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Review Article

Sleep influences on cardio-metabolic health in Indigenous populations

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ABSTRACT

Indigenous populations continue to be among the world's most marginalized population groups. Studies in Indigenous populations from high income countries (including the United States, Canada, Australia, and New Zealand) indicate increased risk of sleep disorders compared to non-Indigenous populations. Poor sleep, whether it be short sleep duration or fragmented sleep, is a well-established risk factor for cardio-metabolic diseases. Given the implications, targeted improvement of poor sleep may be beneficial for the health and well-being of Indigenous people. In this narrative review, we will: (1) discuss the effects of sleep on the cardio-metabolic processes; (2) examine sleep in Indigenous populations; (3) review the association between sleep and cardio-metabolic risk in Indigenous populations; and (4) review the potential role of sleep in cardiovascular disease risk detection and interventions to improve sleep and cardio-metabolic health in Indigenous people. In particular, this review highlights that the assessment of sleep quality and quantity may be a beneficial step toward identifying Indigenous people at risk of cardio-metabolic diseases and may represent a key intervention target to improve cardio-metabolic outcomes.

1. Introduction

There is a large inequity in health in Indigenous populations globally who continue to be among the world's most marginalized population groups [1]. A gap in life expectancy of up to 12 years exists between Indigenous and non-Indigenous people [2,3]. In high income countries, such as the United States, Australia, and New Zealand, high risk of cardio-metabolic diseases is thought to be a major contributor to this gap [2,4]. Accordingly, there is a concerted effort by the World Health Organization to improve health outcomes across Indigenous populations to reduce the current inequity that exists. To achieve this goal, new and novel targets are needed to identify individuals at high risk and improve health outcomes. Poor sleep, whether it be short sleep duration or fragmented sleep, is associated with higher risk for metabolic syndrome [5,6], mental health problems [7–9], and cardiovascular disease (CVD) [10–13]. The complex interplay between these factors is highlighted by the fact that poor sleep [14] and mental health [15] are known predictors of CVD risk [16]. Indigenous populations in high income countries are known to have poor sleep quality compared to non-Indigenous populations [17]. Therefore, sleep may be a key, yet overlooked factor for poor metabolic health and CVD risk in Indigenous populations. Indigenous people living in high income countries such as the United States, Canada, Australia, and New Zealand share similar historical experiences, socio-economic disadvantage, and health status and are often considered natural comparators in Indigenous health literature [18]. These Indigenous populations also have earlier onset of chronic conditions such as diabetes and CVD when compared to the non-Indigenous population groups [19].

This review will largely focus on individuals from these four countries and will: (1) discuss the effects of sleep on the cardio-metabolic processes; (2) examine sleep in Indigenous populations; (3) review the association between sleep and cardio-metabolic risk in Indigenous populations; and (4) review the potential role of sleep in CVD risk detection and interventions to improve sleep and cardio-metabolic health in Indigenous people. Given the broad scope of this review, limited published information, and variable methodology and quality of relevant studies, this

Abbreviations: CVD, cardiovascular disease; NREM, non-rapid eye movement; N1, NREM stage 1 sleep; N2, NREM stage 2 sleep; N3, NREM Stage 3 sleep; OSA, obstructive sleep apnea; PSG, polysomnography; REM, rapid eye movement.

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is a narrative review rather than a structured and systematic review of the available literature. Fig. 1 illustrates the overall themes of the review, linking poor sleep to both cardio-metabolic risk, mental health problems, and CVD risk. Similar to the model described by Grandner et al., which details a social–ecological model of poor sleep and its adverse effects on health outcomes, sleep in Indigenous populations is influenced by a complex array of determinants with unfavorable downstream effects on cardiovascular and metabolic disease risk [20].

2. Sleep and the cardiovascular system

Obtaining adequate sleep (a sufficient amount of sleep of acceptable quality) is imperative to sustaining optimal daytime functioning and health [21]. Both experimental and observational studies show that sleep has a marked effect on cardiovascular and endocrine systems and that disrupted sleep has adverse effects on these systems.

In order to understand sleep and its effect on the cardiovascular system, a brief understanding of sleep state architecture is crucial. In adults, sleep is divided into two distinct states known as rapid eye movement (REM) and non-rapid eye movement (NREM) sleep [21]. NREM sleep is further divided into sleep stages N1, N2, and N3. REM and NREM sleep are physiologically distinct and have a marked effect on both the cardiovascular and respiratory systems, in that blood pressure, heart rate, and respiratory rate are lower and less variable in NREM sleep compared to REM sleep [22]. Thus, sleep is not just a static state, it is associated with complex changes in cardiovascular and respiratory control, which depend on both sleep state, and the time spent in the sleep period [22]. Accordingly, during sleep, shifts in sympathetic and parasympathetic dominance occur [22], whereby sympathetic activity decreases during the night and parasympathetic activity increases [23]. In addition, autonomic control is sleep state dependent, whereby REM sleep is dominated by the sympathetic nervous system and NREM by the parasympathetic nervous system [22]. Notably, during the night, sleep onset sees a rapid decline in blood pressure reaching a nocturnal nadir within the first sleep cycle, followed by a gradual rise in blood pressure toward wakefulness levels for the remaining sleep period [24]. This nocturnal decline in blood pressure, termed “dipping,” is thought to serve as a physiological restorative process of sleep via a reduction in the hemodynamic burden to the heart and vasculature [25].

