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Cardiovascular risk factors and their associations with alcohol consumption: are there differences between Māori and non-Māori in Aotearoa (New Zealand)?

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Abstract

Aims To describe the relationship between indicators of alcohol consumption and major known cardiovascular risk factors, and to test whether these relationships are different for Māori and non-Māori.

Methods Data from five New Zealand studies (national and population specific) conducted since 1988 were made available to the investigators and were re-analysed by Māori and non-Māori classification using multivariate modelling adjusting for sex and age. Three indicators of alcohol consumption were used: frequency of drinking, volume drunk on a typical or usual occasion, and average daily consumption. Interaction terms were used to test for differences between Māori and non-Māori in the associations between alcohol consumption and cardiovascular risk factors (tobacco smoking, systolic and diastolic blood pressure, high density lipoprotein (HDL), the ratio of total cholesterol to HDL, serum glucose, reported diagnosis of diabetes, and body mass index).

Results There were a total of 44,830 people in the combined study populations of whom 6926 (15.4%) were Māori. For the risk factors examined, in general Māori had higher levels of risk compared to non-Māori. The pattern of associations between each of the three indicators of alcohol consumption and lipid factors, diabetes, serum glucose level, and obesity were not shown to be different in Māori and non-Māori. However for systolic blood pressure and tobacco smoking, the patterns of association were different.

Conclusion There are clear associations for most of the cardiovascular risk factors examined and alcohol consumption. These associations are consistent for Māori and non-Māori, except for blood pressure and cigarette smoking. As the study is hypothesis-generating, further investigation is required for confirmation.

The relationship between alcohol consumption and cardiovascular disease mortality has been investigated in numerous studies, including several case control studies in non-Māori New Zealanders. Most studies show that low to moderate alcohol consumption is associated with a reduced risk of coronary disease in middle-aged and older people.[1–3] However little is known about this relationship in Māori.

We have previously reported the differences in drinking patterns of Māori and non-Māori, and showed that (although average daily consumption was similar) compared to non-Māori, Māori drink alcohol less often but in greater volume.[4]

In the absence of direct evidence from case-control or cohort studies on the relationship between alcohol and cardiovascular disease (CVD) in Māori, we have

attempted to examine the relationship indirectly by describing the relationship between alcohol consumption and CVD risk factors in Māori and non Māori.

The aims of this paper are to:

- Describe the relationship between indicators of alcohol consumption and major cardiovascular risk factors: tobacco smoking; systolic blood pressure (SBP) and diastolic blood pressure (DBP); high-density lipoprotein (HDL) and the ratio of total cholesterol to HDL; serum glucose; and body mass index (BMI).
- Test whether the relationships found are different for Māori and non-Māori.

Methods

Data collection—We identified five large New Zealand studies that gathered information about cardiovascular risk factors and alcohol consumption, had large Māori representation, and for which data were available to the authors. Included were two national cross-sectional surveys (New Zealand Health Survey 1997 and the Sleep Survey 1999) and baseline data from three cohort studies (Fletcher Challenge /University of Auckland Survey 1992, NZ Blood Donors Health Study 1998-1999, and the Workforce Diabetes Survey 1988-1990). Details of sampling, inclusion criteria, and data collection procedures are available in the literature.[5–9]

Of the five studies, two were based on randomly selected population samples: the NZ Health Survey (merged with its subset the NZ Nutrition Survey), and the Sleep Survey. All studies included information about age, sex, ethnicity, and drinker/non-drinker status and tobacco smoking status. Only drinker/non-drinker status and average daily volume could be assessed in The Workforce Diabetes Study, whereas all other studies were also able to assess frequency of drinking and volume consumed per occasion.

Only adults aged 18 to 74 years were included in the study, and they were classified in three age-groups (18–34, 35–54, and 55–74 years). Participants selected the ethnic group(s) with which they identified, and for this purpose were classified as Māori if there was any mention of Māori ethnicity, or otherwise as non-Māori.

Three indicators of alcohol consumption were used:

- Frequency of alcohol consumption was categorised in a slightly different manner in each survey. To obtain a comparable measure across the surveys, the mid-point of the interval selected in each study questionnaire was used to estimate the number of days on which alcohol was consumed each year.
- Volume of alcohol consumed on a typical or usual occasion was calculated for each participant according to the type of alcoholic drink typically consumed, and the number of drinks consumed.
- Estimates of average daily consumption were calculated from the number of days on which alcohol was consumed and the volume consumed on a typical or usual occasion.

