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The Combined Burden of Diabetes and Cardiovascular Disease in Indigenous Australians

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Abstract Type 2 diabetes mellitus (T2DM), cardiovascular disease, and chronic kidney disease are significant contributors to the 17-year disparity in life expectancy between Indigenous and non-Indigenous Australians. These three conditions are prevalent from a young age in Indigenous Australians and clearly contribute to their premature mortality. Risk factors that both exacerbate and promote these conditions include central obesity, dyslipidemia, cigarette smoking, albuminuria, inflammation, and poor socio-economic status. Although rates of screening for T2DM are higher in Indigenous Australians than in non-Indigenous Australians, gaps in clinical management of both T2DM and cardiovascular disease exist. To enhance survival and quality of life, prevention strategies are required at a population level and from a young age in Indigenous Australians.

Keywords Cardiovascular disease · Diabetes · Indigenous Australians · Aboriginal · Metabolic syndrome

Introduction

The term "Indigenous Australians" collectively refers to the two groups of Indigenous Australian people: Aboriginal people and Torres Strait Islander people. Aboriginal people are the original inhabitants of mainland Australia, Tasmania, and other smaller islands of Australia; Torres Strait Islander people are the original inhabitants of the Torres Strait Islands, located in the far northeast of Australia, at the most northern part of Queensland (close to Papua New Guinea). The 2006 Australian census reported that within the total population of 19.9 million Australians, 455,028 people self-identified as Indigenous Australians (2.3% of the total population) [1]. Of those identified as Indigenous Australian, 90% were of Aboriginal origin only, 6% were of Torres Strait Islander origin only, and 4% were of both Aboriginal and Torres Strait Islander origin. The estimated resident Indigenous population of Australia in 2006 (after adjustment for net undercount and unknown Indigenous status) was 517,174 or 2.5% of the total Australian population [1]. The census count of Indigenous Australians has doubled over the past 20 years (from 277, 593 in 1986; Indigenous Australians were not formally included in the Australian census until after 1967). This can largely be explained by natural reasons, but non-demographic factors also play a role (improvements in Census collection methods and people being identifed for the first time as Indigenous) [1]. The Indigenous Australian population has a much younger age structure than the non-Indigenous Australian population: the median age of Indigenous Asutralians is 21 years compared to 37 years for non-Indigenous Australians; and 38% of Indigenous Australians are under 15 years of age compared to 17% for the non-Indigenous Australian population [1]. Indigenous Australians are a very hetergeneous group of people, with significant hetereogeneity not only between Aboriginal and Torres Strait Islander peoples but also within Aboriginal peoples. There is significant diversity within and between Aboriginal and Torres Strait Islander people for factors such as language, culture, socioeconomic status, education, body build, body composition, and ethnic admixture.

In 2006, approximately one third of Indigenous Australians lived in major cities (32%), 21% in inner regional areas, 22%

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in outer regional areas, 10% in remote areas, and 16% in very remote areas [1]. Remoteness is defined according to the ARIA (Accessibility/Remoteness Index of Australia) codes such that remote (and very remote) locations have very restricted (or very little for very remote) accessibility of goods, services, and opportunities for social interaction [2]. A greater proportion of Indigenous than non-Indigenous Australians live in remote or very remote regions (26% of Indigenous Australians compared to less than 2% of non-Indigenous Australians) [1].

Indigenous Australians have rates of cardiovascular disease (CVD) mortality 7 to 10 times higher than non-Indigenous Australians (age 25–64 years), a prevalence of type 2 diabetes mellitus (T2DM) 10 times higher (age 20–50 years), and a life expectancy that is 15 to 20 years shorter [3, 4]. Early mortality due to chronic disease (T2DM, chronic kidney disease [CKD], and CVD) accounts for almost half of the 17 years disparity in life expectancy of Indigenous Australians. Not all of the higher cardiovascular risk of Indigenous Australians is explained by traditional risk factors, suggesting the importance of consideration of non-traditional risk factors (such as inflammatory markers, markers of oxidative stress, homocysteine, albuminuria, and psychosocial stress) in this population.