In summary, sleep has a marked effect on the control of both heart rate and blood pressure and associated parasympathetic and sympathetic activity. How this may in turn provide a link between sleep, hypertension, and cardio-metabolic risk will be outlined below.

3. Sleep and its link to cardio-metabolic risk

The clustering of several risk factors such as obesity, high blood pressure, and abnormal levels of blood glucose and lipids (termed cardio-metabolic risk) increases the risk of both type 2 diabetes and

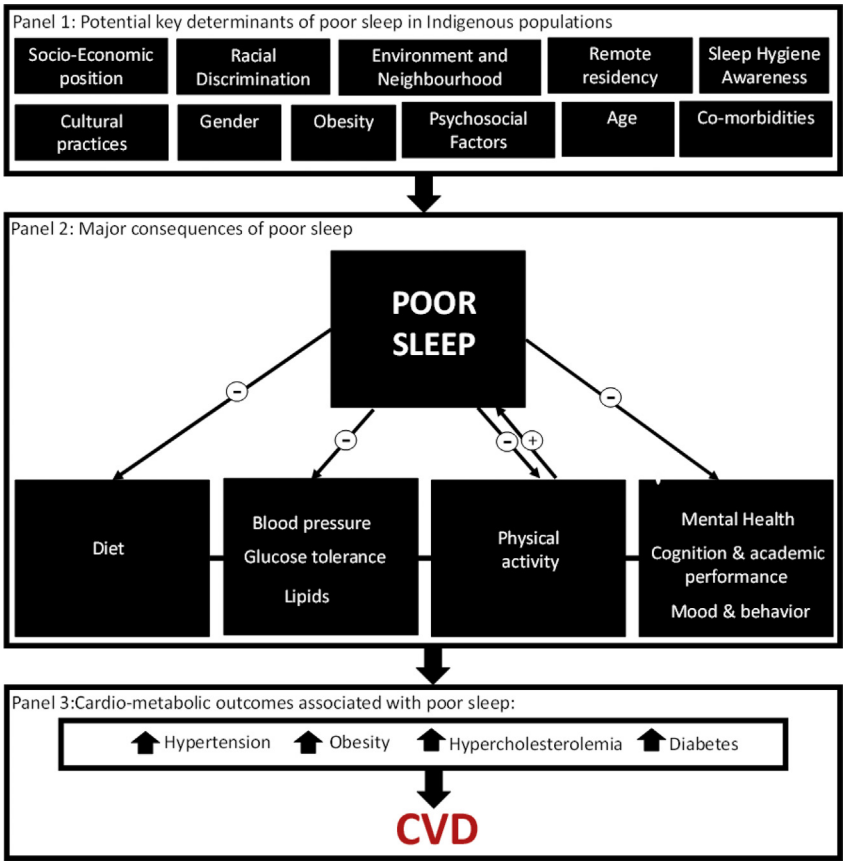

Fig. 1. The figure outlines the central theme of the review: a complex array of determinants, consequences, and cardio-metabolic outcomes of sleep. Panel 1: outlines the potential determinants leading to poor sleep in Indigenous populations. Panel 2: illustrates the negative consequenes as sleep on cardio-metabolic factors, dite, physical activity, and brain functioning. These consequences as well as their interactions are known to increase cardio-metabolic risk. Panel 3: The known outcomes associated with poor sleep forming a cluster of cardio-metabolic risk factors that predispose and individual to CVD. Together this schema highlights that poor sleep can yield a powerful influence not only on cardio-metabolic risk, CVD risk, but mental health, and education outcomes.

CVD. It has been well-established that short sleep duration has a negative impact on cardio-metabolic health with adverse outcomes such as increased risk of obesity [26], hypertension [27], hypercholesterolemia [27,28], diabetes, and coronary heart disease [14]. Studies have shown that sleep duration may have a U shaped effect on cardiovascular events, with adults sleeping <6 h and >10 h per night having increased risk of all − cause mortality and cardiovascular events [29]. Poor sleep quality is also known to increase the risk of hypertension [27]. For example, obstructive sleep apnea (OSA), which causes sleep fragmentation due to repetitive arousals with or without hypoxaemia, is associated with an increased risk of hypertension [31]. Hypoxic episodes that frequently occur in OSA are associated with a rise in blood pressure and sympathetic activity. In OSA, hypertension may result from both a response to hypoxia and sleep fragmentation, though the contributing features are difficult to disentangle [32]. We do know, however, that sleep fragmentation caused by factors other than hypoxia, has a negative impact on the cardiovascular system, such as fragmented sleep due to noise [33].

Studies have revealed that sleep duration is associated with increased risk of obesity [34]. The Wisconsin Sleep Cohort study (n = 1024) showed a U shaped association between objectively measured sleep duration and body mass index (BMI), with the nadir occurring at 7–8 h of sleep per night [35]. In a large longitudinal study (n = 9588), individuals who self-reported sleep duration less than 7 h per night had higher average BMI and an increased likelihood of obesity compared to those who slept more than 7 h per night.