Frequency of drinking, volume drunk on a typical occasion, and average daily consumption were categorised into five ordinal groups roughly approximating quintiles. The cut-points of each group were selected to ensure that reasonable numbers of participants in each study were classified in each group.

In all, eight cardiovascular risk variables were classified if available in the survey data. Six cardiovascular risk factors were continuous measures: systolic blood pressure (SBP) and diastolic blood pressure (DBP) in mmHg, high density lipoprotein (HDL) in mmol/L, the ratio of total cholesterol to HDL, serum glucose in mmol/L, and body mass index (BMI) calculated as weight in kilograms divided by height in metres squared.

In addition, participants were classified as either ‘yes’ or ‘no’ for ‘being a current smoker’ and ‘having had diabetes diagnosed by a doctor’. Weighting to represent the total population was not undertaken since three of the surveys used were convenience samples of particular groups.

Statistical analyses—In all analyses, generalised estimating equations were used to assess the association of one measure of alcohol consumption with each cardiovascular risk factor variable in turn being the dependent variable. Each model adjusted for survey, sex and age group, age and age-squared (in years), ethnicity (Māori or non-Māori), and two interaction terms. The first interaction term, survey

by measure of alcohol, was used to adjust for the differing questionnaires and definitions employed in the surveys. A logistic link was used in those models where the dependent variable was binary. Participants for whom either consumption data or cardiovascular risk factor data were missing were dropped from the models for that analysis, but do appear in other models.

Since the question addressed in this paper relates to the differences between Māori and non-Māori in the association of the indicators of alcohol consumption and individual cardiovascular risk factors, the test for difference was the statistical significance of the second interaction term—between ethnicity and the alcohol consumption indicators. Estimates obtained from the models are reported, with their 95% confidence intervals (using exponential transforms for the logistic models) to describe the associations. Lack of significance of the second interaction term was interpreted as lack of a significant difference by ethnicity and/or lack of statistical power to detect a difference.

Generalised estimating equations were also constructed to test whether BMI and tobacco smoking accounted for some of the association of the alcohol variables with SBP, given the known relationships between these variables.[10–12] Similar models as before were created, each with three additional predictors: BMI, BMI squared, and tobacco smoking. The predictors of interest in these final models were the interaction of the terms for ethnicity and alcohol consumption.

Results

Data for a total of 44,830 participants were reviewed. The five studies and demographic characteristics are described elsewhere,[4] but briefly, 45.4% were women, 29.4% were aged over 50 years, and 29.4% were aged under 35 years; 15.5% identified themselves as Māori.

Table 1. Indicators of alcohol consumption in five New Zealand studies

Variable	Total	Blood Donor	Fletcher Chal/UoA	NZHNS	Sleep	Workforce Diabetes
N	**44,830**	**17,437**	**7,936**	**6,909**	**6,928**	**5,620**
Drinker (%)	**83.6**	**81.0**	**89.1**	**80.7**	**83.3**	**87.4**
Non-drinker (%)	**16.4**	**19.0**	**10.9**	**19.3**	**16.7**	**12.6**
Frequency of drinking (%)						
Q1 (0–9 days per year)	19.4	19.2	19.6	19.7	16.8	–
Q2 (10–34)	21.2	17.2	10.6	25.6	39.2	–
Q3 (35–74)	21.0	17.9	10.6	25.6	39.2	–
Q4 (75–184)	21.3	23.7	28.4	19.9	8.5	–
Q5 (185+)	17.6	22.0	20.3	11.9	8.9	–
Volume pure alcohol drunk on typical occasion (%)						
Q1 (0–<5 grams)17.3	19.4	11.4	19.6	16.9	–	
Q2 (5–<20)	24.3	21.1	14.3	44.6	23.8	–
Q3 (20–<40)	34.2	38.0	40.7	17.1	34.2	–
Q4 (40–<60)	9.2	8.9	8.3	9.5	10.6	–
Q5 (60+)	14.9	12.7	25.3	9.2	14.5	–
Average daily volume pure alcohol drunk (%)						
Q1 (0–<0.2 grams)	20.3	19.4	17.4	19.6	30.4	15.5
Q2 (0.2–<2.0)	18.5	15.1	9.7	33.4	23.8	16.5
Q3 (2.0–<6.0)	18.2	19.9	18.4	21.5	11.9	16.9
Q4 (6.0–<15.0)	23.7	28.1	29.1	15.7	15.8	22.1
Q5 (15+)	19.3	17.5	25.5	9.9	18.1	29.0

Blood Donor = NZ Blood Donors Health Study 1998–1999; Fletcher Chal /UoA = Fletcher Challenge /University of Auckland Survey 1992; NZHNS = New Zealand Health Survey 1997; Sleep = Sleep Survey 1999; Workforce Diabetes = Workforce Diabetes Survey 1988–1990.

