Influence of Dietary Patterns on Prevalence of Cardiovascular Disease Between Indigenous and Non-Indigenous Australians

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Abstract

In Australia, cardiovascular disease (CVD) is a major health problem that contributes to 26% of total deaths each year with Indigenous people experiencing the greatest burden of disease. The health gap between Indigenous and non-Indigenous populations regarding CVD outcome is a key area of concern and is important to consider how lifestyle choices, including diet, can influence and modify the prevalence of disease. Through a robust analysis of the data collected from the 2011-2012 Australian National Health Survey (ANHS) and using a sample of 5000 made up of 2500 Indigenous and 2500 non-Indigenous respondents, the results concluded that diet is a key predictor of CVD risk. Results reinforced existing understandings surrounding health disadvantages with Indigenous populations have increased rates of major CVD risk factors including type 2 diabetes (T2D), obesity and dyslipidaemia to a statistically significant degree ($p>0.05$). Moreover, through the application of a logistic regression model, results deduced that there is a strong relationship between CVD risk and dietary patterns between the two populations. The discrepancies in diet and health outcomes from this study consequently revealed there is a need for future research to explore ways in which adequate nutrient intake and food security in Indigenous populations can be improved.

1. Introduction

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality in Australia [4]. Despite substantial declines in CVD mortality over the past four decades, it continues to account for 26% of all deaths in the country [4], and holds a substantial financial burden on Australian society, estimated at $12.7 billion in both direct and indirect healthcare costs attributed to CVD [1]. CVD encompasses a spectrum of diseases and conditions affecting the heart and blood vessels, with significant risk factors including obesity, Type 2 Diabetes (T2D), dyslipidaemia, and hypertension [2] identified as strong contributors to the increased susceptibility of developing CVD.

CVD presents a substantial health burden for Aboriginal and Torres Strait Islader communities [1]. Literature underscores the disproportionate impact on Australian Indigenous communities, revealing CVD mortality rates that are 1.6 times higher than their non-Indigenous counterparts [3]. Significantly, CVD stands as the second leading cause of death among Indigenous Australians, responsible for 23% of total deaths [5]. This underscores the profound influence of CVD as the leading cause contributing to the gap in death rates between the two communities [5].

This discrepancy in CVD prevalence is a recurring issue in Indigenous communities worldwide, including Canada, USA, and New Zealand [6]. The evident health gaps in CVD outcomes are primarily attributed to the enduring negative consequences of colonisation, which disrupted the ecological lifestyle, traditional diets, and cultural heritage of Aboriginal and Torres Strait Islander communities [7]. This difference raises critical factors contributing to this health inequity, including the quality of dietary patterns.

Diet quality is increasingly recognized as a pivotal determinant of shaping cardiovascular health. Poor dietary choices, characterised by excessive consumption of ultra-processed foods, low intake of fruit and vegetables, whole grains, fibre, along with high levels of sodium, added sugars, and unhealthy fats, pose significant risks to the development of chronic diseases [8]. This issue is particularly important for the Indigenous Australian population, as recent studies reveal that they are more likely to not adhere to the Australian Dietary Guidelines (ADG) [9], evident as 41% of their daily energy intake is derived from discretionary items high in saturated fat, added sugar and sodium [9]. The interplay between diet and CVD is relatively comprehensive and clear; however, knowledge regarding both, the specific factors underlying excess CVD prevalence and effective strategies to instigate dietary changes within Aboriginal and Torres Strait Islander Australians is still insufficient [10].

This project aims to identify potential dietary pattern disparities in CVD risk factors among a nationally representative sample of Indigenous and non-Indigenous Australian adults, to better understand the modifiable risk factors and ultimately contribute to future strategies and reduce health discrepancies. Understanding the nuances of dietary patterns and their relationship with cardiovascular outcomes is fundamental in creating effective future policies and programs, aimed towards the overarching goal of achieving health equity for all Australians.

Through a thorough analysis of a range of variables, the main findings are that Indigenous people exhibited a significantly higher prevalence in all CVD risk factors encompassing obesity, hypertension, T2D, and dyslipidaemia. A clear disparity in dietary patterns is also evident with Indigenous Australians having a significantly lower consumption of ‘healthy’ foods such as fruits,

vegetables, and lean protein sources, as well as ‘unhealthy’ items including sodium and added sugars. Our findings also determined the influence of key nutrients and food groups that negatively affect role CVD outcomes, underscoring the necessity for a comprehensive, whole-systems approach to address poor diet, specifically in Indigenous communities.

2. Results

2.1 CVD Risk Factors Prevalence between Indigenous and non-Indigenous

The preliminary investigation of the research report analysed the prevalence of CVD amongst Indigenous and non-Indigenous Australian adults to assess any potential disparities. This was achieved through the identification of a range of risk factors defining CVD including obesity, hypertension, dyslipidaemia, T2D, and abnormal Apolipoprotein B (ApoB) levels, using pre-existing ranges from published literature [11].

Table 1. Two-way relative frequency table showing percentage of Indigenous and non-Indigenous in risk factor groups.