Results of prospective cohort studies have also revealed that both short sleep (of generally <6 h per day) and poor sleep quality are associated with an increased risk of incident diabetes [36,37]. These findings are consistent with a recent systematic review and meta-analysis investigating sleep disturbances compared to traditional risk factors for diabetes development showing that pooled risk ratios for diabetes development and sleeping >5 h, 6 h, and >9 h/d were 1.48, 1.18, and 1.36, respectively [38]. Of note, this meta-analysis showed that OSA and difficulty initiating and maintaining sleep had the largest estimated impact on diabetes risk [38], with effect sizes only slightly smaller than having a family history of diabetes or being overweight, but larger than being physically inactive [38].

In addition to the direct association between sleep disturbances and poor cardio-metabolic outcomes, the contribution of psychological factors which may impact both sleep and cardio-metabolic risk cannot be overlooked. Mental health disorders are associated with an increased risk of metabolic syndrome [40] and higher rates of overweight and obesity [41]. Sleep plays an important role in emotional regulation [42] and nearly all mood and anxiety disorders co-occur with one or more sleep abnormalities [43]. Poor sleep is associated with depression, anxiety, aggression, and delinquent behaviors [7–9]. In adolescents, insufficient sleep is associated with poor academic achievement and contributes to problems with emotional–behavioral regulation and, as a consequence, to a range of potentially self-harming and other-harming behaviors, such as drug use, risky driving, hyperactivity, and aggression [44]. The relationship between poor sleep, mental health problems, and cardio-metabolic risk is highlighted in conditions where these factors co-occur. Insomnia, a sleep disorder which is highly comorbid with psychiatric disorders (depression, anxiety, and suicide) [45] is known to also be associated with high cardio-metabolic risk [39]. In particular, insomnia with short sleep duration is associated with high risk of hypertension, diabetes, and mortality.

3.1. Poor sleep and increased cardio-metabolic risk: potential mechanisms

A number of mechanisms underlying the association between poor sleep and increased cardio-metabolic risk have been proposed. Sympathetic overactivity, related to poor sleep quantity and quality is thought to play a major role in the development of hypertension [46,47]. Short sleep duration is associated with alterations in the hypothalamic–pituitary–adrenal axis [48]. Sleep deprivation is associated with increased blood pressure [49,50] and urinary excretion of norepinephrine [46], which suggests an increase in sympathetic nervous system activity. As a consequence, both short and disrupted sleep impairs nocturnal dipping, thought to be a major restorative process for the cardiovascular system [51,52]. Non-dipping is a clinical feature of pathologies where significant sleep disruption occurs, such as OSA [53]. Notably, non-dipping is a risk factor for left ventricular hypertrophy and CVD [25,30].

Sleep restriction has negative impacts on metabolism and endocrine function. Both sleep restriction and disturbed sleep reduce insulin sensitivity [6], the acute response to glucose, and increase evening cortisol concentrations resulting from elevated hypothalamic–pituitary–adrenal axis activity, all predisposing an individual to type 2 diabetes [54]. In addition, circadian misalignment [55] and OSA are associated with low insulin sensitivity or insulin resistance and increased diabetes risk [56].

Poor sleep has also been linked to obesity via disruption in energy balance [57]. In addition to the known effect sleep disruption has on insulin sensitivity, altered energy balance is thought to be mediated by changes in appetite and satiety hormones that occur as a result of inadequate sleep. Reduced sleep duration has been associated with decreased leptin (a hormone that regulates energy stores and satiety) and elevated ghrelin levels (a hormone that enhances appetite) [57]. Potential mechanisms involved in altered leptin levels include upregulation of central orexin activity (a peptide which promotes feeding and is closely coupled with arousal maintenance). Elevated orexin activity promotes sympathetic activation of appetite stimulating neuropeptide Y neurons, and in the periphery, a reduction in leptin release as well as total body insulin sensitivity [58]. Altered appetite regulation results in greater energy intake and storage in adipocytes with concomitant reduction in energy utilization [59]. These factors, in addition to decreased daytime physical activity and energy expenditure due to sleepiness, and increased waking hours available for eating, are thought to underlie the association between poor sleep, weight gain, and obesity.

Another potential mechanism by which poor sleep exerts negative effects on metabolic and cardiovascular health may be through alteration of inflammatory processes during sleep [60]. Sleep restriction can increase production of interleukin-17 together with C-reactive protein, two known inflammatory markers associated with CVD risk [61,62].

In regards to the inter-relationship between sleep, mental health, and cardio-metabolic risk, stress is a likely important mechanism. This relationship is neatly modelled in insomnia patients where the joint effect of stress and psychological factors are strongly emphasized in the pathogenesis of the disorder [63]. Stress is associated with the activation of the hypothalamic–pituitary–adrenal and the sympatho-adrenal medullary axes, where corticotrophin releasing hormone, cortisol, and catecholamines are known to cause arousal and sleeplessness [63]. Using objective measures of sleep, studies show that cortisol levels, norepinephrine, and catecholamine metabolites levels are all

elevated in insomnia patients compared to controls [64]. These alterations in autonomic and neuroendocrine pathways are thought to lead to increased cardio-metabolic risk.

Although the mechanisms that link poor sleep and cardio-metabolic risk are not fully understood, the clear adverse effect of sleep disruption on daytime functioning represents the beneficial role of sleep. In the context of Indigenous populations who suffer disproportionately from sleep problems when compared to non-Indigenous people [65–67], the necessity to assess sleep and its interactions with cardio-metabolic risk factors may be important for improvement of health. Below we discuss the disparity in health between Indigenous and non-Indigenous people, increased risk of cardio-metabolic disease, and the potential role of poor sleep in these populations.