A comparison of alcohol consumption data from the five studies is described in Table 1. Most participants reported that they did consume alcohol (overall 83.6% of participants), this varied from 80.7% in the New Zealand Health and Nutrition Survey to 89.1% in the Fletcher Challenge/University of Auckland Study. Considerable variation was seen in the proportions of people classified at different levels of alcohol consumption, as described by frequency of drinking and volume consumed on a typical occasion.

Table 2 shows that Māori and non-Māori participants had similar distributions by sex and age, but there are differences in indicators of alcohol consumption. Unadjusted comparisons indicate that Māori tend to drink alcohol less often (albeit more volume per occasion) than non-Māori.

Table 2. Demographic characteristics and indicators of alcohol consumption in five New Zealand studies, unadjusted

Variable	All N=44,830 100.0%	Māori N=6,926 15.4%	Non-Māori N=37,904 84.6%
Sex and age group (%)			
Men aged 15–34 years	6,404	14.6	14.2
Men aged 35–49 years	10,465	22.7	23.5
Men aged 50–74 years	7,615	13.8	17.6
Women aged 15–34 years	6,770	16.2	14.9
Women aged 35–49 years	8,013	20.3	17.4
Women aged 50–74 years	5,563	12.5	12.4
Non-drinker (%)	**7,367**	**21.1**	**15.6**
Drinker (%)	**37,463**	**78.9**	**84.4**
Frequency of drinking (%)			
Q1 (0–9 days per year)	8,023	25.2	19.4
Q2 (10–34)	8,229	33.9	18.3
Q3 (35–74)	8,118	24.0	19.9
Q4 (75–184)	8,248	11.3	22.8
Q5 (185+)	6,811	5.7	19.6
Volume pure alcohol drunk on typical occasion (%)			
Q1 (0–<5 grams)	6,663	22.1	16.4
Q2 (5–<20)	9,320	14.3	26.3
Q3 (20–<40)	13,132	23.6	36.3
Q4 (40–<60)	3,521	11.4	8.7
Q5 (60+)	5,730	28.5	12.2
Average daily volume pure alcohol drunk (%)			
Q1 (0–<0.2 grams)	8904	27.6	19.0
Q2 (0.2–<2.0)	8104	21.7	17.9
Q3 (2.0–<6.0)	8002	16.5	18.6
Q4 (6.0–<15.0)	10,395	15.5	25.2
Q5 (15+)	8,475	18.7	19.4

Table 3 shows cardiovascular risk factors for Māori and non-Māori. The cardiovascular risk factor indicators were all worse for Māori than for non-Māori.

Table 3. Cardiovascular risk factors in five New Zealand studies, unadjusted

Variable	Information available N=44,830	Māori N=6,926	Non-Māori N=37,904
Lipid profile			
HDL (mmol/L), mean (SE)	8,473	1.21 (0.011)	1.29 (0.004)
Ratio total cholesterol: HDL, mean (SE)	8,471	5.14 (0.060)	5.04 (0.019)
Blood pressure			
Systolic (mmHg), mean (SE)	32,656	126.1 (0.33)	125.8 (0.10)
Diastolic (mmHg), mean (SE)	32,655	80.4 (0.22)	78.0 (0.06)
Lifestyle			
Current cigarette smoker, % (SE)	44,827	44.1 (0.6)	21.4 (0.2)
Body build			
Body Mass Index, mean (SE)	34,720	29.0 (0.10)	26.5 (0.03)
Diabetes related			
Serum glucose (mmol/L), mean (SE)	13,510	5.16 (0.05)	4.92 (0.01)
Reported diagnosis by doctor, % (SE)	37,899	4.0 (0.3)	1.4 (0.1)

SE = Standard error; Crude means and percentages (unadjusted for sex, age or survey); Obese is defined as BMI over 30 if non-Māori and non-Pacific, over 32 if Māori or Pacific.