	CVD Risk Factors					
	Obese	**Hypertensive**	**Dyslipidemia**	**Type 2 Diabetic**	**Abnormal ApoB**	**Current daily smoker**
Indigenous	61%	18%	73%	25%	26%	45%
Non-Indigenous	42%	20%	50%	13%	19%	11%

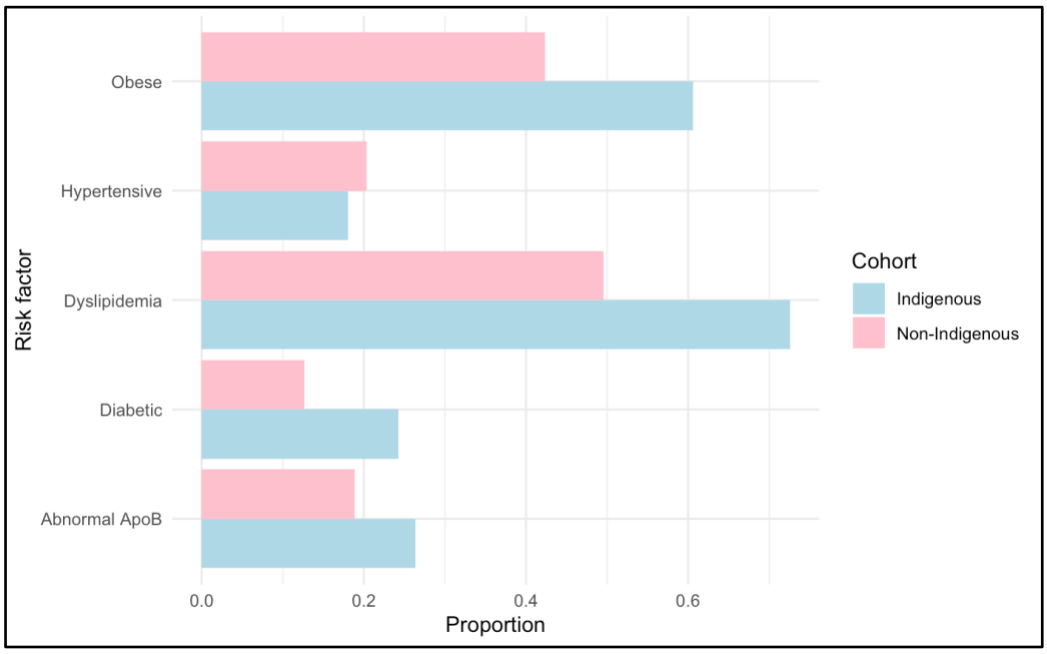

Figure 1. Comparing the prevalence of CVD risk factors between the Indigenous and Non-Indigenous cohorts.

Figure 1 shows that there is a significant disparity in the prevalence of all CVD risk factors between the Indigenous and non-Indigenous cohorts. The prevalence of obesity is significantly higher among the Indigenous population, with 61% of Indigenous individuals classified as obese, compared to 42% of non-Indigenous individuals. The observed disparity in obesity aligns with the AIHW, which indicates that Indigenous adults are 1.2 times more likely to be living with obesity compared to non-Indigenous adults [12].

Dyslipidaemia is higher among the Indigenous population, with 73% of Indigenous individuals affected, compared to 50% of non-Indigenous individuals. Dyslipidaemia is attributed to abnormally elevated cholesterol or fats in the blood [13] suggesting that dietary patterns may have a role in influencing a larger percentage of the Indigenous population falling within the high-risk categories.

The Indigenous cohort demonstrated a notably higher prevalence of T2D, with 24% of Indigenous participants being affected, nearly two times higher than the 13% observed in their non-Indigenous counterparts. The prevalence of abnormal ApoB levels is also consistently higher among the Indigenous population, with 26%, compared to 19% of non-Indigenous individuals. The frequency of abnormal ApoB levels (greater than 1.1 g/L) is substantial as studies have shown that high-fat diets and lipoprotein cholesterol can contribute significantly, thus further motivating our analysis [14]. Conversely, the findings indicated a contrasting trend for hypertension prevalence, in which the non-Indigenous cohort exhibited a higher prevalence, with 20% of non-Indigenous individuals having elevated blood pressure, compared to 18% of Indigenous individuals.

Table 1 also illustrates the frequency of both Indigenous and non-Indigenous individuals who reported smoking status as a current daily smoker. This is an additional risk factor for CVD and has been found that there is a greater proportion of Indigenous than non-Indigenous who participate in this risk behaviour. 45.26% of Indigenous participants compared to 11.13% of non-Indigenous. This is therefore a confounding variable that has the potential to contribute to overall CVD factors.

2.2 Dietary Patterns Among Indigenous and Non-Aboriginal Groups

Based on the observed disparity in CVD risk prevalence, the report now moves on to identifying potential dietary pattern disparities between the two cohorts. Preliminary analysis found 116 variables relating to diet quality in common between the two cohorts. Upon further investigation, the analysis indicated that 110 variables display a significant difference in distribution ($p < 0.01$) when comparing between the two cohorts. A selection of six essential food groups based on the ADG was used to assess diet quality, as shown by Figure 2.

