ORIGINAL ARTICLE

Diet and Lifestyle in Cardiovascular Risk: A Comparison of Migrants and Non-Migrants in Australia

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ABSTRACT

Cardiovascular disease (CVD) is a leading health issue in Australia and despite its high impact, the specific causes of CVD remain uncertain, though dietary and lifestyle factors are known to influence risk. This study analysed dietary choices and lifestyle factors (smoking, alcohol, physical activity) to examine their impact on cholesterol levels and hypertension risk in migrants versus Australian-born individuals. The analysis began with dietary data cleaning and transformation, removing confounders such as participants on medication. LDL and HDL variables were converted from categorical to numeric and to solve the right censored nature of the categorical bins we employed a mixture of distributions model for male and female LDL and HDL levels based on each gender's published mean and sample size. Finally, we employed a linear mixed model to answer our research questions, accounting for random effects from confounders and interaction effects with migration status. Results revealed that fruit intake positively impacted cholesterol more for migrants from other English-speaking countries than for native Australians. Additionally, Australian-born individuals were at a higher risk of elevated LDL and hypertension due to poor lifestyle choices, suggesting lifestyle behaviours in migrant groups may offer some protection. These insights provide a basis for tailored health interventions to reduce CVD risk across diverse populations.

1 | Introduction

Cardiovascular disease (CVD) is a prominent issue in Australian society. In 2022, CVD accounted for approximately 24% of deaths in Australia, with an additional 6.3% of the population having their health impacted by this disease [1]. The exact cause of CVD is unclear, however, numerous risk factors in an individual's life can influence the risk of developing CVD with cholesterol levels and blood pressure being main contributing risk factors [2]. Lifestyle changes and diet play a large role in influencing these risk factors. Previous evidence also suggests that some migrant populations are disproportionately affected by CVDs [3]. In 2023, Australia's estimated resident population reached 26.6 million, with the immigrant population totalling 8.2 million [4]. With immigration in Australia increasing and the implication that migrants often adopt new lifestyles and diets when migrating to a new country, it is evident to explore whether there is a connection between immigration status and CVD through these risk factors. Looking at the potential influence of immigrant status on CVD risk factors can help determine if immigrants are at greater risk of developing CVD compared to Australian born populace.

Numerous studies have demonstrated that abnormal levels of cholesterol are associated with a high attributable risk for the occurrence of cardiovascular disease (CVD) [5, 6, 7]. Cholesterol is made up of low-density lipoproteins (LDL), high-density lipoproteins (HDL) and triglycerides. LDL is the "bad" cholesterol as elevated levels can lead to cholesterol accumulating in the blood leading to plaque formation which can increase the chances of CVD such as coronary artery disease (CAD) [8, 9, 10]. HDL is the "good" cholesterol as elevated levels have been shown to have an inverse effect on CVD risk [8, 10, 11]. Elevated triglycerides are also negative as they have been associated with an increased risk of coronary heart disease [10]. These components of total cholesterol can be altered with various modifiable factors, one of which being diet. Diet plays a vital role in adjusting cholesterol levels. Diets rich in fruit and vegetables have been seen to reduce LDL cholesterol. In contrast, diets rich in trans and saturated fats have been shown to increase LDL and decrease HDL, which greatly increases CVD risk [12, 13, 14]. Immigration status and diet influencing cholesterol has yet to be fully explored. Immigrants generally have a healthier diet than the native populace when first entering a country, however, after settling in they may have an increasingly unhealthier diet due to other factors. Furthermore, migrants have been shown to be less likely to achieve adequate fruit and vegetable intake compared to non-migrants [15]. Poor diet and lower fruit and vegetable intake may negatively influence cholesterol and lead to an increase in CVD risk.

Among the risk factors for CVD, high blood pressure (BP) is associated with the strongest evidence for causation and it has a high prevalence of exposure [16]. When treating blood pressure, exercise is a key component of lifestyle therapy for its prevention and treatment. Studies have demonstrated that exercise can reduce systole and diastole BP by 5-7 mmHg [17]. On the other hand, a sedentary lifestyle will increase systolic blood pressure and elevate total BP [18]. Smoking and alcohol consumption impact health negatively as they negatively impact BP. Studies have demonstrated that heavy smokers are at a greater risk of hypertension compared to non-smokers [19]. Furthermore, studies have showcased that heavy alcohol consumption does increase risk of hypertension as long as gamma glutamyltransferase (GGT) ≤ 30 IU/L [20, 21]. Those with GGT < 30 IU/L showcase similar risk to nondrinkers [21]. Studies have yet to explore a potential link between exercise, alcohol and smoking with migration status to influence BP. Migrants have been shown to have lower physical activity levels compared to the non-immigrant populace [22, 23]. Studies have also demonstrated that there is considerable variation in smoking prevalence and alcohol consumption among migrants from different countries. People born in Europe have a higher chance of smoking and significantly higher levels of alcohol use problems than Australians [24, 25].