Results from the regression models are shown in Figures 1–3. Figure 1(a) and 1(b) show the association between lipid profile and alcohol consumption. There was no evidence of a different association for Māori compared to non-Māori. However, there are quite different associations for tobacco smoking (Figure 1[c]). For Māori, low and high levels of frequency of drinking are associated with a lower proportion of current smokers. For non-Māori, there is a consistent slight increase in smoking with frequency of drinking (p=0.0001).

Figure 2 shows that the associations between blood pressure and one of the measures of alcohol intake (volume usually consumed) are clearly different for Māori compared to non-Māori. For non-Māori, increasing volume usually consumed is associated with higher systolic, diastolic, and adjusted systolic blood pressure. In contrast, the pattern is more variable for Māori.

Although Māori have a higher proportion with a diabetes diagnosis, and higher serum glucose levels, no statistically significant differences between Māori and non-Māori were evident in the associations with alcohol consumption (Figure 3).

The associations of body build with alcohol consumption are also shown in Figure 3. Māori have a higher BMI than non-Māori on average; among Māori, the inverse association of BMI with frequency of drinking followed a similar pattern to non-Māori although the gradient was steeper (p<0.0001).

Figure 1. Relationship between lipid profile, tobacco smoking and alcohol consumption, by ethnicity, adjusting for age, sex and survey

