

# Correlation between epicardial adipose tissue and body mass index in New Zealand ethnic populations

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## ABSTRACT

**AIM:** We aimed to investigate the correlation between epicardial adipose tissue (EAT) and body mass index (BMI) in different ethnic groups in New Zealand.

**METHODS:** The study included 205 individuals undergoing open heart surgery. Māori and Pacific groups were combined to increase statistical power. EAT was measured using 2D echocardiography.

**RESULTS:** There were 164 New Zealand Europeans (NZE) and 41 Māori/Pacific participants. The mean (SD) age of the study group was 67.9 (10.1) years, 69.1 (9.5) for NZE and 63.5 (11.4) for Māori/Pacific. BMI was 29.6 (5.5) kg/m<sup>2</sup> for NZE and 31.8 (6.2) for Māori/Pacific. EAT thickness was 6.2 (2.2) mm and 6.0 (1.8) mm for NZE and Māori/Pacific, respectively. Using univariate linear regression, BMI showed moderate correlation with EAT in NZE ( $R^2=0.26$ ,  $p<0.001$ ); however, there was no significant correlation between BMI and EAT in Māori/Pacific patients ( $R^2=0.05$ ,  $p=0.17$ ). Using multivariate analysis, BMI remained a significant predictor of EAT thickness in NZE ( $R^2=0.27$ ,  $p<0.001$ ).

**CONCLUSIONS:** BMI was associated with EAT thickness in NZE patients, but not in Māori/Pacific patients. The same level of BMI can carry different connotations of risk in different ethnic groups, with BMI likely being an inconsistent measure of obesity in Māori/Pacific patients.

Obesity is a global epidemic linked to increased cardiovascular risk.<sup>1,2</sup> In particular, increased visceral adiposity is associated with dyslipidaemia,<sup>3,4</sup> insulin resistance,<sup>5,6</sup> hypertension<sup>7</sup> and higher cardiovascular risk.<sup>8,9</sup> Epicardial adipose tissue (EAT) is a type of visceral adipose tissue surrounding the heart; it is defined as the adipose tissue found between the myocardial surface and the visceral pericardium.<sup>10,11</sup> EAT does not function as a mere fat depot; instead, it produces multiple biomolecules and has a vasocrine and paracrine regulating effect on the heart and blood vessels.<sup>11,12</sup> Recently, a number of studies investigating the

relationship between EAT and cardiovascular disease have shown a significant association between EAT and myocardial ischaemia, coronary artery calcification, atrial fibrillation and major adverse cardiac events.<sup>13–18</sup>

Obesity and overweight are currently defined and classified by body mass index (BMI).<sup>19</sup> However, BMI has limited ability to predict visceral adiposity, which has a central role in the development of adverse outcomes associated with obesity.<sup>20–22</sup> Despite its limitations, BMI is the most widely used anthropometric measure of obesity.<sup>23</sup> However, EAT (measured by standard echocardiography) has the potential to be

an accessible measure of visceral adiposity. The relationship between EAT and BMI has intrigued researchers interested in characterising this unique visceral fat depot and a significant positive correlation has been reported between EAT and BMI.<sup>24</sup> In this study we investigate whether the correlation between EAT thickness and BMI is ethnicity-specific in a cohort of New Zealand Europeans (NZE) and Māori/Pacific people.

## Methods

### Study population

This study is a retrospective analysis of a subset of patients from the HeartOtago Heart Tissue Sample Study approved by the local Human Ethics Committee (Approval number: LRS12-01-001) for which prospective enrolment is used. Informed consent was obtained from patients undergoing clinically indicated coronary artery bypass graft surgery (CABG) and/or valve replacement surgery in Dunedin Hospital in the period from 2014 to 2019.

### Clinical and biochemical data

Clinical data were collected by a trained investigator blinded to the echocardiographic analysis. Clinical and demographic data, including comorbidities, medications and relevant medical history, were collected at the recruitment visit preoperatively. Laboratory biochemical data including triglycerides, total cholesterol, low-density lipoproteins and high-density lipoproteins were extracted from patients' records and the most recent preoperative test results were collected.