This study aims to investigate how diet and lifestyle factors differentially impact CVD risk measures in migrants compared to non-migrants, assessing whether migration status influences the strength of these associations.

2 | Methodology

2.1 Formulation of Research Questions

Our study examines cardiovascular risk through two focused research questions:

- Investigate the relationship between dietary decisions and cholesterol levels.
- Investigate the impact of lifestyle choices on hypertension risk.

This division allows for a targeted analysis of two key, distinct components of cardiovascular health—cholesterol and blood pressure—each of which is influenced by different modifiable risk factors. Diet is directly linked to cholesterol, as food choices impact lipid levels and ratios, a critical marker for cardiovascular health. Meanwhile, lifestyle behaviors such as exercise, smoking, and alcohol use play a significant role in blood pressure regulation. By addressing these components separately, our approach allows for a clearer understanding of how specific types of decisions influence cardiovascular outcomes, while aligning with established clinical pathways linking diet to lipid levels and lifestyle to hypertension.

2.2 Data Acquisition and Preparation

We sourced our data from the Australian National Health Survey (NHS) [26], which provided comprehensive health-

Code	Medication Classification
B01 17430	Antithrombotic Agents
B06 20190	Other Hematological Agents
C01 20370	Cardiac Therapy
C02 21950	Antihypertensives
C03 23030	Diuretics
C04 24020	Peripheral Vasodilators
C05 24460	Vasoprotectives
C07 25090	Beta Blocking Agents
C08 25910	Calcium Channel Blockers
C09 26300	Agents Acting on the Renin-Angiotensin System
C10 27090	Lipid Modifying Agents

Table 1: Medication classifications used to exclude individuals with medications potentially affecting cholesterol and hypertension outcomes

related metrics, demographic information, and behavioral indicators essential for analyzing cardiovascular risk profiles. We extracted key variables aligned with our study objectives and a detailed set of all the variables we used in our study is found in Appendix A Table 7.
Our data cleaning process involved transforming implicit missing values, converting variables to appropriate data types, and excluding individuals taking medications that could influence cholesterol and hypertension outcomes. Medication status was determined using the following variables:

- **CARDASP**: Daily aspirin use for heart or circulatory conditions (hypertension medication)
- **DIABMQ01**: Daily insulin usage
- **ATCCURFB**: Other specified medications

Individuals were excluded based on **ATCCURFB** classifications for medications affecting cholesterol and hypertension, as detailed in Table 1

2.3 Transformation of HDL and LDL

Initial data pre-processing involved cleaning and expanding the categorical LDL and HDL data for male and female participants separately. The transformation of LDL and HDL cholesterol levels from categorical to continuous values was essential for meeting the analytical needs of our study. While categorical approaches could be applied to predict distinct cholesterol categories, converting these categories into continuous values provided a more precise framework for our research objectives and ensure robustness.
To address the right-censored nature of the data, we fit censored normal distributions to the expanded LDL and HDL data for each gender. Using the `fitdistcens` function, we estimated the mean ($\hat{\mu}$) and standard deviation ($\hat{\sigma}$) for each gender's distribution.

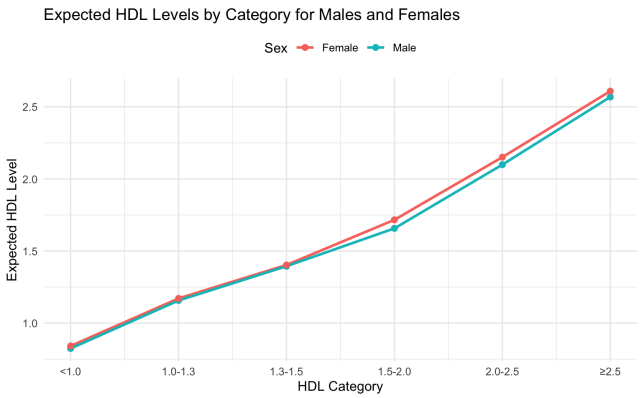
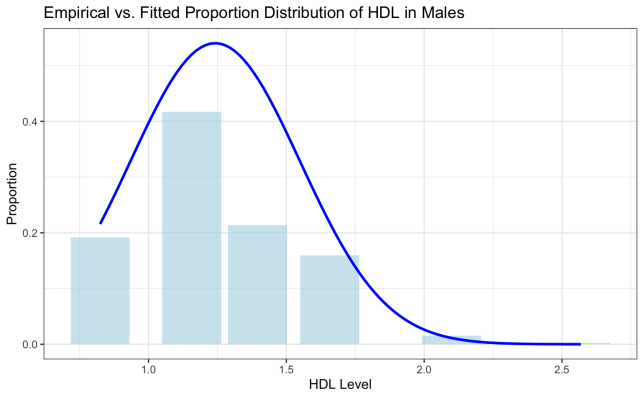
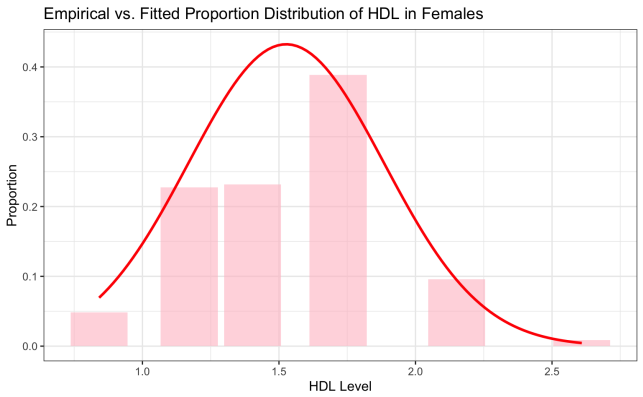