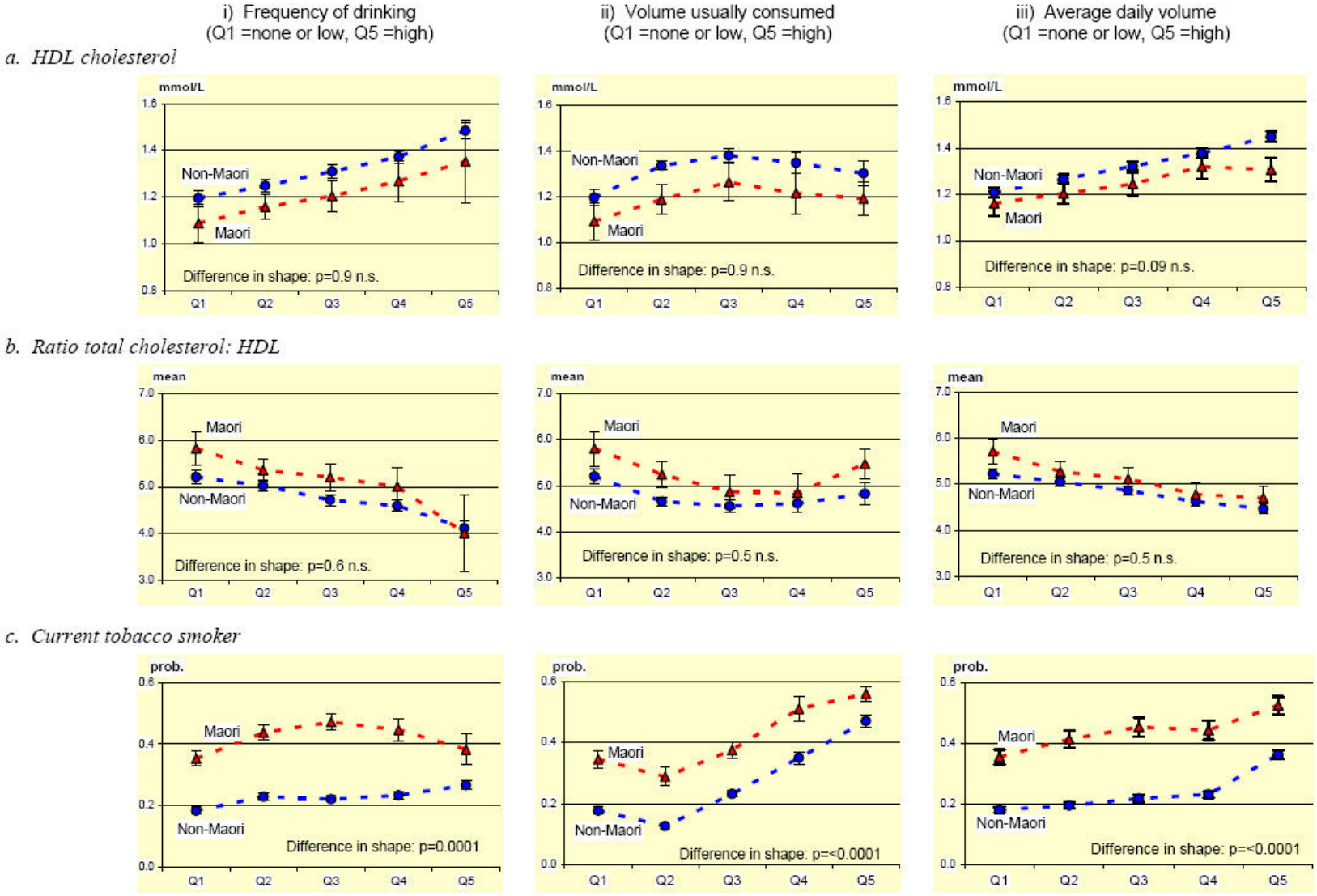


Figure 2. Relationship between blood pressure and alcohol consumption, by ethnicity, adjusting for age, sex and survey

