so-called ‘wealthy’ countries, including Australia, New Zealand, and the United States. For example, the Aboriginal people of Australia have a life expectancy of 62 years, versus 81 years for non-indigenous Australians;^[2] the Māori of New Zealand have a life expectancy of

73 years, versus 81 years for their white kinspeople;^[3] and the Native Americans have a live expectancy of 75 years, versus 78 years for their white counterparts.^[4]

Cardiovascular disease (CVD) is the driving force behind the discrepancy in life expectancy between indigenous and non-indigenous populations in many countries.^[5] Preceding CVD, many indigenous groups exhibit a cluster of *modifiable* cardiometabolic risk factors, including overweight-obesity, diabetes mellitus, high cholesterol, and high blood pressure. In turn, modifiable lifestyle risk factors contribute to the development of this cluster of cardiometabolic conditions [Figure 1]. Modifiable lifestyle risk factors include, but are not limited to, physical inactivity, poor nutrition, risky alcohol behavior, and cigarette smoking. Notably, these metabolic and lifestyle risk factors are relatively simple to monitor and track [Table 1].

The current review will look at modifiable *cardiometabolic* (overweight-obesity, diabetes mellitus, high cholesterol, and high blood pressure) and *lifestyle* (physical inactivity, poor nutrition, risky alcohol behavior, and cigarette smoking) risk factors among the indigenous populations from Australia (Aboriginal Australians and Torres Strait Islanders), New Zealand (Māori), and the United

States (Native Americans). Discussion will focus on the causal relationship between modifiable lifestyle risk factors and cardiometabolic outcomes, as well as, simple measurements for tracking these risk factors.

MODIFIABLE CARDIOMETABOLIC METABOLIC RISK FACTORS

The following section will highlight the prevalence of modifiable cardiometabolic risk factors for CVD: Overweight-obesity, diabetes mellitus, high cholesterol, and high blood pressure. Each of these risk factors is relatively simple to monitor [Table 1].

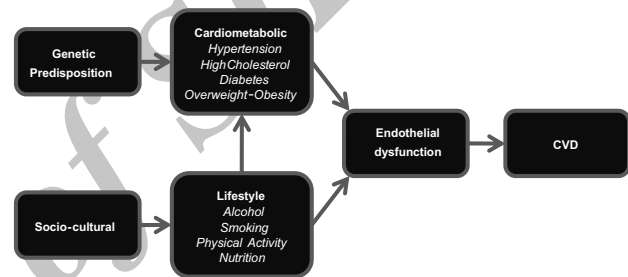


Figure 1: Causation pathway for cardiovascular disease

Table 1: Lifestyle and metabolic risk factor analysis

Risk factor	Metric	Optimal	Low risk	High risk	Disease outcomes	Ref.
Endothelial dysfunction	Flow-mediated dilation (%)	≥10	≤7	≤4.5	Atherosclerosis	6,7
High blood pressure	Systolic blood pressure (mmHg)	<120	130–139	≥140	CVD; hypertension; renal failure	8
	Diastolic blood pressure (mmHg)	<80	80–89	≥90		
High blood glucose	Fasting plasma glucose (mg/dl)	<100	100–125	≥126	CVD; diabetes; cancers	9
High cholesterol	LDL-cholesterol (mg/dl)	<100	100–129	≥190	CVD	10
Overweight-obesity	Waist:hip ratio ¹	M: ≤0.90 F: ≤0.80	M: ≥0.92 F: ≥0.82	M: ≥0.98 F: ≥0.88	CVD; hypertension; diabetes; cancers	11-13
Alcohol use	Standard drinks/day	M: ≤1 F: ≤1	M: ≥3 F: ≥2	M: ≥5 F: ≥4	CVD; respiratory disease; cancers; diabetes; digestive disorders	14
Poor nutrition	Fruit and vegetable (servings/day)	≥5	< 5	< 1	CVD; cancers	15,16
Physical inactivity	Moderate physical activity (minutes/day)	≥30 days (5 days/week)	<30 days	Sedentary	CVD; cancers; diabetes; hypertension	17
Tobacco smoking	Cigarettes/day	0	≥1	≥1	CVD; respiratory disease; cancers; diabetes; hypertension	18,19

M: Males; F: Females; CVD: Cardiovascular disease; HT: Hypertension; LDL: Low-density lipoproteins

OBESITY

Much higher rates of obesity have been found for the indigenous populations of Australia,^[20] New Zealand,^[21] and the US^[15] Excess body fat increases the risk of developing a range of health problems, including high blood pressure, diabetes mellitus, and CVD. Population studies typically estimate the prevalence of overweight/obesity by calculating an individual's Body Mass Index (BMI) score.^[22] Despite widespread use, BMI has been heavily criticized.^[23-28] The BMI is calculated by dividing weight by height, and it is assumed that weight equates to body fat.