Figure 1: Expected HDL against Categorical bins



(a) Male HDL Distribution



(b) Female HDL Distribution

Figure 2: Empirical vs Fitted Distributions of HDL

For each interval, we calculated the expected continuous value using a truncated normal distribution approach. This process enabled us to transform each LDL and HDL category into a single continuous value that represents the average cholesterol level within the given range. The fitted models for LDL and HDL variables could be found in Figure 2
Finally, we calculated the LDL:HDL ratio for each participant by dividing their expected LDL value by their expected HDL value. This ratio serves as a key variable in our analysis,

enabling comparisons of cholesterol profiles across participants based on migration background, dietary patterns, and lifestyle factors.

Algorithm 1 Transformation of LDL and HDL Variables and Calculation of LDL:HDL Ratio

Input: Categorical LDL and HDL levels with intervals, sample counts for each interval

Output: Expected continuous values for LDL and HDL, and calculated LDL:HDL ratios for each participant

Define $E(\text{interval\_norm}(\mu, \sigma, \text{lower}, \text{upper}))$: **begin**
  if *upper* $= \infty$ **then**
    Set upper $= \mu + 10 \cdot \sigma$
  Calculate numerator $= \int_{\text{lower}}^{\text{upper}} x \cdot f(x \mid \mu, \sigma)\, dx$, where $f(x \mid \mu, \sigma)$ is the PDF of $\mathcal{N}(\mu, \sigma^2)$ Calculate denominator $= F(\text{upper} \mid \mu, \sigma) - F(\text{lower} \mid \mu, \sigma)$, where F is the CDF of $\mathcal{N}(\mu, \sigma^2)$ **return** $E = \frac{\text{numerator}}{\text{denominator}}$
for *each cholesterol type (LDL, HDL)* **do**
  for *each gender (male, female)* **do**
    Define data with columns: Range, Lower, Upper, and Count **for** *each category* **do**
      Calculate total count by multiplying Count by 1000 Create censored_data with columns left = Lower, right = Upper, and count = Count Expand censored_data by replicating each row by its count
    Fit censored normal distribution $\mathcal{N}(\hat{\mu}, \hat{\sigma}^2)$ on expanded censored_data **for** *each category* **do**
      Calculate expected value E for interval using $E(\text{interval\_norm}(\hat{\mu}, \hat{\sigma}, \text{Lower}, \text{Upper}))$
    Assign categories for each range
for *each participant* **do**
  Calculate LDL:HDL ratio using transformed LDL and HDL expected values:

$$\text{LDL:HDL} = \frac{E(\text{LDL})}{E(\text{HDL})}$$

return Transformed continuous LDL and HDL values with calculated LDL:HDL ratios

2.4 Modeling

To address our research questions, we implemented a linear mixed effects model, which allowed us to investigate the impact of dietary and lifestyle factors on cholesterol levels and hypertension risk with random effects for potential confounder and interaction effects for migration status. This choice was made due to the model's capacity to account for both fixed effects (dietary and lifestyle determinants) and random effects (individual-specific differences) in a single framework, thus controlling for unobserved heterogeneity among participants. Specifically, the inclusion of random intercepts allowed us to account for variability attributable to unmeasured confounding factors, while interaction terms enabled us to explore how dietary choices, cholesterol levels, and lifestyle behaviors impacted CVD risk differently between migrant and native-born populations. Our model is specified as follows:

$$Y_{ij} = \beta_0 + \sum_{k=1}^{p} \beta_k X_{ijk} + \sum_{m=1}^{q} \delta_m (X_{ijk} \times Z_{ij}) + \gamma_j + u_i + \epsilon_{ij} \tag{1}$$

The model includes a fixed intercept (β_0), representing the baseline outcome level, and fixed effects ($\beta_k X_{ijk}$) for dietary and lifestyle factors, allowing us to quantify their direct impact. Interaction terms ($\delta_m(X_{ijk} \times Z_{ij})$) reveal how these effects differ by migration status. Random intercepts (γ_j) account for unmeasured differences between groups (e.g., migration background), while individual-level random effects (u_i) capture unique personal variability. Finally, the residual error (ϵ_{ij}) represents random variation in the outcome not explained by the model. Together, these terms enable a comprehensive analysis of dietary and lifestyle factors on cardiovascular risk across populations.