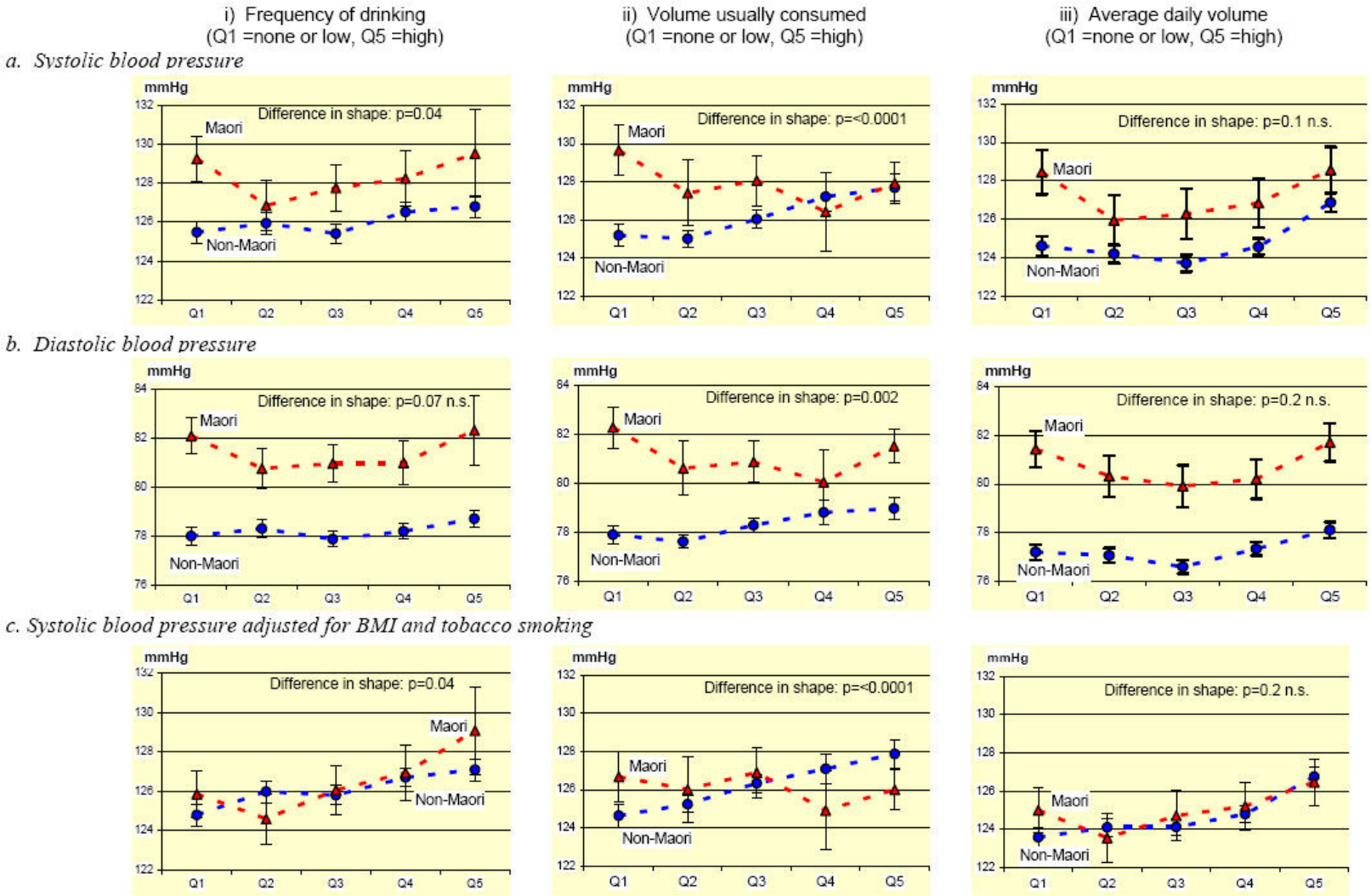


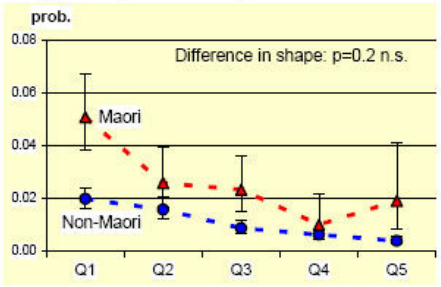
Figure 3. Relationship between diabetes-related factors and alcohol consumption, by ethnicity, adjusting for age, sex and survey

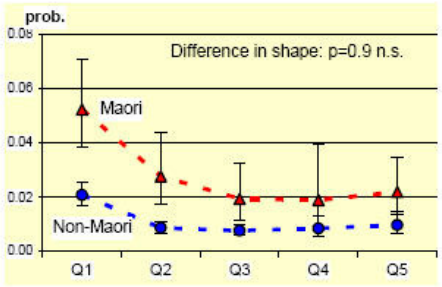
i) Frequency of drinking (Q1 =none or low, Q5 =high)

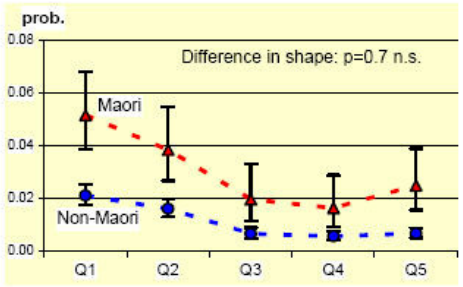
ii) Volume usually consumed (Q1 =none or low, Q5 =high)

iii) Average daily volume (Q1 =none or low, Q5 =high)

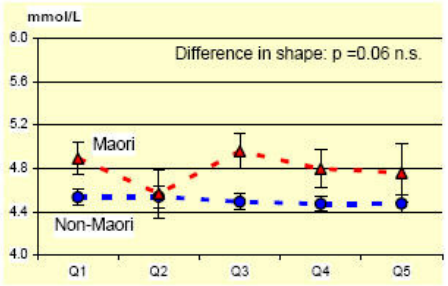
a. Reported diagnosis of diabetes by doctor