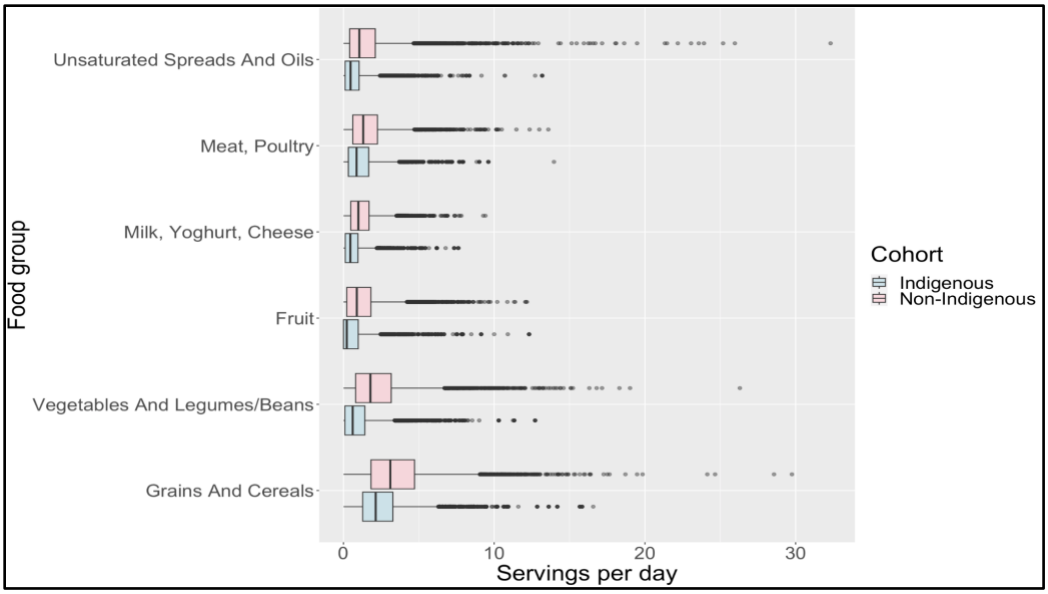

Figure 2. Distribution in recorded food intake (servings per day) between the Indigenous and non-Indigenous cohorts for various food groups.

The study revealed significant disparities in dietary patterns in the consumption of food groups between the two groups of people. The most significant disparities were observed in the consumption of vegetables and legumes/beans and grains and cereals, with a significant median difference of 1.2 servings and 1 serving, respectively. Therefore, Indigenous have less vegetable and grain intake than non-Indigenous. Notably, Indigenous intake more grain than vegetables.

Furthermore, disparities were also observed in the median servings of fruit, unsaturated products (spreads, oils) and dairy (milk, yoghurt, cheese). The difference, amounting to at least half a serving, is 0.67, 0.58 and 0.53, respectively. That emphasised distinct dietary preferences between the two cohorts, showing the potential areas for targeted nutritional interventions. Interestingly, the consumption of *meat and poultry* had the most minor observed disparity in Figure 2, with a median difference of 0.44 servings. This finding suggests a relatively consistent dietary preference for meat and poultry between both cohorts.

Additionally, a selection of seven nutrient groups were also included in the assessment of dietary patterns to better pinpoint specific dietary components that may be contributing to the observed CVD risk disparities as shown by Figure 3.

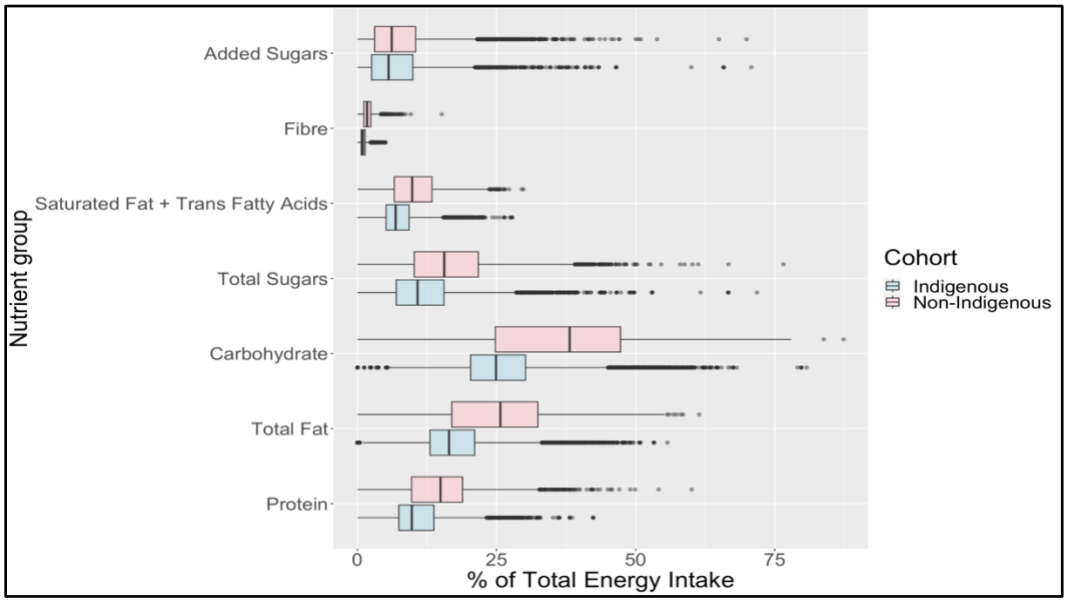

Figure 3. Distribution of energy intakes (as a percentage of the recorded total energy intake) for the Indigenous and non-Indigenous cohort in various nutrient groups.