3 | Results

3.1 Diet and Cholesterol

Model Specification

We used a linear mixed-effects model to assess the relationship between dietary and lifestyle factors and LDL levels. The model was specified as follows:

$$y = \beta_0 + \big(X_1 + X_2 + X_3 + X_4 + X_5 + X_6\big) \times X_7 + (1|X_8) + \epsilon \tag{2}$$

Where:

- X_1: Usual daily serves of vegetables (DIETQ5)
- X_2: Usual daily serves of fruit (DIETQ8)
- X_3: Fat content of main type of milk usually consumed (MILKFATU)
- X_4: Salt use at the table (SALTATI)
- X_5: Salt use in cooking (SALTACI)
- X_6: Main type of milk usually consumed (DIETQ1)
- X_7: Country of birth category (COBCODBC)
- X_8: Random intercept for age group
- β_0: Intercept term
- ϵ: Residual error term

The model includes an interaction term between each dietary/lifestyle variable (X_1 to X_6) and **COBCODBC** (country of birth) to assess whether the effects of diet and lifestyle factors on LDL levels vary by country of birth. A random intercept for **AGEB** is included to account for variability in LDL levels across age groups.

Table 2: Summary of Model Results for LDL HDL Ratio

Predictors	**Estimates**	**p-value**
(Intercept)	2.63	<0.001
DIETQ5	-0.05	<0.001
DIETQ8	-0.04	0.007
MILKFATU [2]	-0.09	0.016
MILKFATU [3]	-0.17	0.001
DIETQ1 [2]	-0.18	0.016
COBCODBC [2]	-0.26	0.048
DIETQ8 × COBCODBC [2]	-0.08	0.051
Random Effects		
σ^2	0.75	
τ_{00} AGEB	0.04	
ICC	0.05	
N AGEB	17	
Observations	3617	
Marginal R^2 / Conditional R^2	0.026 / 0.078	

Table 3: Summary of Mixed-Effects Model Results for LDL Expected

Predictors	**Estimates**	**p-value**
(Intercept)	3.08	<0.001
DIETQ5	0.02	0.245
MILKFATU [5]	0.27	0.079
COBCODBC [3]	-0.48	0.034
DIETQ8 × COBCODBC [3]	0.14	0.002
MILKFATU [3] × COBCODBC [3]	0.57	0.020
MILKFATU [4] × COBCODBC [3]	2.31	0.018
Random Effects		
σ^2	0.66	
τ_{00} AGEB	0.14	
ICC	0.17	
N AGEB	17	
Observations	911	
Marginal R^2 / Conditional R^2	0.048 / 0.211	

Table 2 presents a summary of the LDL:HDL ratio model, showing only the significant predictors. Table 3 provides a summary of the absolute LDL model, also displaying only significant variables.

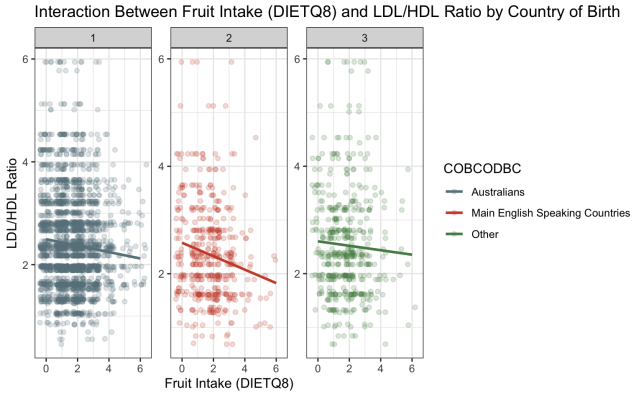


Figure 3: Interaction Between Fruit Intake and LDL/HDL ratios between migration groups

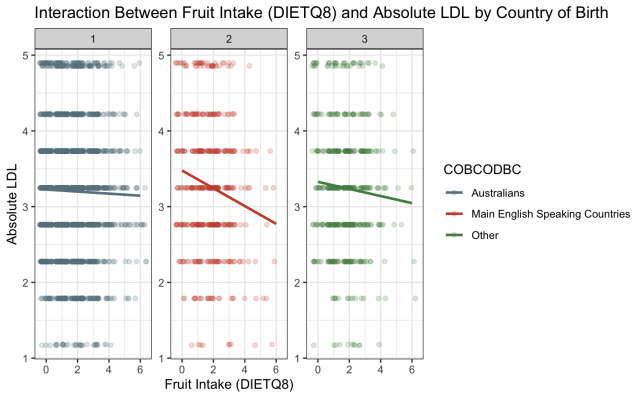


Figure 4: Interaction Between Fruit Intake and absolute LDL between migration groups

Interaction of Fruit and LDL by Country of Birth

The analysis revealed a statistically significant interaction effect between daily fruit intake and country of birth on absolute LDL levels ($p = 0.002$ for individuals from Main English-speaking countries). This suggests that the impact of fruit intake on LDL levels varies based on country of birth. Specifically:

- For individuals from Main English-speaking countries other than Australia, higher daily fruit intake is significantly associated with lower LDL levels, as evidenced by a downward trend in the plot for this group.
- For native Australians and individuals from other countries, the interaction between fruit intake and LDL levels was not statistically significant ($p = 0.123$ for the "Other" category), indicating no substantial effect of fruit intake on LDL levels in these groups.