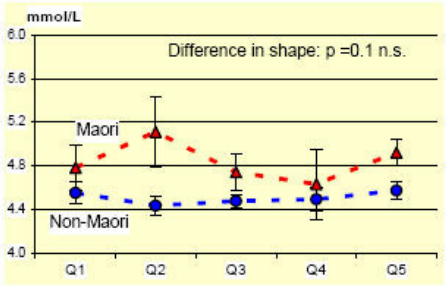


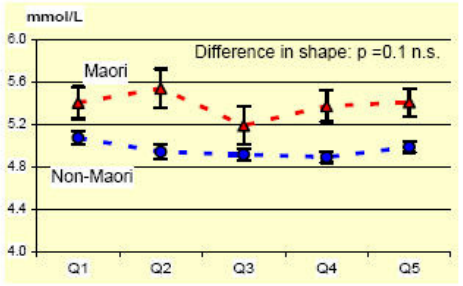




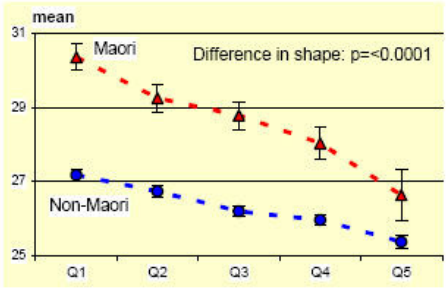
b. Serum glucose

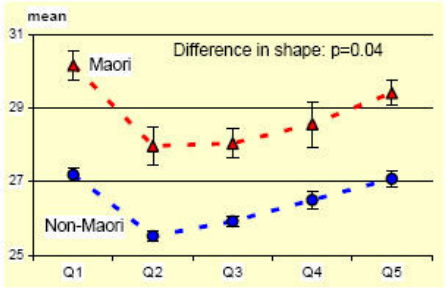


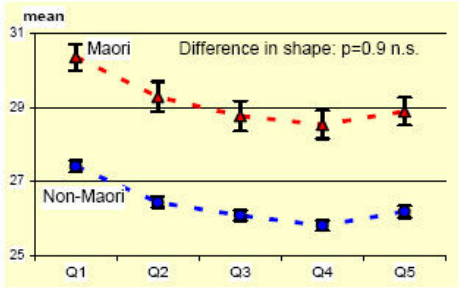




c. BMI







Discussion

This research has been undertaken using a kaupapa Māori framework whereby the study analysis was undertaken from a Māori perspective. This is distinct from other methodologies that may "minoritise" Māori with insufficient data quantity or quality to undertake analyses necessary to inform Māori health development. Where appropriate, this type of analysis enables disparities to be identified and their elimination prioritised. This is consistent with the Treaty of Waitangi.

The data from the five studies combined shows that:

- For all risk factors reported, Māori have higher cardiovascular risk compared to non-Māori. For many of the risk factors, there are clear associations between one or more indicators of alcohol consumption and the risk factor of interest, which do not differ between Māori and non-Māori.
- For the lipid measures used, reported diagnosis of diabetes, and serum glucose level there were no statistically significant differences in their associations with alcohol consumption between Māori and non Māori. However, for systolic blood pressure there is a highly significant difference in volume usually consumed and a significant difference in frequency of alcohol consumption.

These differences remain after adjustment for BMI and tobacco smoking. For BMI, as frequency of drinking increases among Māori, the associated decrease in BMI is more pronounced than in non-Māori. However no differences are apparent for either measure of volume of alcohol consumed. For tobacco smoking, highly significant differences exist in the shape of relationships between ethnicity all three indicators of alcohol used.

In a previous paper, we showed that drinking patterns for Māori and non-Māori were different. Overall, Māori drink less often with higher volumes per occasion, resulting in similar average daily levels of alcohol consumption.[4] Ethnic differences in drinking patterns have also been reported elsewhere. For example, African Americans appear to have higher frequency of heavy drinking occasions and higher proportions of non-drinkers.[13,14]

Ethnic-specific differences in cardiovascular mortality may relate to alcohol consumption.[15] Sempos found that after analysing the relationship between average volume of alcohol consumed and all cause mortality that no J-shaped relationship existed for African Americans whereas it did for whites (Caucasians).[15]

Sempos has also explored the association between average volume of alcohol consumed and coronary artery disease mortality and morbidity in African Americans and whites. In general, average moderate alcohol consumption in African Americans was associated with higher levels of coronary artery disease risk with fewer apparent protective effects when compared to whites.[16]

We found that for some cardiovascular risk factor associations (notably systolic and diastolic blood pressure by volume usually consumed, and tobacco smoking by any of the three measures of alcohol consumption), the associations with alcohol consumption do vary between Māori and non-Māori—results which may support a plausible biological pathway through which ethnic differences in cardiovascular mortality may arise.

Research has consistently found that high alcohol intake is associated with hypertension.[17–19] However there is less certainty regarding the relationship of light to moderate alcohol consumption with blood pressure. Some studies report that low alcohol intake may be associated with decreased blood pressure[18,20] whilst others demonstrate a gradual increase in blood pressure as alcohol intake increases.[17,21] These discrepancies may reflect differences in investigational design, measurement methods and populations.[22]

A previous study undertaken in New Zealand demonstrated a U-shaped relationship between average daily volume of alcohol consumed, and systolic and diastolic blood pressures, in both men and women, with light to moderate drinkers having lower blood pressure than either non-drinkers or heavy drinkers.[23] Our study confirms that relationship for both Māori and non-Māori.

For non-Māori, but not Māori, a more linear relationship with blood pressure exists between both frequency of drinking and volume usually consumed. These findings suggest that ethnic variation in the relationship between usual volume of alcohol consumption and blood pressure may partially explain inconsistencies in the published international literature regarding the relationship of alcohol consumption and blood pressure. Ethnic differences have also recently been reported for black (African American) men in the US compared to whites regarding the association between low-to-moderate alcohol consumption and hypertension.[22]

Several international studies have shown that alcohol consumption is strongly associated with tobacco use. In general, smokers are more likely to consume alcohol than non-smokers.[24–26] Our study shows that among drinkers, as average daily volume and volume usually consumed on a typical occasion increases so does the likelihood of being current a smoker for both Māori and non-Māori. Again, however, a difference exists for Māori, in that (in the upper quintiles of frequency of drinking) the probability of being a smoker decreases, whilst for non-Māori it increases.