The diet assessment among the studied cohorts revealed disparities in nutrient intake in Figure 3. The most striking disparities observed are in energy intake from carbohydrates (13.2%), followed by total fat (9.2%). They are indicating the dietary preferences within two cohorts. Moreover, the disparities in protein intake, total sugars and saturated/trans fatty acids were identified with differences of 5.2%, 4.8%, and 3%, respectively. Furthermore, energy intake from fibre shows a minor difference of less than 1% between the two cohorts. However, it is noteworthy that the non-indigenous cohort consumed nearly double the amount of fibre compared to the Indigenous cohort.

2.3 Association between Dietary Pattern and CVD Risk

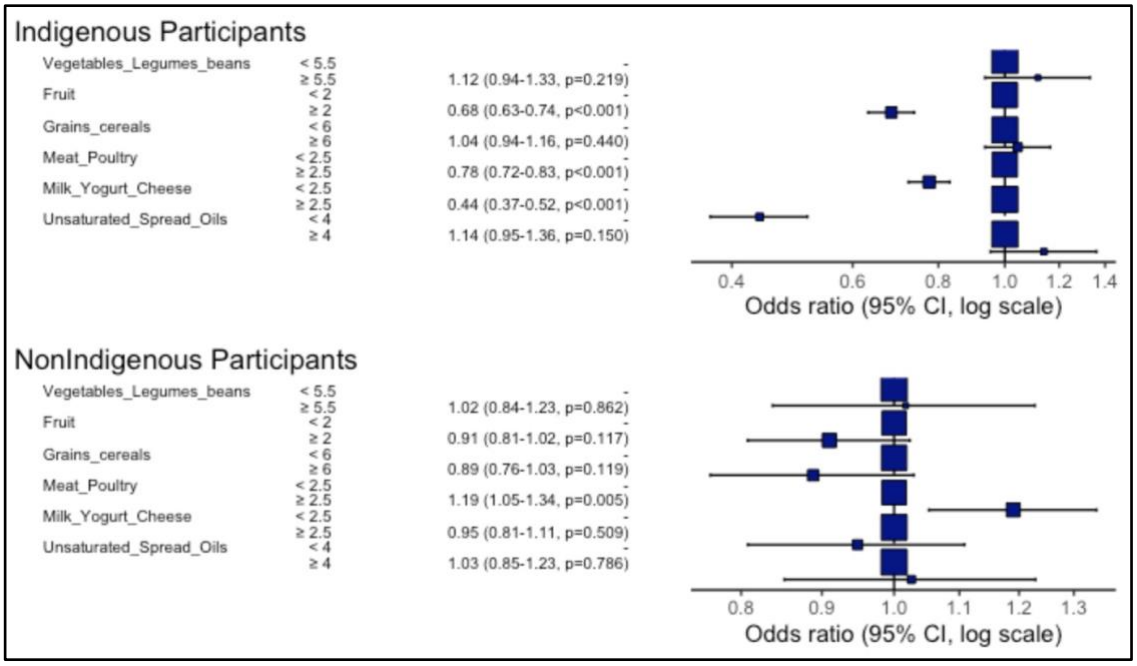

Figure 4. Odds Ratio Plot of the association between CVD risk prevalence and food group consumption in Indigenous and non-Indigenous cohorts. Values were determined based on the recommended number of serves for adults provided by the National Health and Medical Research council's 2013 ADG.

For Indigenous participants, consuming more than 2 servings of fruit per day PR 0.68, CI 95% 0.63-0.74, p<0.001), more than 2.5 servings of meat and poultry (PR 0.78, CI 95% 0.72-0.83, p<0.001), and more than 2.5 servings of milk, yoghurt, and cheese (PR 0.44, CI 95% 0.37-0.52, p<0.001) is significantly associated with a reduced prevalence of CVD risk. Conversely, the non-Indigenous cohort exhibited a different trend, where higher consumption of meat and poultry is significantly associated with increased prevalence in CVD risk (PR 1.19, CI 95% 1.05-1.34, p<0.001). Among both the Indigenous and non-Indigenous cohorts, no significant association is observed between CVD risk prevalence and the consumption of vegetables and legumes/beans, grains and cereals, as well as unsaturated spreads and oils.