These findings suggest that fruit intake may have a beneficial effect on LDL reduction primarily for individuals from Main English-speaking countries other than Australia.

Interaction of Fruit and LDL/HDL by Country of Birth

The interaction between daily fruit intake and country of birth on the LDL/HDL ratio showed mixed results:

- There was a trend towards a significant interaction for individuals from Main English-speaking countries (p = 0.051). Although just above the conventional threshold for significance, this near-significant result suggests a potential differential impact of fruit intake on the LDL/HDL ratio in this group, favoring lower ratios with higher fruit intake.
- For native Australians and individuals from other countries, no significant interaction effect was observed (p = 0.908 for the "Other" category), indicating no substantial influence of fruit intake on the LDL/HDL ratio within these groups.

These results align with the findings for absolute LDL levels, suggesting that fruit intake may benefit individuals from Main English-speaking countries other than Australia by reducing the LDL/HDL ratio, with limited effect observed for native Australians and other populations.

Summary of Findings

In summary, a significant interaction between fruit intake and country of birth on LDL levels indicates that individuals from Main English-speaking countries other than Australia experience a greater reduction in LDL with higher fruit intake, whereas Australians and individuals from other countries do not. For the LDL/HDL ratio, a similar trend is observed, with a near-significant interaction suggesting that fruit intake may benefit individuals from Main English-speaking countries in reducing the LDL/HDL ratio.

Conclusion on Cholesterol Metrics

Given the ongoing debate over the optimal metric for assessing cardiovascular risk—whether to use absolute LDL levels or the LDL/HDL ratio—our study highlights the value of examining both. Our results show that dietary influences can manifest differently across these metrics, underscoring the importance of a multifaceted approach when assessing cholesterol-related health outcomes.

3.2 Lifestyle and Hypertension

Alcohol Consumption

$$\text{SYSTOL} = \beta_0 + (W_1 + W_2) \times X_7 + (1|X_8) + (1|X_9) + \epsilon \quad (3)$$

where:

- W_1: Normalized total alcohol consumption (TOTPAC_normalise)
- W_2: Normalized weekly alcohol consumption (ALCWKLY_normalise)
- X_7: Country of birth category (COBCODBC)
- X_8: Random intercept for age group (AGEB)
- X_9: Random intercept for sex (SEX)
- β_0: Intercept term
- ϵ: Residual error term

Table 4: Summary of Linear Mixed Model Results for SYSTOL by Alcohol Consumption and Country of Birth

Predictors	**Estimates**	**p-value**
(Intercept)	124.58	<0.001
COBCODBC3	1.27	0.020
ALCWKLY_normalise	0.37	0.005
Random Effects		
AGEB Variance	121.16	(SD = 11.01)
SEX Variance	9.22	(SD = 3.04)
Residual Variance	308.69	(SD = 17.57)
Observations	20214	
Groups (AGEB)	16	
Groups (SEX)	2	

Our analysis revealed that normalised total weekly alcohol consumption (quantified in millilitres) exerts a statistically significant positive influence on both systolic (SYSTOL) and diastolic (DIASTOL) blood pressure levels, with both measures increasing concomitantly with alcohol intake ($p < 0.05$). In statistical terms, the $p < 0.05$ value denotes significance at the5% threshold, providing sufficient evidence to reject the null hypothesis. Here, $p < 0.05$ underscores that alcohol consumption significantly affects systolic and diastolic blood pressure; the intercept in the model suggests a base systolic blood pressure of 127.0215 mmHg, implying a high baseline level before considering alcohol's impact, with a standard error of 3.4252, and a t-value of 37.085, indicating a strong and reliable estimate and indicating that the observed rise in blood pressure associated with elevated alcohol intake is unlikely to be attributable to random variation. Consequently, we can infer with confidence that higher alcohol consumption is correlated with increased blood pressure levels.
However, the interaction effect between alcohol consumption and migrant status did not reach statistical significance (interaction effect $p > 0.05$), indicating that the association's strength between alcohol intake and hypertension markers does not significantly differ between migrant and non-migrant groups. This finding suggests that while alcohol consumption is a robust predictor of elevated blood pressure across the population, migrant status does not substantially modify this relationship.

Smoking Behaviour

$$\text{SYSTOL} = \beta_0 + (Z_1 \times X_7) + (1|X_8) + (1|X_9) + \epsilon \quad (4)$$

Where:

- Z_1: Smoking status (SMKSTAT)
- X_7: Country of birth category (COBCODBC)
- X_8: Random intercept for age group
- X_9: Random intercept for sex
- β_0: Intercept term
- ϵ: Residual error term

The model includes an interaction term between smoking status (Z_1) and **COBCODBC** (country of birth) to determine if the effect of smoking status on systolic blood pressure varies by country of birth. Random intercepts for **AGEB** (age group) and **SEX** are included to account for within-group variability.