Epidemiology of Type 2 Diabetes Mellitus among Indigenous Australians

Compared to the overall Australian population (which is predominantly of Caucasian ancestry as well as a mixture of Southern Europeans, Chinese, Vietnamese, Asian Indians, and South Asians), Indigenous Australians have a significantly higher prevalence of T2DM, most markedly in young adults. Indigenous Australians have a rate of T2DM that is 10 times higher than the general population in the 20- to 50-year age group [4]. Recent prevalence rates of T2DM in Indigenous Australians range from 10% in a central Australian homelands population [5] to 26% in the Torres Strait [6]. However, due to the early age of onset of T2DM and the younger age profile of Indigenous populations, crude prevalence rates underestimate the magnitude of the problem. Aboriginal populations display a different disease profile with age compared to the Australian population. Specifically, T2DM prevalence increases continuously with age in the non-Indigenous population, but in the Aboriginal population the prevalence of T2DM increases until the sixth decade of life (age 50–60 years) and then either plateaus [7] or declines [8, 9]. The speculation is that the decline in T2DM prevalence in Aboriginal people over the age of 55 years may be due to either a "healthy survival" or a cohort effect whereby younger people have riskier behaviors than older people (especially in the most remote Indigenous communities). Importantly, the high prevalence rates of obesity, T2DM and related chronic diseases in Indigenous Australians are similar to the 20th century experience of other Indigenous populations that have experienced dramatic lifestyle changes and urbanization. Examples include Native Americans, especially Pima Indians [10], Native Canadians [11], Asian Indians [12], and Nauruans and other Pacific Islander inhabitants [13].

Epidemiology of Cardiovascular Disease among Indigenous Australians

For two recent time periods (1996 to 1998 and 2000 to 2002), the Australian Institute of Health and Welfare (AIHW) reported that Indigenous Australians were twice as likely to die from CVD than non-Indigenous Australians [3, 14]. In the young to middle-age adult population, the difference is even greater. CVD mortality is between 7 and 10 times higher for Indigenous Australians aged 25 to 64 years than non-Indigenous Australians [14].

Interestingly, the incidence of coronary heart disease events among Indigenous Australians is equally high in urban and remote regions [15, 16•]. Incidence of coronary heart disease events was 11.6/1,000 person-years for a cohort of 906 urban Aboriginal people from Perth and 11.0/1,000 person-years for a cohort of 867 Aboriginal people from a remote community in the Northern Territory [15, 16•]. In contrast, disease burden from non-communicable diseases is higher in remote than urban-dwelling Indigenous Australians. Although 26% of Indigenous Australians live in a remote location, this group of people bear 40% of the burden of disease of Indigenous Australians (39% for cardiovascular disorders and 38% for diabetes) [17•]. Thus, the burden of disease is disproportionately greater for remote than for urban-dwelling Indigenous Australians.

A cohort study of Indigenous and non-Indigenous acute coronary syndrome patients from Northern Territory, Australia (CASPA Study) reported their preliminary findings: Indigenous patients were younger but had more comorbid chronic conditions than non-Indigenous patients. In addition, they found that Indigenous patients had longer delay times from the onset of symptoms to arrival in the emergency department. Interestingly both groups experienced similar in-hospital mortality, but Indigenous patients who did not undergo an invasive diagnostic or therapeutic intervention were approximately three times more likely to die during follow-up [18•]. Predictors of late mortality in this Indigenous Australian cohort were end-stage kidney disease, congestive heart failure prior to or during hospitalization, ethnicity, and the prescription of evidence-based therapy at discharge [18•].

Features of the Cardiovascular Risk Profile in Indigenous Australians

Many studies have demonstrated the high prevalence of traditional CVD risk factors in Indigenous Australians: cigarette smoking, hypertension, diabetes, central obesity, and hypercholesterolemia/dyslipidemia [5, 19–21]. Thompson et al. [21] described how 83% of a self-selected group (n= 738) of the Perth Aboriginal community fell within the "high risk" or "highest risk" National Heart Foundation of Australia (NHFA) categories for CVD. Those participants aged 25 to 64 years represented approximately 20% of the Perth Aboriginal population in that age group. Nine percent of the group were in the NHFA highest-risk category (known vascular disease) and 74% regarded as "high risk" (at least 1 coronary risk factor) [21].