Romero-Corral *et al.*,^[23] undertook a meta-analysis to determine the nature of the relationship between obesity and cardiovascular mortality in patients with CHD. Patients with severe obesity (BMI ≥ 35) had the greatest relative risk (RR) for cardiovascular mortality (RR=1.88), compared to people with a normal BMI (BMI 20 – 24.9). However, overweight patients (BMI 25 – 29.9) had the lowest risk (RR=0.88), and obese patients (BMI 30 – 35) had no increased risk (R=0.97). The authors suggested that these findings could be explained by the lack of discriminatory power of BMI to differentiate between body fat and lean mass.

Waist circumference, waist-to-height ratio, and waist-to-hip ratio (WHR) take into consideration body-fat distribution, especially abdominal obesity, and appear to be better predictors of CVD than BMI.^[29] A recent study compared the predictive power of BMI, waist circumference, waist-to-height ratio, and WHR for diabetes mellitus, hypertension, and dyslipidemia in Australian Aboriginal and Torres Strait Islander adults.^[28] The WHR was found to have the greatest predictive power. A number of studies, investigating a range of ethnic groups, have found WHR to better predict cardiometabolic and cardiovascular risk factors than BMI.^[24-27] However, studies over the past two decades indicate that cutoffs for WHR differ by ethnic groups, therefore, the reference values provided in Table 1 should be used to provide a guideline,^[13] not to ascertain the absolute risk.

DIABETES MELLITUS

The prevalence of diabetes mellitus is up to three times greater when comparing the indigenous populations of Australia,^[20] New Zealand,^[21] and the US^[15] to their non-indigenous counterparts.

Diabetes mellitus is a metabolic disease in which high blood glucose levels result from defective insulin secretion, insulin action or both.^[30] Diabetes is a worldwide epidemic and a major risk factor for CVD.^[15,31-35] The prevalence of diabetes for all age groups worldwide was estimated to be 2.8% in 2000 and is projected to be 4.4% in 2030.^[31] CVD accounted for the primary cause of death for all patients with diabetes.^[15] Several modifiable risk factors play a role in the onset of diabetes mellitus, including obesity, physical inactivity, and poor nutrition, as does genetic predisposition and aging.^[36-40] Diabetes mellitus risk can be monitored by measuring fasting blood glucose. A fasting blood glucose of <100 mg/dl is considered optimal.^[8]

HIGH CHOLESTEROL AND OTHER LIPIDS

The prevalence of high total cholesterol is greater for the indigenous population of the US^[15] compared to the general population, but not for the indigenous populations in Australia^[20] and New Zealand.^[21] However, the lack of differences in cholesterol between the indigenous and non-indigenous populations in Australia and New Zealand, in these large surveys, may be misleading, as only total cholesterol was measured in the Australian survey, and in the New Zealand survey, the participants reported medicated high cholesterol levels, and thus, there were likely undiagnosed cases.

The two most common blood lipids are cholesterol and triglycerides. These two blood fats are carried on particles called lipoproteins, the most important of which are low density lipoprotein (LDL) and high density lipoprotein (HDL). Both carry cholesterol, but it is high LDL-cholesterol levels that have been shown to be pro-atherogenic,^[41-45] whereas, low levels of HDL-cholesterol are associated with increased *Coronary Heart Disease* (CHD), morbidity, and mortality.^[46-49] Conversely, high HDL-cholesterol levels convey reduced risk.^[46-49] In population studies, serum total cholesterol is often used as a surrogate for LDL-cholesterol levels, however, measurement of LDL-cholesterol concentrations confer greater predictive value for cardiovascular events.^[41-45] High cholesterol usually has no symptoms, and people may not be aware they have the condition

unless they have had a blood test. Therefore, the best way to determine the true prevalence of high cholesterol in the community is through blood samples.^[21] An LDL-cholesterol level <100 mg/dl is considered optimal.^[50]