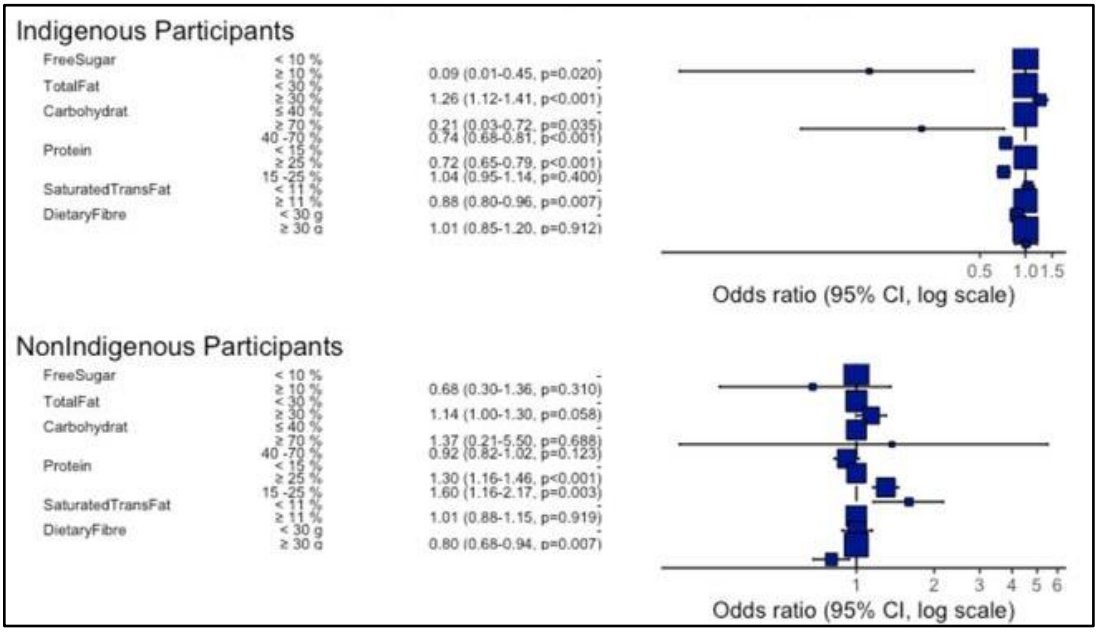

Figure 5. Odds Ratio Plot of the association between CVD risk and nutrient consumption in Indigenous and non-Indigenous cohorts. Values were determined based on the recommended % of total energy intake provided by the World Health Organization for a healthy diet.

In both Indigenous and non-Indigenous cohorts, a clear dietary pattern is observed. A higher total fat intake >30% is significantly associated with increased prevalence of CVD risk in the Indigenous cohort (PR 1.26, CI 95% 1.12-1.41, p<0.001). The non-Indigenous cohort exhibits a similar trend, where a higher total fat intake is associated with increased CVD risk, approaching statistical significance (PR 1.14, CI 95% 1.00 - 1.30, p=0.058). Additionally, while there is a correlation between increased carbohydrate intake (>70%) and increased risk of CVD (PR 1.37, CI 95% 0.21-5.50. P=0.688), it is not statistically significant.

Figure 5 also depicts a trend whereby higher intake of dietary fibre (≥ 30g) in the non-Indigenous cohort is associated with decreased CVD risk prevalence (PR 0.80, CI 95% 0.68-0.94 p=0.007). This aligns with published literature that outlines that individuals with high dietary fibre intake reduce their risk of developing several chronic diseases compared to those who consume less than the ADG recommendation (25-30 g/day) [15]. Comparatively, no significant difference is observed in this same nutrient component within the Indigenous cohort (p=0.912).

Interestingly, a higher intake of saturated/trans fatty acids (≥ 11%) is associated with a decreased CVD risk prevalence within the Indigenous cohort (PR 0.88, CI 95% 0.80-0.96 p = 0.0007), while no significant relationship is observed within the non-Indigenous cohort.