Cigarette smoking

Rates of cigarette smoking are significantly greater in Indigenous Australians than the general Australian population. In fact and unfortunately, 50% of Indigenous Australians throughout Australia are reported to be smokers. Furthermore, the prevalence of smoking in the remote communities in the Top End of the Northern Territory may be as high as 70% [5, 20, 22, 23]. In all but one of these studies, smoking rates were self-reported; however, in one key study urine cotinine levels confirmed high levels of smoking [22]. Quantitative levels of cigarette smoking have not been otherwise reported. Cigarettes are brought into these remote areas by road or sea transport as with other general freight.

Central obesity

There is a high prevalence of central obesity in Indigenous Australians [5, 24]. Furthermore, the prevalence of overall obesity continues to be high even with the introduction of health promotion and weight loss programs [24, 25]. Of particular concern is the high and rising rate of central obesity in young Indigenous women (ages 15–34 years) [26]. Both overweight and obesity (specifically central obesity) are closely related to the high prevalence of T2DM [27]. Indigenous Australians have greater central obesity than Australians of European background. Piers et al. [28] demonstrated that when compared to Australians of European background, Aboriginal men and women men had both greater waist-hip ratios and more abdominal fat for any given weight or body mass index. Hence, waist-hip ratio is the preferred measure of central obesity in Indigenous Australians and this is consistent with observations made in the INTERHEART Study [29]. The combination of high central fat and low peripheral (hip) adiposity may play an important role in the cardiometabolic risk experienced by Indigenous Australians [30•, 31].

Dyslipidemia

Hypertriglyceridemia, often associated with low high-density lipoprotein (HDL) cholesterol, is the most common lipid abnormality observed in Indigenous Australians. Importantly, high triglycerides and low HDL cholesterol (a scenario that is often referred to as the dyslipidemia of insulin resistance) are two of the five features that define the metabolic syndrome [4, 19, 32•, 33]. Other dyslipidemic characteristics commonly observed in Indigenous Australians are small low-density lipoprotein (LDL) particle size, which is equally severe in men and women [32•]. When compared to the general Australian population (as reported in the Australian Diabetes, Obesity and Lifestyle [AusDiab] study), Aboriginal and Torres Strait Islander people have a lower prevalence of hypercholesterolemia but a higher prevalence of dyslipidemia (high triglycerides and low HDL cholesterol) [5, 32•]. Aboriginal populations have even more pronounced dyslipidemia than Torres Strait Islanders [4, 32•].

Non-traditional risk factors

Traditional CVD risk factors do not explain all of the increased CVD risk in Indigenous Australians. In one remote Aboriginal community in the Northern Territory, the Framingham Risk Score, particularly in younger women, significantly underestimated the risk of coronary heart disease [15]. To explore this discrepancy, non-traditional CVD risk factors have recently been included in assessments of CVD risk in Indigenous Australians. These non-traditional risk factors include inflammatory markers, markers of oxidative stress, homocysteine, albuminuria, and evaluation of psychosocial stress. The relative contribution of non-traditional CVD risk factors to the pathogenesis of CVD remains unclear, but the INTERHEART Study, which includes 52 countries worldwide, found a combination of certain modifiable risks for acute myocardial infarction accounted for more than 90% of the risk in all populations in both sexes at all ages. These risk factors were smoking, hypertension, T2DM, dyslipidemia, central obesity, psychosocial stress, a lack of fruit and vegetables in the diet, alcohol consumption, and a sedentary life style [34]. Most significantly, an adverse lipid profile, smoking, and psychosocial factors were the most important risks [34]. In the INTERHEART study, an "adverse lipid profile" was defined by a high ratio of apolipoprotein B to apolipoprotein A1. A high apolipoprotein B to apolipoprotein A1 ratio is a proxy measure for both high LDL cholesterol and low HDL cholesterol. Both of these lipid

characteristics are key factors in the development of coronary heart disease.