HYPERTENSION

The indigenous populations of Australia^[20] and New Zealand,^[21] but not the US,^[15] have higher rates of hypertension compared to the general population — however, it should be noted that the prevalence of hypertension in the US among the total population is particularly high.^[15] Hypertension is a major risk factor for CVD. For every 20 mmHg systolic or 10 mmHg diastolic increase in resting blood pressure, there is a two-fold increase in mortality from both ischemic heart disease and stroke.^[51] Hypertension is associated with shorter overall life expectancy, shorter life expectancy free of CVD, and more years lived with CVD.^[52] A systolic blood pressure <120 mmHg and a diastolic blood pressure <80 mmHG is considered optimal.^[8] Blood pressure should be monitored using the auscultatory method, with a properly calibrated device, following five minutes of quiet rest in a chair.^[8]

MODIFIABLE LIFESTYLE RISK FACTORS

The following section will discuss the prevalence of poor nutrition, risky alcohol behavior, physical inactivity, and cigarette smoking. Although not exhaustive, these variables represent *modifiable* lifestyle risk factors, which have been proven to modulate the cardiometabolic factors discussed earlier in the text.

ALCOHOL

In Australia,^[20] New Zealand,^[53] and the US,^[54] there is a lower prevalence of any alcohol consumption among the indigenous population compared to the general population. However, the indigenous people of each country are more likely to exhibit risky alcohol behavior (binge drinking: ≥ 5 standard drinks/day for males, ≥ 4 standard drinks/day for females). Although some scientific evidence indicates that light-to-moderate alcohol consumption may significantly reduce the risk

of CVD and all-cause mortality, but excessive alcohol intake is toxic to both the heart and overall health.^[14,55,56] In contrast, excessive alcohol intake is toxic to both the heart and overall health.^[14,55,56] In particular, binge drinking, even among otherwise light drinkers, increases cardiovascular events and mortality.^[14,55,56] The American Heart Association guidelines caution people not to start drinking if they do not already drink alcohol, because it is not possible to predict in which people alcohol abuse will become a problem.^[57]

NUTRITION

Low fruit and vegetable consumption has been reported for the indigenous people of Australia,^[20] New Zealand,^[21] and the US^[58] A diet high in fruits and vegetables can reduce the risk for many leading causes of death.^[15,16,59-61] In a meta-analysis of prospective cohort studies, each daily serving of fruits or vegetables was associated with a 4% lower risk of CHD (RR: 0.96, 95% CI: 0.93 to 0.99) and a 5% lower risk of stroke (RR: 0.95, 95% CI 0.92 to 0.97).^[60,61] Five or more daily servings of fruit and vegetables are considered optimal.^[15,16] FFQs, including the freely available National Cancer Institute Diet History Questionnaire (<http://riskfactor.cancer.gov/dhq2/>),^[62,63] allows for the assessment of the usual patterns of food intake over an extended period of time.^[64,65] FFQs are inexpensive in both time and cost, in comparison to other measurement tools, which is an important consideration in studies involving large cohorts.^[66]

PHYSICAL INACTIVITY

Higher rates of sedentary behavior have been reported for the indigenous populations of Australia^[20] and the US,^[67] but not New Zealand.^[21] However, while similar physical activity levels have been reported for the Māori of New Zealand compared to the non-indigenous population, large scale studies are limited. It has been estimated that physical inactivity is responsible for 12% of the global burden of heart attacks.^[68] Regular physical activity reduces CVD risk in its own right and also improves CVD risk factors such as overweight, high blood pressure, high cholesterol, and diabetes.^[69-74] The American College of Sports Medicine (ACSM) recommends at least 30 minutes of moderate-intensity physical activity (e.g., walking briskly,

mowing the lawn, dancing, swimming, bicycling) at least five days a week.^[17]

A number of tools have been developed to measure physical activity, ranging from objective measures such as accelerometry to subjective questionnaires.^[75] Physical activity questionnaires are prone to technical error, but are inexpensive, practical to use in population studies, and can provide information about the type of physical activity and context.^[75] The International Physical Activity Questionnaire (IPAQ) (<http://www.ipaq.ki.se/ipaq.htm>) is a freely available, cross-national monitoring tool, which has been validated for use in adults^[76-80] and children.^[81-84]