4. Indigenous health and cardio-metabolic risk

4.1. The gap in cardiovascular health

The life expectancy gap between Indigenous and non-Indigenous peoples in affluent countries is up to 12 years [3,68]. Chronic non-communicable conditions such as diabetes, cardiovascular, kidney, and respiratory diseases represent almost 70% of the mortality in these populations [68]. In high income countries, CVD is also the major contributor to disability [69] and is considered the primary influencing factor in the life expectancy discrepancy between Indigenous and non-Indigenous populations [4]. High cardio-metabolic risk is thought to be the driver of CVD in these populations. It is important to note, steps to address the issue have been initiated with the recent International Delphi study which aimed to establish a consensus opinion on appropriate strategies for preventing cardio-metabolic diseases in Indigenous populations [70].

4.2. The context of Indigenous health

Collectively, disparities in Indigenous health are shaped by social inequality, historical trauma, and discrimination [71,72]. European settlement has had a dramatic effect on the lifestyles of Indigenous people around the world. Within a historical context, colonization has had a devastating impact on Indigenous people bringing about the introduction of diseases, the dispossession of land and livelihood, warfare, and the suppression of ancient cultures [73]. These conditions translated into subsequent poverty and imposed an inequity between Indigenous and non-Indigenous people. During early European colonization, the same benefits of healthcare, education, and employment were not afforded to Indigenous people in a culture dominated by Western society [74] and Indigenous people had little power to influence public policy decisions that affect their lives and health [75]. In modern times, perpetuation of this inequality is thought largely to be the product of contemporary structural and social factors. Indigenous people currently experience disadvantage in education, employment, income, housing, access to services, social networks, connection with land, racism, and incarceration [76]. Subsequently, health inequity is amplified in communities that have been dominated by the pitfalls of Western culture (eg, alcoholism and drug use) [77]. These factors are known to impact health in individuals, communities, and societies. Thus, in the context of cardio-metabolic health, changes in traditional ways of life, environment, and livelihoods have negatively impacted levels of physical activity and nutrition and are thought to play a key role in the development of CVD and diabetes in Indigenous people [78]. Moreover, psychosocial issues, social, and emotional well-being all contribute to the higher prevalence of cardio-metabolic diseases in this vulnerable population group [78].

4.3. United States and Canada

American Indian and Alaska Native peoples compose 1.7% of the population of the United States [79,80]. CVD is the number one killer of American Indians and Alaskan Natives, which reflect 5.2 million people of the United States population. This population group has a greater burden of major cardio-metabolic risk factors including hypertension, dyslipidaemia, obesity, diabetes, and smoking [81]. Mortality rates for Alaska Native populations are 60% higher than those of the United States white population and mortality rates in American Indian populations are about twice that of the general United States population [82].

The Aboriginal people of Canada – First Nations, Inuit, and Metis represent about 4% of the total population and they are 10.5 times more likely to die from coronary heart disease [83]. The rate of diabetes is three to five times higher among the First Nations on reserves when compared to general Canadian population [84].

4.4. New Zealand and Australia

In New Zealand, the Indigenous people (Maori) comprise 15% of the total population. Maori are overrepresented in the lower social economic status groups, are more likely to smoke, and more likely to be obese [85]. Despite improvements, major disparities between Maori and non-Maori exist across all health indicators including hypertension, heart disease, and cancer [86]. CVD incidence begins to rise after age 35 years among all age groups, but the rise in incidence is greater among Māori people [87,88].

Indigenous Australian (Aboriginal and Torres Strait Islander) population represents 3.3% of the total Australian population [89]. Between Indigenous and non-Indigenous Australians, the life expectancy gap is around 10–11 years and approximately one-quarter of this gap is attributable to CVD [90]. Compared to non-Indigenous Australians, these populations are 1.3 more likely to have CVD and three times more likely to have a major coronary event, such as a heart attack [91].

4.5. Biomedical and lifestyle risk factors for CVD in Indigenous populations

The risk factors for CVD in Indigenous populations are complex and include a combination of historical, social, cultural, and economic factors in addition to the well-known traditional biomedical and lifestyle risk factors [78]. Though each risk factor is equally important, some are beyond the scope of this review and therefore we will focus primarily on biomedical and lifestyle risk factors. Traditional cardio-metabolic risk factors, in part, are a likely contributor to the greater burden of CVD borne by Indigenous people [92]. On top of these well-known medical risks, increased risk behaviors such as smoking, alcohol consumption, physical inactivity, and unhealthy diets remain prevalent among these populations [91,93]. However, traditional factors used to indicate future risk of CVD (eg, age, sex, total cholesterol, high-density lipoprotein, blood pressure, diabetes, and smoking) do not provide equivalent predictive utility in Indigenous compared to non-Indigenous people [93–95]. For example, prediction functions (based on classic risk factor prevalence and event rates) used to estimate the incidence of CVD in a community sample of Indigenous Australians only accounted for 50% of CVD cases [95]. Therefore, factors other than the well-known established risk factors need to be considered for CVD risk in Indigenous populations. Poor sleep has been proposed to play a role in this increased cardio-metabolic risk in Indigenous populations.