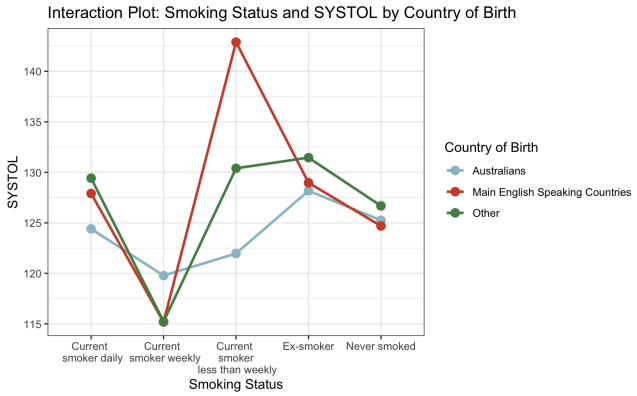


Figure 5: Interaction Plot of Smoking against SYSTOL

We can see from Table 5 that smoking behaviour has a significant impact on both systolic (SYSTOL) and diastolic (DIASTOL) blood pressure levels ($p < 0.05$). Specifically, the coefficient for current daily smokers (SMKSTAT4) is -3.2702, with a standard error of 0.6585 and a t-value of -4.966, demonstrating a significant increase in systolic blood pressure. Individuals who have never smoked have significantly lower systolic blood pressure compared to current daily smokers, and ex-smokers also exhibit substantially lower systolic blood pressure than current daily smokers. This suggests that both those who have never smoked and those who have quit smoking maintain lower systolic blood pressure levels compared to daily smokers.

Table 5: Summary of Linear Mixed Model Results for SYSTOL by Smoking Status and Country of Birth

Predictors	**Estimates**	**p-value**
(Intercept)	127.4	<0.001
SMKSTAT2	-2.70	0.017
SMKSTAT4	-2.92	<0.001
SMKSTAT5	-1.83	<0.001
SMKSTAT5 × COBCODBC2	-2.27	0.011
Random Effects		
AGEB Variance	134.16	(SD = 11.58)
SEX Variance	6.11	(SD = 2.47)
Residual Variance	328.40	(SD = 18.12)
Observations	37512	
Groups (AGEB)	16	
Groups (SEX)	2	

In comparing migrants and non-migrants, we found that the difference in systolic blood pressure between non-smokers and daily smokers is more pronounced among individuals born in Australia. Compared to those born in other major English-speaking countries and other regions, the transition from never smoking to daily smoking among Australians has a greater negative impact on systolic blood pressure ($p < 0.05$). Current smoker weekly (SMKSTAT2) with a coefficient of 0.2374, a standard error of 1.3047, and a t-value of 0.182), suggesting the effect of smoking on blood pressure is consistent across different countries of birth.

Exercise

$$\text{SYSTOL} = \beta_0 + (W_1 + W_2 + W_3 + W_4 + W_5) \times X_7 + (1|X_8) + (1|X_9) + \epsilon \quad (5)$$

Where:

- W_1: Whether exercise last week met 150 minutes (EXREGUIC)
- W_2: Whether exercise last week met 150 minutes and 5 sessions (EXREGUID)
- W_3: Total minutes walked for fitness, recreation, or sport last week (EXFSRMNE)
- W_4: Total minutes of moderate exercise last week (EXLWMBC)
- W_5: Total minutes of vigorous exercise last week (EXLWVBC)
- X_7: Country of birth category (COBCODBC)
- X_8: Random intercept for age group (AGEB)
- X_9: Random intercept for sex (SEX)

- β_0: Intercept term
- ϵ: Residual error term

Table 6: Summary of Linear Mixed Model Results for SYSTOL by Exercise and Country of Birth

Predictors	**Estimates**	**p-value**
(Intercept)	125.5	<0.001
COBCODBC2	-2.25	0.001
EXREGUID2	1.73	<0.001
COBCODBC2 × EXREGUID2	3.50	0.003
COBCODBC3 × EXLWVBC	-0.0073	0.033
Random Effects		
AGEB Variance	120.43	10.97
SEX Variance	5.56	2.36
Residual Variance	334.37	18.29
Observations	36476	
Groups (AGEB)	15	
Groups (SEX)	2	