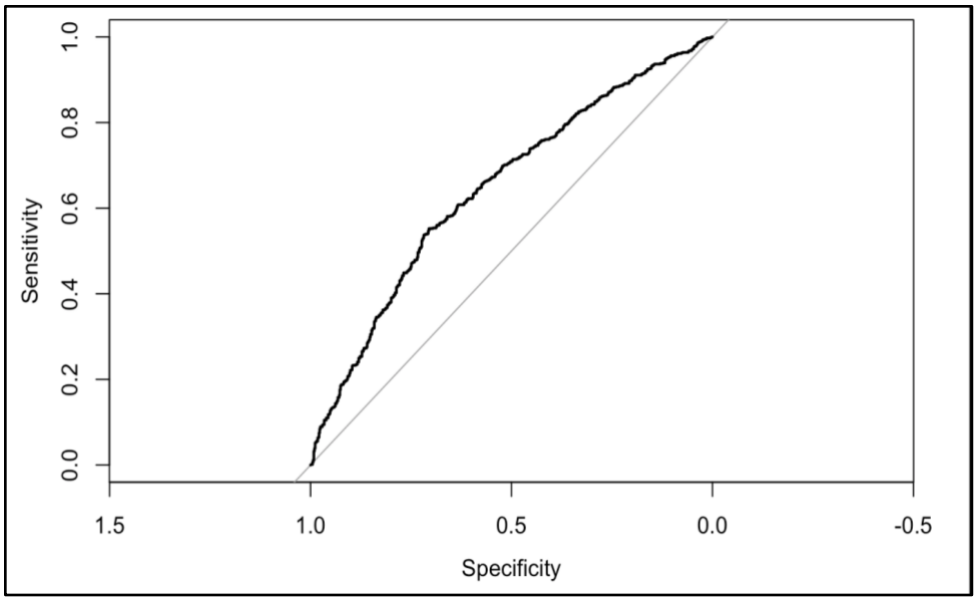

Figure 6. Residual plot derived from the predictive model and is a key performance indicator. The homoscedasticity of the plot is an additional reflection of the appropriateness of a linear regression model for the dataset.

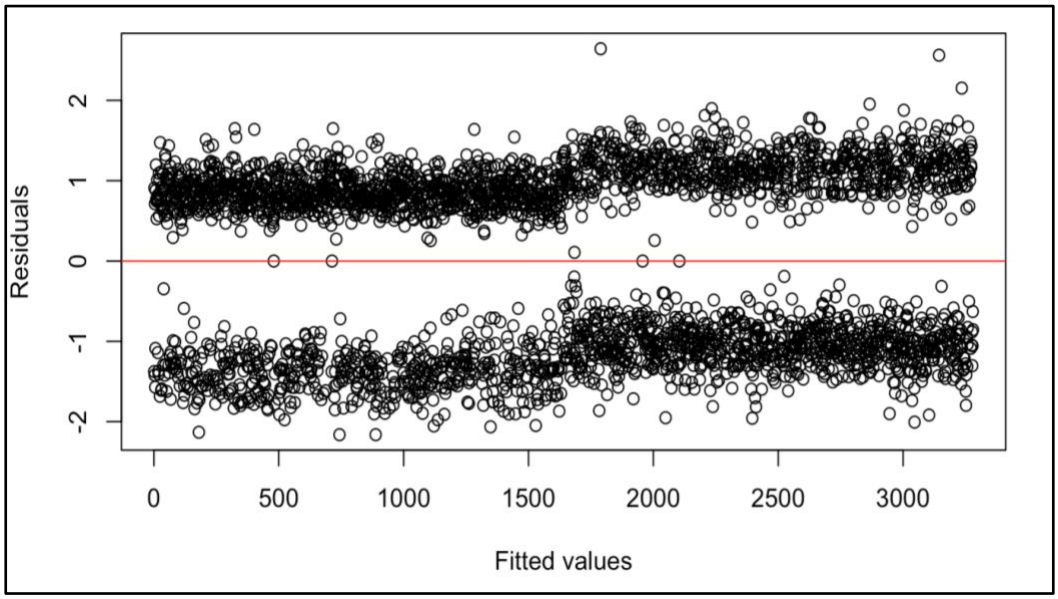

Figure 7. Receiver Operating Characteristic (ROC) curve is an additional tool that is reflective of the performance of the predictive model. The ROC curves aid in the visualisation and establishment of the relationship between overall CVD risk and dietary patterns.

In the logistic regression analysis investigating the association between dietary patterns and high CVD risk, the model demonstrated an accuracy of 66.08%, significantly surpassing the No Information Rate of 59.06%, indicating a meaningful predictive capability. However, the Kappa statistic of 0.2604 suggests only a fair agreement between predictions and observed outcomes, highlighting room for improvement. The model exhibited higher specificity (82.93%) compared to sensitivity (41.77%), indicating a robust ability to identify low CVD-risk individuals but a limited capacity to detect high-risk cases.

Additionally, the residual analysis revealed potential outliers, suggesting that the predictive model might not have captured all factors influencing CVD risk. A significant result from Mcnemar's test revealed an imbalance in prediction errors, indicating potential bias. Notably, the analysis underscored differences in CVD risk factors between Indigenous and Non-Indigenous groups, emphasising the impact of varying dietary patterns across the two populations. While the model incorporated various nutritional variables, their specific contributions to predicting high CVD risk were not detailed.

Overall, a moderate association between dietary patterns and high CVD risk was found, with the model's stronger performance in identifying low-risk individuals and limitations in detecting high-risk cases. This implies the need for further refinement and a more nuanced approach to achieve a comprehensive understanding of the complex relationship between diet and CVD risk, considering cultural and lifestyle factors.

3. Discussion

Since colonisation, Indigenous diets have transformed from a traditional hunter-gatherer diet to a modern, ultra-processed diet. Traditional diets were high in protein, fibre, polyunsaturated fat and complex carbohydrates compared to a contemporary diet, which includes a highly refined carbohydrate diet with added sugars, saturated fat, and sodium [16]. This shift has led to an increase in dietary patterns that are recognised as a significant risk factor for CVD and its related chronic disease [17]. The data examined within the report is consistent with pre-existing literature and reveals Indigenous Australians consume significantly fewer servings of essential food groups as recommended by the ADG, including fruits, vegetables and legumes, proteins, grains and cereal, and dairy foods [18].