5. Sleep in Indigenous populations

Studies in Indigenous populations, consistent across the United States, Canada, Australia, and New Zealand, indicate increased risk of sleep disorders compared to non-Indigenous populations [96–99]. To date the majority of data collected in Indigenous populations have relied on subjective measures of sleep including self-report questionnaire on sleep quality, or validated sleep quality questionnaires such as the Pittsberg Sleep Quality Index or the Epworth Sleepiness Scale. Few studies have used objective measures such as gold standard polysomnography (PSG). PSG, accurately detects sleep state architecture, sleep quantity and quality, and diagnosis of sleep-disordered breathing, but can be expensive and relies on trained technicians to setup and analyse sleep recordings. Furthermore, PSG usually involves an overnight stay in a sleep clinic which is not always accessible to Indigenous people living in remote areas. While self-report data are useful and can be applied to large populations, there are discrepancies between these subjective measures compared to PSG [100]. Furthermore, validated sleep questionnaires are not always appropriate for Indigenous populations. For example, the Epworth Sleepiness Scale asks about driving a car, which is not always applicable, particularly for those living in remote areas, or those who are financially disadvantaged who do not own a car. Nonetheless, as detailed below, the data collected to date, either by subjective or objective means consistently show that Indigenous people suffer disproportionately from sleep problems compared to non-Indigenous people.

5.1. United States and Canada

In the Unites States, a national survey showed that Indigenous people exhibit higher incidence of insufficient sleep (defined as ≥14 days in the past 30 days of insufficient rest or sleep) compared to non-Hispanic white individuals [101]. In support of these findings, telephone survey data from 444,306 respondents from the Behavioral Risk Factor Surveillance System showed that age adjusted healthy sleep duration was lower in American Indians/Alaska Natives (59.6%) compared to non-Hispanic whites (66.8%), Hispanics (65.5%), and Asians (62.5%) [27]. These self-reported sleep differences between Indigenous and non-Indigenous people are also supported by objective measurement of sleep. Using PSG measures, Redline et al., studied sleep architecture in adults aged 37–92 in a sample of 2685 subjects and showed that American Indians had lighter sleep (ie, a higher percentage stage 1 than whites or blacks; a higher percentage stage 2 and lower percentage stage 3–4 than whites, blacks, Hispanic, or Asian Americans) relative to other ethnic groups [66]. In Canada, Indigenous people have a high prevalence of symptoms related to sleep disorders such as insomnia, restless leg syndrome, and occurrence of reported apneas [102].

5.2. New Zealand and Australia

In New Zealand, Māori suffer disproportionately from poor sleep with higher incidence of insomnia and OSA [97–99]. Using questionnaire data, Paine et al., showed that Māori people were more likely than non-Māori to report difficulty falling asleep, have three or more nocturnal awakenings and wake too early in the morning [88,98]. Daytime sleepiness is also more common among the Indigenous Māori population compared with non-Māori adults [103]. These sleep problems have an effect on health, with self-reported chronic sleep problems being associated with poor or fair self-rated health (adjusted OR 3.94, 95% CI 3.01–5.16), even after controlling for ethnicity, gender, age-group, and area of socio-economic deprivation [98]. Sleep problems have also been studied in Māori children and findings from questionnaire data indicated that they have significantly higher rates of habitual snoring compared to non-Māori children [104].

In Australia, studies on sleep in Indigenous people remain scant. A population based study of men and women aged >45 years (314 Indigenous Australians and 59,175 non-Indigenous Australians) showed that 35% Indigenous Australians reported sub-optimal sleep quantity (defined at sleeping <7 h per day or sleeping >9 h per day) compared to 21% in non-Indigenous Australians [105].

Sleep quantity and quality has also been assessed in Indigenous Australian children. Using objective assessment of sleep, a small study ($n = 21$) showed that Indigenous Australian children had relatively less sleep at night, went to bed at a later time on school nights, and showed relatively poor sleep efficiency and fragmentation compared with non-Indigenous urban Australian school-aged children [106]. These findings are consistent with studies using subjective sleep assessment.

Using questionnaire data, Blunden et al., reported that 50% of Indigenous children aged 7–9 ($n = 513$) had sub-optimal sleep patterns (ie, they had short sleep duration, long sleep duration, variable sleep duration, or were early risers) [107]. In addition, face–face interview in 1650 children, studied in the Torres Strait region in five Indigenous communities showed that children aged 0–17 years had 14% prevalence of snoring, 4% reported snoring, and 6% reported restless sleep [108].

5.3. Obstructive sleep apnea in Indigenous people

Indigenous population across high income countries have higher prevalence of OSA compared to non-Indigenous people. Previous studies have found that the American Indians were 1.7 times more likely to have an apnea hypopnea index (AHI) > 15/h than Caucasians [109]. New Zealand Maori are more likely than non-Maori to report OSA symptoms and are 4.3 times more likely to have an AHI > 15/h [65,98,103,110]. In Australia, Indigenous Australians are 1.8 times more likely to have OSA compared to non-Indigenous populations [67]. The high risk of OSA among Indigenous populations in high income countries may be related to high prevalence of risk factors for sleep disorders such as obesity, chronic diseases, craniofacial structure, experience of poor living conditions, and lower socio-economic status [17]. OSA has an impact on health and well-being and 5.3% of the risk of stroke, 1.1% of heart failure, and 3.6% of coronary heart disease, and 6.2% of depression can be attributed to OSA [111]. All these conditions tend to have a greater prevalence and impact on Indigenous populations and therefore in absolute terms, OSA is likely to have a far greater impact in this setting.