Albuminuria is another potentially important marker of CVD risk, and macroalbuminuria was found to be the strongest predictor of cardiovascular mortality in a longitudinal study from an Indigenous community in Northern Australia with high rates of renal disease [20]. Both microalbuminuria and macroalbuminuria (based on a spot urine albumin creatinine ratio and microalbuminuria 3.4–33.9 g/mol, macroalbuminuria >34 g/mol) were important predictors of coronary events in this cohort [35]. High rates of albuminuria have been reported in many remote Aboriginal communities. This is important because high levels of albuminuria are closely related to other components of the metabolic syndrome [36].

Inflammatory markers, such as C-reactive protein (CRP), are other potentially useful CVD risk markers, and very high levels of vascular inflammation and endothelial activation have been described in Indigenous Australians [37, 38]. High CRP (≥3 mg/L) was reported as an independent predictor of incidence of CVD events (hazard ratio 2.40; 95% CI, 1.25–4.62) in a remote Aboriginal community of the Northern Territory [39•].

Clustering of Cardiovascular Risk Factors

An investigation of carotid intima-media thickness (CIMT) comparing both urban and remote Indigenous Australians to Australians of European background reported that the increased cardiovascular risk in Indigenous Australians may be explained by the clustering of both traditional and non-traditional risk factors [40]. In this study, traditional cardiovascular risk factors (age, male gender, hypertension, and hyperglycemia) were important contributors to CIMT in each of the three population groups. Components of the metabolic syndrome (central obesity, dyslipidemia) together with cigarette smoking, albuminuria, and CRP clustered with "Indigenous participant" on factor analysis; thus, the study proposed that together with traditional vascular risk factors, these non-traditional factors occur together and contribute to increased CIMT [40].

Social and Environmental Contributors

In an investigation in the Darwin region of Australia, it was reported that the rates of T2DM increased with declining income and socio-economic status in urban Indigenous Australians [41]. Darwin, the capital of the Northern Territory, is the northern-most capital city in Australia, and Indigenous people represent approximately 29% of the Northern Territory population and 10% of the Darwin Region population. A striking picture has been reported for end-stage kidney disease (ESKD) among Indigenous Australians, where a 20-fold gradient between non-Indigenous and Indigenous Australians was reported by Cass et al. [42, 43]; rates of ESKD were highest in the most remote and socio-economically disadvantaged regions of Australia. Kidney disease among Indigenous Australians is discussed further in the following text.

Developmental Origins

It is important to consider potential inter-generational effects among the high-risk Indigenous Australian population. With rates of obesity and T2DM increasing in women of child-bearing age, so do rates of gestational diabetes and pre-existing T2DM in pregnancy, with adverse effects on the offspring, particularly if glucose during pregnancy is not tightly controlled [44]. A follow-up study of Pima Indian children born to mothers with and without T2DM found higher rates of obesity, T2DM, and systolic hypertension (independent of adiposity) in the infants of mothers with diabetes during their pregnancy [45, 46]. However, in studies of both Pima Indians and native Canadians, it was found that breastfeeding appeared to minimize the risk of early-onset obesity and T2DM in offspring of mothers with diabetes in pregnancy [47, 48].

Table 1 Proportions of indigenous participants with T2DM meeting therapeutic targets

Therapeutic target	DRUID study [55]	ABCDE project [56]
HbA1c <7%	29%	27% (range 0–55%)
BP <130/80 mm Hg	45%	36% (range 0–59%)
Total cholesterol <5.5 mmol/L	65%	79% (33–100%)
Total cholesterol <4.0 mmol/L	–	29% (0–71%)
Triglycerides <2 mmol/L	43%	–
HDL cholesterol >1 mmol/L	52%	–
Urine albumin creatinine ratio <3.4 mg/mmol	61%	38% (0-86%)

ABCDE Audit and Best Practice for Chronic Disease Extension; *BP* blood pressure; *DRUID* Darwin Region Urban Indigenous Diabetes; *HbA1c* glycated hemoglobin; *HDL* high-density lipoprotein; *T2DM* type 2 diabetes mellitus.