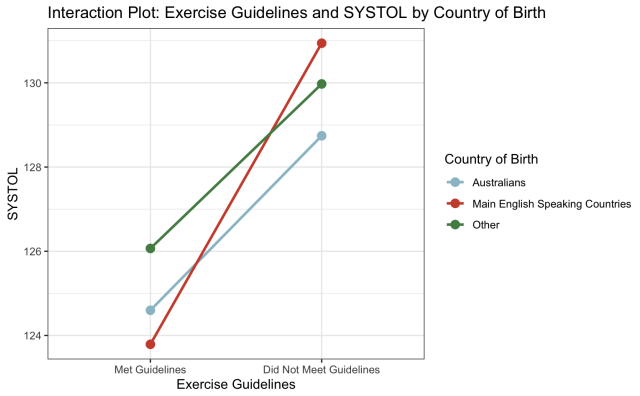


Figure 6: Interaction Plot of Exercise Guidelines against SYSTOL

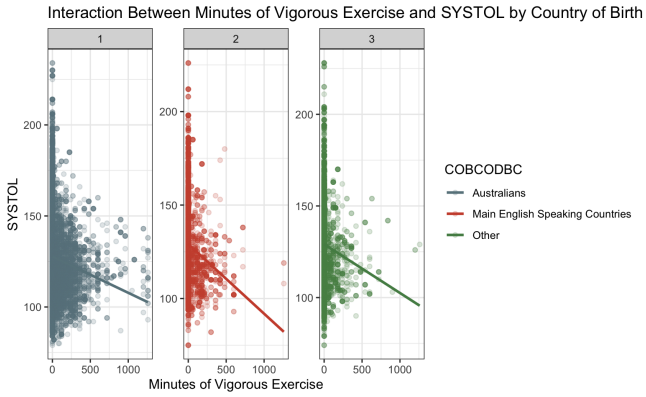


Figure 7: Interaction between Number of Minutes of Vigorous Exercise against SYSTOL

The analysis illustrates that exercise behaviour significantly impacts systolic blood pressure (SYSTOL), with variations across individuals of different birth origins. Specifically, individuals who did not meet the recommended exercise guidelines (150 minutes of physical activity and five sessions per week) exhibited significantly higher systolic blood pressure levels; Exercise guideline 150M (EXREGUIC) shows a negative interaction coefficient of -0.4956 with a standard error of 0.5247 and a t-value of -0.945, though not statistically significant, it points towards a trend that warrants further investigation. Particularly among those born in Australia.
Additionally, the duration of vigorous exercise is inversely related to systolic blood pressure, indicating that longer periods of intense physical activity are associated with lower systolic levels.
In summary, our analysis demonstrates that alcohol consumption, smoking behaviour, and exercise habits each exert significant influences on systolic (SYSTOL) and diastolic (DIASTOL) blood pressure levels, with notable variations across individuals of different birth origins. Alcohol intake consistently correlates with elevated blood pressure across all groups unaffected by migrant status, suggesting the need for universal health guidelines to manage hypertension risks associated with alcohol. Smoking impacts blood pressure significantly, with non-smokers and ex-smokers showing lower systolic levels than daily smokers; this effect is especially pronounced among Australians, indicating that smoking cessation strategies may benefit from birth-origin-specific considerations. Exercise adherence, particularly meeting recommended guidelines and engaging in vigorous physical activity, is linked to lower blood pressure, with the most substantial benefits observed in individuals from ”Other” countries. These findings underscore the importance of personalized health recommendations considering birth origin, lifestyle, and cultural factors, enabling more targeted and effective hypertension prevention and management strategies.

4 | Discussion

In this study, it was found that smoking elevates hypertension risks more for Australians than immigrants, while fruit consumption provides greater cholesterol-lowering benefits for immigrants than for Australians. Insufficient exercise increased hypertension risk among immigrants; however, engaging in vigorous exercise offers immigrants greater benefits. There was no difference in the effects of alcohol on LDL/HDL levels between the groups.
The elevated risk of hypertension from smoking in Australian-born residents may be influenced by smoking behavior. Previous studies have found that the adverse effect of smoking was stronger after 37.7 years of smoking[27]. Previous evidence suggests that immigrants from non-English speaking countries had lower levels of smoking; however, this prevalence increased with a longer time of residence in Australia[28]. Australians in this study could have longer periods of smoking when compared to immigrants, contributing to an in-

creased CVD risk. Therefore, anti-smoking campaigns, especially focusing on smoking duration in Australians, may be beneficial. Genes may play a role in this observed difference, though there are differing findings on this topic. Mendelian randomisation studies looking at genetic influences in this context have not provided a clear causal link between smoking and hypertension[29]. However, other studies suggest that genes play a role in the development of chronic obstructive pulmonary disease (COPD). A variant of the HHIP gene is found more commonly in people with COPD, and smokers with this variant experience greater lung decline[30]. Therefore, genetic variants may differ between Australians and migrants, meaning genes could play a role in the elevated risk of hypertension.