5.4. Potential determinants of poor sleep

There are multiple candidates that may underlie poor sleep in Indigenous populations. Remote residence (via poor access to sleep diagnostic services and treatments [102]), lower health self-efficacy, lack of awareness of sleep health, competing health priorities, environmental factors, such as limited or no access to electricity, and a crowded sleeping area to name a few [67]. However, few studies have specifically focused on the determinants of poor sleep in Indigenous populations. It has been previously suggested that determinants of poor health status in Indigenous people is caused by a complex combination of determinants such as historical, institutional, socio-cultural, economic, environmental, and lifestyle related factors as well as access to healthcare [70]. These determinants may be adapted to address sleep health disparities among Indigenous populations.

Jackson et al., [112] published a framework on the role of sleep and CVD disparities (namely racial/ethnic and socio-economic inequities) that can be translated to the Indigenous context. This model is based on the notion that sleep is influenced by a complex and dynamic interplay between the individual and their physical, social, and institutional environments across the lifespan. In this conceptualized framework, sleep has: (1) distal influences: social conditions and policies (eg, socio-economic status and public policy) and institutional contexts (eg, occupational patterns and healthcare systems); (2) intermediary influences: social contexts (eg, racial discrimination and racial ethnic integration), social relationships (eg, family and social networks), and physical contexts (eg, building quality and neighborhood stability); and (3) proximal influences: individual demographics (eg, age and health status), individual risk behaviors (eg, smoking, alcohol use, and diet), biological responses (eg, obesity, previous illness, depression, and stress), and biologic/genetic pathways (eg, genetic mechanisms) [112].

Elements of the model described by Jackson et al. are supported by the few studies that have investigated determinants of poor sleep in Indigenous populations. In a study by Paine et al. investigating ethnic disparities in sleep disturbance, both socio-economic position and racial discrimination explained most of the disparity in difficulty falling asleep and frequent nocturnal awakening between Māori and Europeans [99]. Psychological distress may also influence poor sleep within Indigenous groups who experience higher rates of mental health problems [113–115] and depression [45]. Racial discrimination is also an important key determinant to consider with studies showing that discrimination is associated with sleeping difficulties in Indigenous children [71] and adults [99]. Furthermore, biomedical determinants may contribute to poor sleep in Indigenous people via the strong association between obesity and OSA [17].

Of note, these factors are not mutually exclusive and more work is needed in understanding the mechanisms underlying these associations [116]. Indeed, further research in general is required in understanding the determinants of poor sleep in Indigenous people. What is becoming increasingly clear is that poor sleep quantity and quality are common among Indigenous populations, whether measured subjectively or objectively. Sleep problems in Indigenous people are particularly concerning, as outlined above, optimal sleep is vital for cardio-metabolic health [88,117,118].

6. Sleep and cardio-metabolic risk in Indigenous populations

6.1. United States and Canada

Although there are few studies, the role of poor sleep in cardio-metabolic risk in Indigenous groups has been investigated. One population based study, the Native Elder Care study, assessed self-reported sleep and CVD in 449 American Indians >55 years [119]. Those who reported a shorter sleep duration of <5 h per night compared to 7 h per night had a higher likelihood of CVD (OR 2.89, 95% CI 1.17–7.16) [119]. In a more recent study of American Indian and Alaska Native people, diabetes risk and sleep duration were investigated [117]. Sleep duration was recorded by self-report in a sample of 1899 participants with pre-diabetes who completed a lifestyle intervention consisting of diet, exercise, and behavior modification sessions to promote weight loss [117]. At three-year follow-up, short sleep duration (<6 h per night) was associated with increased risk of diabetes incidence after adjustment for age, sex, socio-economic characteristics, health behaviors, and health status. Notably, across the three years of the study, those who had a shorter sleep duration lost significantly less weight than those who slept 7 h or more [117].

6.2. New Zealand and Australia

In New Zealand, associations between poor sleep and cardio-metabolic risk have also been identified. A large cohort study (n = 12,500) of Māori people ≥15 years of age showed a higher prevalence of self-reported sleep complaints with diagnosed high blood pressure, diabetes, heart disease, obesity, smoking, and hazardous drinking [88]. A study on heart failure and sleep-disordered breathing found that Māori or Pacific Islanders were overrepresented by more than twice the population ethnic prevalence and were twice as likely to have OSA or central sleep apnea and heart failure as compared with non-Indigenous New Zealanders [120].

To date in Australia, there have been no studies investigating the relationship between sleep and cardio-metabolic risk in Indigenous Australian adults. However, one planned study, the BIRCH (Better Indigenous Risk stratification for Cardiac Health) project [90] may provide essential information within this field of research. BIRCH is a cross-sectional and prospective cohort study of Indigenous and non-Indigenous Australian adults living in remote, regional, and urban locations. The project aims to identify and assess existing and novel markers of disease (including markers of sleep quality/disorders) and risk in Indigenous Australians. Assessment of sleep quality and sleep disorders will be carried out under the hypothesis that poor sleep may explain more of the actual risk of subsequent development of CVD in Indigenous Australian adults in comparison with non-Indigenous Australians and may aid in more accurate CVD risk prediction for Indigenous Australians.