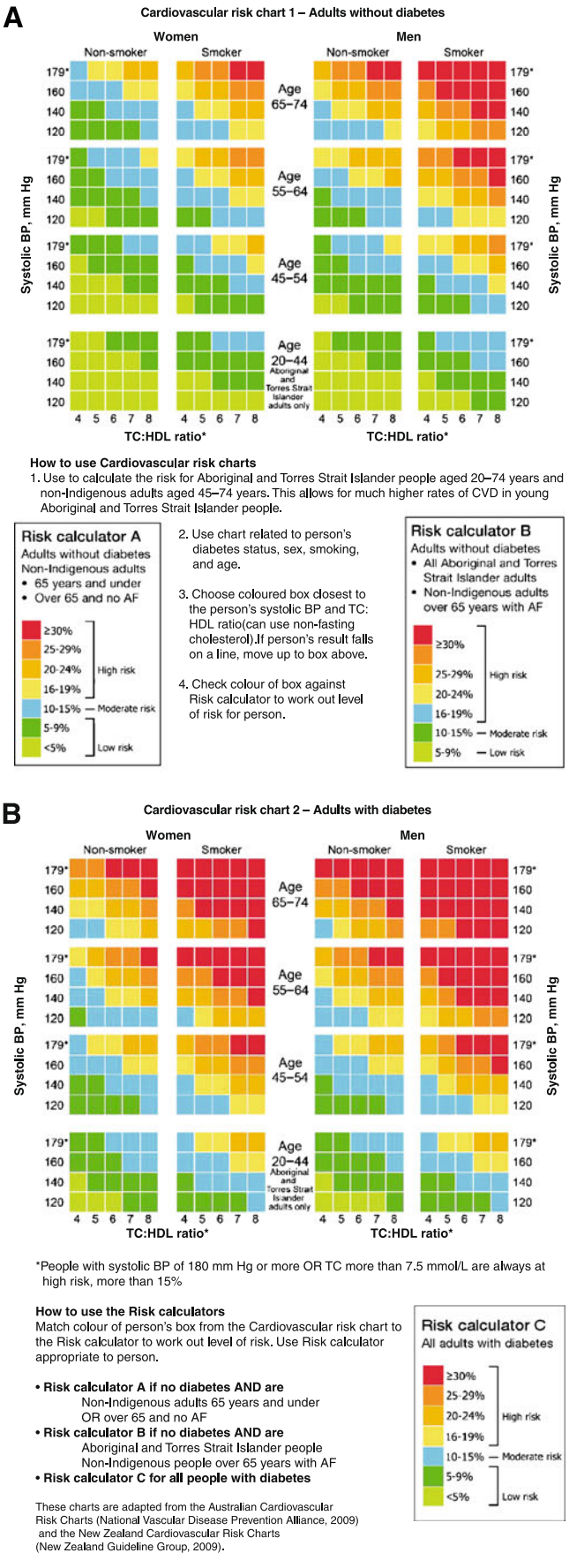

Fig. 1 Cardiovascular risk calculator. *AF* atrial fibrillation; *BP* blood pressure; *HDL* high-density lipoprotein; *TC* total cholesterol. (*Reproduced from* the Central Australian Rural Practitioners Association [CARPA]; with permission)

Overlap between Type 2 Diabetes Mellitus, Cardiovascular Disease, and Kidney Disease

Rates of both diabetes and CVD are high at a young age among Indigenous Australians. There is significant overlap between these chronic conditions and they share many common risk factors [49]. Chronic kidney disease and ESKD also cluster with the chronic conditions of diabetes and CVD. T2DM is common among Indigenous Australians with ESKD: 77% of Indigenous Australians with new ESKD in 2007 had T2DM as a co-morbidity compared to 33% of non-Indigenous ESKD Australians [50]. Rates of ESKD among Indigenous Australians vary by region and are greatest in the most remote regions of Australia, where rates of ESKD are up to 20 times that of the general Australian population [42]. In general, rates of ESKD resulting in renal replacement therapy are 10 times greater in Indigenous than non-Indigenous Australians (with adjustment for age). Rates of those who do not receive renal replacement therapy are not known. Access to renal replacement therapy frequently involves patients relocating permanently or travelling long distances, although dialysis units have recently been constructed in remote locations to help improve some of these barriers to services [51]. Rates of renal transplantation are also considerably lower among Indigenous than non-indigenous Australians, with both lower transplant and patient survival [51].