CIGARETTE SMOKING

Much higher rates of cigarette smoking have been reported for the indigenous populations of Australia,^[20] New Zealand,^[21] and the US,^[85] compared to their non-indigenous counterparts. Cigarette smoking increases the incidence of CVD in a dose-dependent manner,^[86-92] with even occasional smoking increasing the risk of CVD.^[93] The relationship between smoking and CVD lies in the multiple mechanisms that interact to contribute to atherosclerosis, vascular injury, vascular dysfunction, and thrombosis, although the precise mechanisms are largely unknown.^[93-95]

MONITORING CARDIOVASCULAR DISEASE: ENDOTHELIAL FUNCTION

Upsetting the delicate balance of functions performed by the endothelium initiates a number of events that promote atherosclerosis, the precursor to CVD.^[96-98] Although atherosclerosis is commonly described as the presence of plaques that obstruct the lumen of the conduit arteries, endothelial dysfunction precedes plaque formation.^[99-101] Reduced endothelial responses can be observed early in the course of atherogenesis, preceding angiographic or ultrasonic evidence of the atherosclerotic plaque.^[102] There is, therefore, widespread interest in the application of clinical tools, to assess the function and health of this essential monolayer.

Several biomarkers are available to identify the health of the endothelium, including total nitric oxide (NO), asymmetric dimethylarginine

(ADMA), dimethylarginine (DDAH), and endothelin-1 (ET-1). ADMA is an endogenous inhibitor of NO synthases — NO is one of the most important molecules regulating endothelial function.^[103,104] ADMA plasma concentration is elevated in numerous populations with vascular diseases or at high cardiovascular risk.^[105-107] ADMA concentrations have also been found to significantly correlate with flow-mediated dilation (FMD), the gold standard for evaluating endothelial function.^[108]

Endothelial function can also be evaluated non-invasively by using strain-gauge plethysmography,^[109-112] pulse-wave analysis,^[113-115] or flow-mediated dilation (FMD). The FMD test is the standard tool used to assess endothelial function.^[116,117] Reduced FMD, an early marker of atherosclerosis^[116], has been noted for its capacity to predict future CVD events,^[6,118-120] and an impaired vascular response has also been demonstrated in children as young as seven years of age with familial hypercholesterolemia.^[121] A recent meta-analysis by Inaba *et al.*^[6] reported that the relative risk of cardiovascular events for a 1% absolute change in FMD is 0.87. This suggests that a 1% decrease in FMD is associated with a 13% (95% CI: 9 to 17%) increase in the risk of future cardiovascular events. A number of authors have developed standardized guidelines for conducting this test.^[117,122-126]

DISCUSSION

Cardiovascular disease is the driving force behind the discrepancy in life expectancy between the indigenous and non-indigenous groups in many countries.^[5] This trend extends to the indigenous populations of Australia, New Zealand, and the US. The historical underpinnings of this trend can be seen in the devastating effects of colonization on the health outcomes for indigenous people.^[127,128] This review has provided a working model to outline the causal relationship between lifestyle choices, cardiometabolic risk factors (i.e., modifiable lifestyle-related diseases) and CVD. Guidelines have also been provided to monitor these risk factors.

Strategies for implementing change are beyond the scope of this article. There is a paucity of large-scale, methodologically rigorous *interventions*¹ designed to

1 The term 'intervention' is used in accordance with the scientific literature. The authors acknowledge that this term may be construed as culturally insensitive.

improve indigenous health outcomes,^[129] and as such, there is a limited evidence base to guide healthcare scientists and practitioners. Paul *et al.*,^[129] recently identified 811 publications focusing on the major causes of death and illness for indigenous populations in Australia, New Zealand, the US, and Canada, and only 43 of these were identified as *intervention* studies. Of the 43 studies identified by Paul *et al.*, only 19 were judged to be methodologically rigorous.^[130,131] The preponderance of the literature on the health of indigenous populations is focused on describing or understanding problems, rather than on testing the effectiveness of the potential solutions. A small number of intervention studies do meet the standards for appropriate research methodology, but they do not provide a body of evidence in relation to any particular population, health topic, or intervention type. There remains a pressing need to develop culturally sensitive strategies geared toward improving the lifestyles of indigenous populations. The guidelines outlined in the current article will assist future research focusing on indigenous health outcomes.

CONCLUSION

Cardiovascular disease is the driving force behind the discrepancy in life expectancy between the indigenous and non-indigenous groups in many countries. Much of this risk can be offset through lifestyle changes. The current review provides a simple working model for managing and monitoring CVD risk among the indigenous populations.

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