In regards to lipids, it has been estimated that approximately half the protective effect of alcohol on coronary heart disease is related to the beneficial effect of alcohol on HDL cholesterol.[27] Therefore our finding (for the lipid measures used) that there were no statistically significant differences in the associations with alcohol consumption between Māori and non Māori is of importance.

Concerning the other two risk factors used in this study the literature is consistent in demonstrating both a small protective association between light-to-moderate alcohol consumption and diabetes (type 2),[28,29] and an inverse relationship between light-to-moderate alcohol consumption and BMI.[30]

These analyses provide indirect evidence that the protective effect of light-to-moderate alcohol consumption on CVD risk (previously demonstrated in non-Māori New Zealanders) may be similar in Māori, as the pattern of association between alcohol consumption measures and the metabolic-related cardiovascular risk factors is generally similar. However Māori/non Māori differences in blood pressure associated with usual volume of alcohol consumed could adversely impact on this protective association given the substantial differences in usual volume consumed per occasion between the two ethnic groups.

Moreover, while the relationship between cigarette smoking and alcohol consumption is behavioural rather than physiological, the strong association between them and the differences in this association by ethnicity may differentially influence the relationship between alcohol consumption and coronary disease in Māori and non-Māori. Until aetiologic studies are conducted among Māori, however, the balance of benefits and harms of alcohol consumption on cardiovascular risk remains uncertain.

Several associations between alcohol consumption and CVD risk factors are of interest that have potential to partially explain some differences in cardiovascular risk between Māori and non-Māori. However, it should be noted that the alcohol-related associations reported here are not necessarily causal, since cross-sectional studies can only describe relationships. Of note are those with tobacco smoking and blood pressure.

There are several potential biases that may occur with our study methodology. The studies we were able to include were conducted during different time periods (1988–2001), and drinking patterns may have changed over that period. The use of a mixed group of cross-sectional studies, only some of which were population-based, and their different instruments for measuring alcohol consumption make it possible that reported means and proportions maybe inaccurate.

We attempted to overcome these potential problems by adjusting for those variations in the models so the interaction terms of interest are less likely to be affected—since most study-specific differences would be absorbed within the study term in the models.

Measurement bias may be possible given that the indicators of alcohol consumption used were self-reported and these biases may differ by ethnicity. Although these results demonstrate comparisons between Māori and non-Māori (regarding the association of cardiovascular risk factors with alcohol consumption), they should not be interpreted as good indicators of the prevalence of risk factors since not all the studies contributing data were representative of the population. Indeed, for some of the variables examined, the proportion of missing data was significantly higher for Māori compared to non-Māori and this may account for some of the significant differences found, or absence of differences that may exist.

In any study comparing ethnic groups there is a possibility that selection biases may be different for one ethnic group than another. It is unlikely that language itself is an issue, as most Māori speak English as their first language. But it is known that response rates in New Zealand to questions are higher if interviewers and interviewees are of the same ethnicity.[31] Unfortunately, we are unable to assess if this is the case for these studies.

Some confounders known to be associated with cardiovascular risk were not available for analysis—for example, socioeconomic status, salt intake, other dietary factors, appropriate levels of treatment, access to medical care, and levels of certain psychosocial stressors. Further, some associations may have arisen by chance alone (many associations are being described and there is considerable statistical power), so it is important that these results are confirmed.

This research is important in addressing issues related to inequalities. Questions about whether the interaction and association of cardiovascular risk factors with

cardiovascular disease differ by ethnicity are of high importance to the academic discourse on inequalities in this country (cardiovascular disease being the leading cause of death for Māori and non-Māori with significant inequalities existing for Māori).

This paper attempts to investigate ethnic differences in the association of alcohol consumption with cardiovascular risk factors and begins to address the current lack of research available regarding these important issues.

Conclusion

There are clear associations for most of the cardiovascular risk factors examined and alcohol consumption. These associations are consistent for Māori and non-Māori, except for blood pressure and cigarette smoking. As the study is hypothesis-generating, further investigation is required for confirmation.

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