These dietary differences are concerning as evidence from epidemiological studies shows that fruit and vegetable consumption is inversely associated with CVD risk factors including blood pressure **[**19], cholesterol [20], and lipid [21**]**. Further, both wholegrains and dietary fibre consumption was found to be significantly lower within the Indigenous cohort. Published literature outlines that individuals with a higher fibre intake have a strong protective association against the risk of developing several chronic diseases, including lower cholesterol levels and blood pressure [22]. This is supported by a study that stated "intakes greater than 30 g a day would be even more beneficial" to reduce rates of CVD-specific and all-cause mortality compared to those who consume less than ADG recommendation (25-30g per day) [15].

The inverse relationship between meat and poultry consumption and CVD risk observed within the Indigenous and non-Indigenous cohorts may be attributed to the broad classification of meat

and poultry, which includes lean and unprocessed meats to highly processed variants. For example, a recent meta-analysis concluded that a higher intake of processed meat is associated with an increased risk of all-cause and CVD mortality [23]. An additional study suggests that incorporating lean, unprocessed meat as part of a diet low in saturated fat does not significantly affect CVD risk factors, including blood cholesterol levels [24]. Although, no clear association between poultry consumption and increased CVD risk has been established [36].

The positive association found in the results between reduced CVD risk and serves of dairy in the form of milk, yoghurt and cheese aligns with the ADG recommendation that Australian adults consume 2-3 servings of dairy products to support positive health [25]. Similarly, research has identified a statistically significant risk reduction of CVD incidence when there is an increased consumption of fermented dairy diets, inclusive of cheese and yoghurt [26]. However, the results show dairy was consumed less amongst the Indigenous cohort, thus aligning with a study that highlights only 18% of Indigenous people were meeting the recommended daily intake [27]**.**

Notably, a similar trend is observed in both cohorts, whereby an increased total fat intake, exceeding 30% of daily energy intake, is associated with a greater prevalence of CVD risk. This is reinforced in literature that revealed approximately 54% of Indigenous energy consumption is derived from ultra-processed foods that are high in total and saturated fat [28]. Contrastingly, increased consumption of saturated/trans-fat (≥ 11%) is significantly associated with a decreased CVD risk prevalence within the Indigenous cohort. This contradicts current literature that suggests a high intake of saturated fats is positively associated with coronary heart disease and ischemic stroke [29]. This conflicting result may result in inaccurate data collection over 2 days as outlined

in the limitations section. Studies have also discussed the benefits of substituting saturated fatty acids with wholegrains, low-fat dairy, and lean protein to mitigate CVD risk [30].

Ultimately, the differences in dietary patterns and CVD risk between Indigenous and non-Indigenous people are influenced by a system of factors. Pre-existing literature examines how colonialist practices such as protection and assimilation policies have contributed to food insecurity [32]. It has been determined that between 20–25% of Indigenous people experience food insecurity compared with 45% of non-Indigenous populations [31]. This level of food insecurity is associated with chronic diseases as high-quality and nutrient-dense foods are often replaced with high-energy, low-quality food sources [7]. Studies have shown that socioeconomic disadvantage is an influential precursor to food insecurity [35]. This is emphasised in data that details that Indigenous households are 2.5 times more likely to be in the lowest-income bracket households than are non-Indigenous households, and consequently have insufficient income to prioritise purchasing fresh and whole foods [34].

These findings suggest the need for personalised and targeted strategies to improve the dietary habits of Indigenous Australians. The disparity in dietary patterns likely stem from a multifaceted interplay of historical, social, and geographical determinants that challenge food supply and security [34]. Therefore, future food and nutrition programs should emphasise improving food security among Indigenous people, in addition to addressing systematic social inequities that influence CVD risk [33]. Further, these initiatives must be developed collaboratively with the active participation of Indigenous communities, ensuring solutions are tailored to their specific needs and delivered in a culturally appropriate manner [37].

4. Limitations of the study

A primary limitation is the cross-sectional design of this study which prevents the causal inference of temporal relationships between diet and CVD risk and may not capture long-term trends. Moreover, substantial missing data in biomedical and nutrient datasets, as well as a lack of overlapping variables between Indigenous and non-Indigenous datasets limits the depth of analysis, hindering the exploration of stronger relationships. The data accuracy from the 24-hour dietary recall should also be considered as participants often under-report their food consumption in nutrition surveys [27], potentially introducing non-sampling errors. Lastly, the study does not account for confounding variables like lifestyle factors, impairing the validity of results. These limitations must be acknowledged when interpreting the results. Addressing them in future research through improved data quality, longitudinal study design, and comprehensive consideration of confounding variables is essential for reliable results.