The western diet contains more processed foods, which leads to an increased risk of non-infectious diseases such as CVD[31]. When immigrants move to Western countries like Australia, dietary acculturation results in increased intakes of processed foods and higher amounts of sugar, saturated fat, and sodium[32]. For example, Indians, the largest non-English speaking migrant group in Australia, have culturally-based diets made up mainly of carbohydrate-dense foods, and the introduction of western fast foods places the Indian immigrant population at an increased risk of CVD[4, 33, 34]. Further, migration status can create financial pressures that lead to unhealthier food choices, as cheaper, processed foods are consumed more[33]. Therefore, with dietary acculturation, immigrants have an increased risk of CVD, and an increase in the consumption of fruit may provide a greater protective effect. This is because fruits provide beneficial nutrients and phytochemicals that act on biological mechanisms to reduce cholesterol levels and CVD risk[35]. A meta-analysis found that for each 200g/day intake of fruit and vegetables, there was an 8–13% reduction in the relative risk of CVD[35]. Genes may also play a role in the greater influence of fruit for migrants. For example, variants in SLC23A1 have been found to be associated with circulating concentrations of L-ascorbic acid[36], and genome-wide association studies have found that the rs12272004 SNP is associated with higher plasma alpha-tocopherol concentrations[37]. Differences in gene variants between immigrants and Australian-born residents may have contributed to the differential effect of fruit that was found. This highlights the benefit of providing health interventions that emphasise fruit intake for migrants to improve CVD outcomes.

The difference in the effect of exercise on CVD risk profiles could be attributed to genetic differences and the type of exercise performed. For example, endurance training is associated with higher levels of cardiorespiratory fitness, which decreases CVD risk[38]. Higher intensity exercise has also been found to have additional cardiovascular benefits compared to moderate exercise[39]. Hence, exercise type could influence the increased benefit of vigorous exercise on immigrants. Further, polymorphisms in LIPG can influence CVD risk. The alleles of rs2000813, rs2276269, and rs9951026 were associated with less CVD and a better lipid profile, but only in patients who had moderate to high levels of physical activity[40]. Our findings suggest campaigns that focus on increasing vigorous exercise may help decrease CVD in migrant groups.

We found differences in the effects of smoking, fruit intake, and exercise on CVD risk between Australians and immigrants. These findings highlight the importance of accommodating the diverse health profiles and cultural contexts within the Australian population. This study had many limitations, and further studies are required to determine the influence of genetic differences on CVD risk.

5 | Limitations

This study is limited by the fact that data used is old data from about 10 years ago, not reflective of today's data. Instead data should be collected from today's age to correct for outdated data. The data gathered also has more observations of Australians than migrants, leading to imbalance data. Further collection of migrant data will correct this. Bias is common in studies, with recall and cultural bias being potentially present in the data. To reduce these biases, prospective study designs, short recall periods, and recording clients responses fully can be done. Finally we assumed a normal distribution for LDL and HDL, which may be inaccurate given their positively bounded nature.

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Appendix A

Dataset	Variable Code	Description
AHSnhs11bsp	COBCODBC	Country of birth
AHSnhs11bsp	YOABC	Year of arrival in Australia
AHSnhs11bsp	AGEB	Age of person
AHSnhs11bsp	BMISC	Body Mass Index (BMI) - score measured
AHSnhs11bsp	SYSTOL	Systolic Blood Pressure (mmHg)
AHSnhs11bsp	DIASTOL	Diastolic Blood Pressure (mmHg)
AHSnhs11bsp	SEX	Sex of person
AHSnhs11bbi	CHOLRESB	Total cholesterol - ranged (mmol/L)
AHSnhs11bbi	HDLCHREB	HDL cholesterol - ranged (mmol/L)
AHSnhs11bbi	LDLRESB	Fasting LDL cholesterol - ranged (mmol/L)
AHSnhs11bbi	CHOLNTR	Total cholesterol status (mmol/L)
AHSnhs11bbi	HDLCHSEX	HDL cholesterol status - sex dependent (mmol/L)
AHSnhs11bbi	DIABPRVE	Diabetes prevalence - HbA1c (%)
AHSnhs11bmd	ATCCURFB	Type of medication taken in last 2 weeks (code)
AHSnhs11bmd	INSFLAG	Whether insulin used daily
AHSnhs11bsp	CARDASP	Whether uses aspirin daily for heart or circulatory condition
AHSnhs11bsp	SMKSTAT	Smoker status
AHSnhs11bsp	ALCUSUQ2	Frequency of alcohol consumption in the last 12 months
AHSnhs11bsp	TOTPAC	Mls of pure alcohol consumed
AHSnhs11bsp	ALCSTR01	Short term alcohol risk (2001 Guidelines)
AHSnhs11bsp	ALCWKLY	Estimated total weekly consumption (in mls)
AHSnhs11bsp	PHDKGW2	Measured weight (kg)
AHSnhs11bsp	ALINTWK	Average daily intake over week (in mls)
AHSnhs11bsp	EXREGUIC	Whether exercise last week met 150 minutes recommended guidelines
AHSnhs11bsp	EXREGUID	Whether exercise last week met 150 minutes and 5 sessions recommended guidelines
AHSnhs11bsp	EXFSRMNE	Total minutes walked for fitness, recreation or sport in last week
AHSnhs11bsp	EXLWMBC	Total minutes undertaken moderate exercise in last week
AHSnhs11bsp	EXLWVBC	Total minutes undertaken vigorous exercise last week
AHSnhs11bsp	DIETRDI	Whether vegetable and fruit consumption met recommended guidelines

Table 7: Key variables from the National Health Survey dataset used in the study