In Indigenous Australian children, data from the Longitudinal Study of Indigenous Children (LSIC) survey of 1253 children aged 7–12 years has revealed that short sleep duration (assessed via primary carer report) was associated with unhealthy weight independent of age, socio-economic disadvantage, and level of remoteness [118].

6.3. Sleep, mental health, and cardio-metabolic risk in Indigenous populations

There are historical and contemporary factors such as dispossession, discrimination, and racism experienced by Indigenous populations that are known to impact mental health [121]. The burden of mental health disorders in Indigenous people is unacceptably high, with Indigenous people having higher rates of mental illness and psychological distress compared to non-Indigenous people in the United States [122], Canada [123], Australia [115], and New Zealand [114].

6.4. United States and Canada

As mentioned above, a clear association between poor sleep and mental health problems (including depression) has been established [7–9]. In a large population survey, the 2009–2010 Behavioral Risk Factor Surveillance Survey showed that mental distress contributed to the disparity in insufficient sleep in American Indian and Alaska Natives (n = 11,507) compared to non-Hispanic whites (n = 671,448) [101]. In Canada, using community-based study and validated questionnaires, Froese et al., identified that sleep-related symptoms (insomnia symptoms, witnessed nocturnal apneas, and restless leg symptoms) were independently associated with depression scores in 430 Indigenous North American people [102].

6.5. New Zealand and Australia

While an association between poor sleep and mental health has been established in Indigenous people, a limited number of studies

have explored how these conditions impact cardio-metabolic health. Of the few studies, data from a National Health Survey of 4108 Māori and 6261 Europeans showed that Māori people had a significantly higher amount of sleep disturbance and that differences in sleep disturbance were explained mostly by racial discrimination and socio-economic position [99]. Cross-sectional analysis of the survey revealed these sleep problems were associated with higher odds of mental health, diagnosed high blood pressure, diabetes, and obesity [88]. In Australia, data from a longitudinal study in 1239 Indigenous children indicated a high amount of racial discrimination which was associated with poor child mental health status, sleep difficulties, and obesity [71]. To date, these studies have not investigated the combined effects of poor sleep and mental health problems on cardio-metabolic risk and therefore the inter-relationships between these factors remain unknown in Indigenous people.

It is likely that psychophysiological stress may serve as an underlying mechanism linking mental health, poor sleep, and cardio-metabolic disparities. Significantly stress caused by issues such as substance abuse, suicide, and generational stress may all contribute to poor sleep [124,125]. In the American Indian adolescent population, where the suicide rate is high, Arnold et al., [125] investigated the relationship between sleep, depressive symptoms, and suicidality. In a sample of 80 American Indian youth aged 11–18, daytime sleepiness was associated with depression, but not suicidality [125]. In a community sample of 386 American Indian and Alaska Native people, Ehlers et al., showed that a greater level of poor sleep quality was associated with more substance use, anxiety, and affective disorders [124].

Taken together in Indigenous populations, prevalence of poor sleep and mental health problems are high compared to non-Indigenous people. In addition to the independent impact that these factors have on health and well-being, together these factors are all known to increase CVD risk and mortality. Intuitively, sleep should be considered in the risk detection, intervention, and treatment strategies for these major issues in health within this population. However, before such actions are considered, more rigorous research within this field is required, as many of the published studies to date have relied on self-report sleep assessment, rather than objective sleep measures such as PSG. In addition, few studies have looked at sleep and its association with comprehensive cardio-metabolic risk factor assessment. Accordingly there is room for improvement in delineating the association between sleep and cardio-metabolic risk in Indigenous people.

7. Implications and future sleep research in Indigenous populations

7.1. CVD risk detection

Early detection and management of risk factors for CVD are key in the primary and secondary prevention of disease with the ultimate aim of reducing both the incidence of new cases and lessen the severity and impact of established CVD in Indigenous populations. If indeed poor sleep is a contributor to cardio-metabolic disease risk, then sleep provides an easily measured function/behavior to identify at-risk individuals so that preventive treatments can be started sooner. To fully understand the impact of sleep quality and quantity on CVD risk in Indigenous populations, future studies should involve appropriate sleep and cardio-metabolic risk assessment. For example, improved cardio-metabolic risk profiles including anthropometric, blood pressure, and blood biochemistry (glucose and lipid levels) assessment in combination with objective sleep recording measurement would be beneficial.

Detailed studies in sleep and cardio-metabolic risk involving gold standard sleep assessment in Indigenous populations may be difficult. Woods et al., published data showing low adherence to follow-up PSG evaluation in a cohort of Indigenous Australians with diagnosed OSA [67]. Difficulties in remoteness, language, available transport, financial hardship, and accessibility of culturally appropriate healthcare services are likely to contribute to the barriers of sleep assessment in Indigenous populations [67]. A better approach may be to use simpler methods to study the sleep/wake cycle in this population. An alternative to PSG for assessing sleep–wake patterns is actigraphy [126]. Actigraphs are small movement detectors (accelerometers) that distinguish sleep from waking states using algorithms to quantify the reduced movement associated with sleep. Although these devices are unable to determine sleep state and stage with accuracy, actigraphy has advantages over conventional PSG in that the devices are small and can be used over extended periods of time [126]. Furthermore, actigraphy can record continuous high resolution physical activity during the daytime and thus provide an indication of sedentary behavior and its influences on sleep. Accounting for the association between daytime physical activity and sleep is important as acute and regular exercise can affect total sleep time, sleep onset latency, and slow wave sleep [127]. Moreover, physical activity may vary in accord with living conditions and environment; that is, Indigenous people living in very remote regions may lead different lifestyles to those in urban areas [128]. Use of actigraphy may provide a useful and cheaper option to quantify sleep in Indigenous people, which may overcome some of the current barriers to PSG sleep monitoring.