5. Methods

5.1 Study Design and Participants

This research employs a cross-sectional observational study design. Data for this study is derived from two national surveys conducted by the Australian Bureau of Statistics (ABS): the Australian National Health Survey (NHS) 2011-2012 and the National Aboriginal and Torres Strait Island Nutrition and Physical Activity Survey (NATSIHS) 2012-2013. This study utilised biomedical and nutrition variables that overlapped between the two surveys. The dataset dimensions were using raw data from 12,153 sample size for non-Indigenous participants and 4,109 sample size for Indigenous participants.

5.2 Definition of CVD Risk Factors

CVD risk factors were defined and categorised based on established guidelines and accepted clinical practice as illustrated in Table 2.

Table 2. Summary of the defined ranges for CVD risk factors.

CVD Risk Factors	**Defined Ranges**
Obesity	BMI - ≥ 30 [20] Waist circumference - > 102cm (Males) [20] - > 88cm (Females) [20]
Hypertension	Systolic/Diastolic blood pressure - > 140/90mmHg [18]
Diabetes	HbA1C: - At risk when: HbA1C is 6.0-6.4% [19] - Confirmed diabetic when: HbA1C ≥ 6.5 [19] Fasting plasma glucose: - Considered Impaired Fasting Glucose when: 5.6-6.9mmol/L [19]
Dyslipidemia	HDL Cholesterol - Classified as dyslipidemia when <1.00mmol/L [17] LDL Cholesterol - ≥ 3.5mmol/L [17] Triglycerides - ≥ 2.00mmol/L [17]
Abnormal Apolipoprotein B	ApoB Level - > 110 mg/dL [15]

5.3 Dietary Pattern

Dietary data for all respondents is derived from a 24-hour dietary recall questionnaire conducted as part of the NNPAS surveys. The dietary recall collects information on all foods and beverage intake on the day prior to interview, from midnight to midnight [38]. When possible, participants completed a second 24-hour dietary recall at least eight days after the initial interview [38]. The dietary recall encompassed the estimation of various dietary factors, including the total intake of different food groups, the percentage of energy derived from different nutrient components, as well as measures of nutrient density.

5.4 Statistical Methods

5.4.1 Data Integration

In this research project, biomarker data from two distinct population groups, Indigenous and Non-Indigenous, were merged to create a unified dataset for analysis. The process began with the identification of common variables between the two datasets, ensuring that only relevant biomarkers were included. Then the categorical variables within the dataset were transcribed to factor variables with meaningful labels. To achieve this, a dictionary containing variable descriptions and codes was referenced. Descriptions and codes were extracted from this dictionary and assigned as factor levels, ensuring that the resulting factor variables accurately represented the data. This step was crucial for facilitating subsequent analyses involving categorical variables.

5.4.2 Creation of Target Variables for CVD Risk

The research project aimed to assess CVD risk factors within the study population. To achieve this, target variables were defined based on specific criteria related to CVD risk factors shown in Table 2, including obesity, hypertension, diabetes, ApoB status, and dyslipidemia. These criteria were applied to individual biomarker values, allowing for the classification of study participants into relevant risk categories.

5.4.3 Handling Missing Data & Balancing Group Size

Missing data was handled by removing rows with extensive missing data, ensuring that the remaining data remained representative and robust for subsequent analyses. This data-cleaning step was vital for easy analysis and data integrity. To balance group sizes and ensure that both groups had equal representation in the final dataset, 2500 random samples were selected from each

group. This step was essential for minimising potential biases introduced by group size discrepancies.

5.4.4 Comparison of Proportions

To assess significant differences in CVD risk factors between the two population groups, Chi-squared tests were conducted. This determines the statistical significance of observed differences, allowing for a rigorous and objective evaluation of both populations.

5.4.5 Generalised Linear Model (GLM)

GLM was used to see if a prediction could be made using Machine Learning. For the purpose of this study, we defined CVD risk as ‘high risk’ as individuals that fulfilled at least 2 out of the 5 criteria and ‘low risk’ for individuals fulfilling less than 2 criteria. The dataset was split into 70:30 for training and test set and a logistic regression model was fitted to predict CVD risk based on the nutrition variable then afterwards extracted the model coefficients. The model’s performance was evaluated using a ROC curve and a confusion matrix was generated to evaluate the predictive performance.

5.4.6 Estimate Odds Ratio

To visually elucidate the strength of the association between dietary factors and the risk of CVD within the Indigenous and Non-Indigenous groups, odds-ratio plots were generated. These plots were derived from GLM, appropriate for binary outcome variables such as 'high' or 'low' CVD risk. The resulting plots provided a clear graphical representation of the odds ratios with 95%

confidence intervals, highlighting the dietary factors that were significantly associated with CVD risk.

5.4.7 Resources and Materials

R-studio software was used to analyse the dataset and to implement Machine learning prediction techniques. The packages we used in R were the following: tidyverse, dplyr, ggplot, caret, E1071, janitor, naniar, pROC and stringr.

Author Contribution

510428192, 510678960, 490058239: Conceptualisation, Investigation, Resources, Writing - Original Draft, Writing - Review & Editing**;** 480373146, 500129535, 500505887: Methodology, Software, Data curation, Visualization,

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