Not only do the chronic conditions of diabetes, CVD, and ESKD share risk factors (poor diet, cigarette smoking, low birth weight, central obesity, abnormal lipid profile, albuminuria, hypertension), but they interact to amplify mortality risk in this population. It is both the clustering of risk factors and conditions (of which diabetes is one) that impact on prematurely high cardiovascular risk among Indigenous Australians.

There is inadequate longitudinal data on the association between T2DM and CVD in Indigenous Australians, yet it has been reported in one remote Indigenous community in the Top End of the Northern Territory where Wang and Hoy [52] performed a 9- to 10-year follow-up of 889 Aboriginal people aged 20 to 74 years. After adjustment for coronary heart disease risk factors, diabetic women had a coronary heart disease rate ratio (defined as the number of first-ever coronary heart disease events divided by the person-years of follow-up) of 3.7 (95% CI, 1.6–8.9 compared to women without diabetes), whereas the rate ratio for diabetic men was 1.4 (95% CI, 0.4–4.1 compared to men without diabetes). Although the difference was not statistically significant (perhaps related to small sample size), women

with diabetes experienced a higher coronary heart disease risk than men with diabetes. This differs from findings in other (non-Indigenous) populations, where risk of coronary heart disease is similar in men and women with diabetes (ie, diabetes removes the protection afforded to women in relation to coronary heart disease risk) [53].

Clinical Management of Type 2 Diabetes Mellitus and Cardiovascular Disease Risk

Studies of management of T2DM among Indigenous Australians suggest higher rates of screening for diabetes but gaps in care and management of diabetes similar to that seen in the general Australian population [54–56]. Among 99 urban Indigenous Australian participants of the Darwin Region Urban Indigenous Diabetes Study (DRUID) known to have T2DM prior to enrollment, rates of participants who met therapeutic targets are outlined in Table 1. In addition, 60% of this group of participants with microalbuminuria and 90% with macroalbuminuria were treated with either an angiotensin-converting enzyme inhibitor or angiotensin-2 receptor antagonist. Current diabetes care for those participants known to have T2DM was provided by a general practitioner (74%), diabetes/medical specialist (8%), nurse or health worker (3%), other (2%), other health care professional (13%) [55]. A large clinical medical audit study across 62 Aboriginal community controlled health centers from four states or territories of Australia (Audit and Best-practice for Chronic Disease Extension [ABCDE] project) revealed similar rates of participants meeting therapeutic targets to those outlined above (Table 1), although there was a very wide variation across different centers [56]. Patient level characteristics accounted for 64% to 97% of the total variation of measures of diabetes care, whereas health center level factors accounted for 36% of the variation in adherence to scheduled services but for only 3% to 11% of variation in intermediate patient outcomes [56]. When compared to results of the general Australian population as reported by the AusDiab study, these rates of participants with known T2DM meeting therapeutic targets are better for blood pressure and lipid control but worse for glycemic control [55].

Relevant local and national guidelines for assessment of CVD risk in Indigenous Australians are published by the National Heart Foundation of Australia [57] as well as the Central Australian Rural Practitioners Association (CARPA) [58]. Both these guidelines recommend assessment of absolute risk through use of a specific cardiovascular risk chart and calculator (Fig. 1). A recent review of 1,165 randomly selected case records of Indigenous Australian adults aged 18 years and over from eight primary health care settings across regions of Australia revealed significant gaps in CVD risk management [59]. Key findings were that 53% of the sample were not adequately screened for CVD risk according to national guidelines; 40% of those with CVD were not prescribed a combination of blood pressure medications, statins, and antiplatelet agents; and 56% of individuals without CVD but categorized as having high CVD risk (according to National Heart Foundation of Australia Risk Calculator) were not prescribed blood pressure medications and statins. The authors conclude that these management gaps are "similar to those described in non-Indigenous health care settings, suggesting deficiencies across the health system" [59].

Conclusions

Indigenous Australians experience high rates of diabetes and CVD with onset at a younger age than the general Australian population. Population-based prevention measures are urgently needed from a young age in order to reduce cardiovascular risk in this high-risk population. Interventions to reduce risk will need to occur within the health system as well as in other sectors such as education, employment, and nutrition and provided through both governmental and non-governmental agencies.

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