In regards to dection of sleep disordered breathing, in home-portable monitors that run on batteries and include measures of airflow, respiratory effort and oxygen saturation can provide relatively reliable measures of moderate to severe OSA [129]. Application of the portable monitors can be performed by a sleep technician or by the patient/participant after training. The use of abbreviated sleep measures, as a first step to understand the role of sleep in cardio-metabolic risk in Indigenous populations, has advantages. These abbreviated tools for sleep testing may be valuable, particularly for those living in regional and remote areas who (due to their geographical disadvantage) have limited access to health service providers and clinics. These tools allow assessment in the home and therefore do not require overnight stay in a clinic and do not require electricity to function. Furthermore, in communities where overcrowded housing is an issue, these smaller more transportable devices may be more adaptable to living environments. However, as this area of research grows it will be essential to determine whether or not this mode of sleep assessment will be the best method of sleep detection in health service delivery in Indigenous populations.

In addition to the improvement of methodological approaches, a better understanding of the relationship between sleep, mental health, and cardio-metabolic risk in Indigenous people would advance this research agenda. Furthermore, a detailed investigation of determinants of poor sleep in Indigenous people is essential. For example, understanding the influences of poor sleep at an individual, physical, and social levels (eg, demographic, cultural, environmental and genetic moderators of sleep, and cardio-metabolic risk) would provide important information to address sleep problems in Indigenous people. Such research would be highly relevant for the development of programs that could potentially target poor sleep for the improvement of cardio-metabolic risk.

7.2. Sleep intervention strategies for cardio-metabolic risk

Targeted, brief, sleep interventions can improve sleep problems in adults and children [60,130–133]. Improvement of sleep quality

in adults with chronically disturbed sleep can reduce biomarkers of diabetes and CVD risk with these effects persisting at one year follow-up [60]. In adults and adolescents, sleep intervention programs can improve sleep quantity and quality in at-risk populations as well as related anxiety and stress [131,132]. In children, behavioral intervention to improve sleep has been shown to resolve child sleep problems and improvements in psychosocial functioning [130]. Thus, behavioral intervention provides potential to improve poor sleep and well-being. To date, there are few studies that have implemented sleep interventions to determine the effects on cardio-metabolic risk in Indigenous populations. Though this area of research is in infancy, sleep is being recognized as a potential modifiable risk factor for intervention in Indigenous populations for cardio-metabolic abnormalities such as obesity. For example, The Healthy Children, Strong Families 2 study is a current lifestyle intervention trial for 450 American Indian children and their families [134]. The intervention trial is unique in that it includes both sleep and stress as modifiable risk factors in addition to improving diet, physical activity, and reducing sedentary time. Such intervention strategies including modification of all behaviors across the 24-h day/night period may prove to be more beneficial in reducing obesity risk, rather than focusing on daytime behavior only.

Improvement of sleep health in relation to cardio-metabolic risk should also take into consideration some of the key priorities for prevention strategies recommended for Indigenous cardio-metabolic health. For example, educational and behavioral changes should also address physical environmental determinants and be responsive to the local context, including being culturally appropriate [70]. Moreover, development of programs to improve sleep in Indigenous populations need to consider socio-economic and demographic factors, cultural differences, and preferences about sleep and sleep hygiene. Sleep profiles are likely to be impacted by cultural beliefs. For example, Indigenous Australians believe the concept of time is multidimensional rather than linear and unidimensional [135]. 'Dreamtime' is an important cultural belief which can have implications for the sleep behaviors in Indigenous Australians. Indigenous communities believe that the individual who enters the 'dreamtime' by 'dreaming' (which may occur during sleep), overcome the limitations of space and time and can access the strength and resources of the timeless. Thus it may be considered culturally inappropriate to control the sleep on a clock-based framework so as to prevent disrupting the dreamtimes. Given these considerations, the development of targeted intervention will need to involve engagement with community and their leaders to facilitate the process. Ultimately, understanding Indigenous sleep, how much sleep is a contributor to Indigenous well-being, and identifying determinants of poor sleep with and by the community will be vital in order to develop a targeted approach.

8. Conclusion

There is evidence to show that Indigenous people from high income countries have poor sleep quantity and quality. Poor sleep is associated with increased risk of cardio-metabolic conditions and CVD. Although studies are limited, observational studies show that poor sleep in Indigenous people is associated with poor cardio-metabolic health. To date, the majority of these studies have utilized self-report data. Future investigations should consider objective measures, such as actigraphy, as a simple assessment of sleep, particularly in remote communities. A focus on simple and acceptable interventions to enhance the quality and quantity of sleep is also recommended. Sleep may represent an under-recognized target for the improvement of cardio-metabolic risk and may be beneficial in identifying individuals at risk of CVD in Indigenous populations. Accordingly, future research is warranted in the area.

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Conflict of interest

The authors have no conflicts of interest to declare.

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