### Ethnicity

Ethnicity was self-reported; patients were allowed to select more than one ethnicity. In the case of reporting multiple ethnicities, patients' ethnicity was prioritised according to the New Zealand Ministry of Health's ethnicity data protocol:<sup>25</sup> 1) Māori, 2) Pacific, 3) Asian, 4) European. If a patient reported both Māori and/or Pacific and European ethnicity, he/she was allocated to the Māori/Pacific group. Only three potential

participants self-reported their ethnicity as Asian—due to a lack of statistical power, they were not included in further analysis.

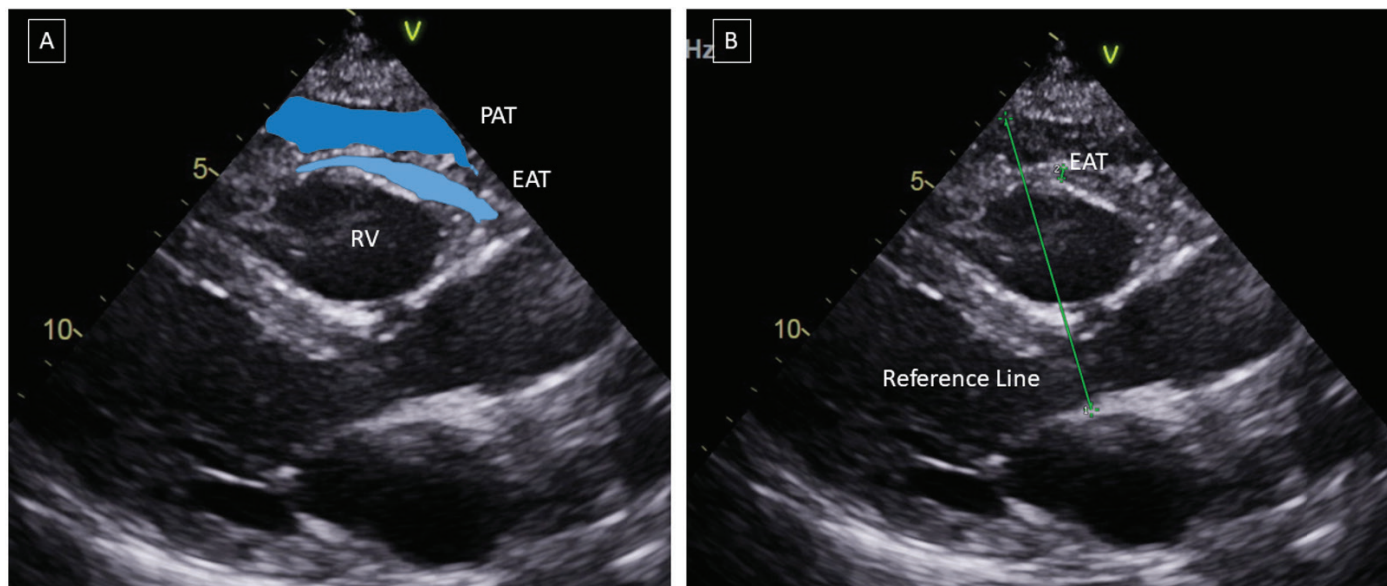
### Anthropometric measurements

Anthropometric measurements were recorded preoperatively. BMI was calculated as weight in kilograms divided by height in square meters. Participants' height was measured on bare feet to the nearest 0.1cm using a wall stadiometer (SECA 216; SECA, Hamburg, Germany). Weight was measured in a light gown to the nearest 0.1kg using a digital weighing scale (SECA 877; SECA, Hamburg, Germany).

### Echocardiographic EAT measurement

Patients underwent comprehensive preoperative echocardiography using commercially available machines (Vivid E9 or E95, GE Healthcare, Chicago, US). Images were digitally acquired and measured according to the recommendations of the American Society of Echocardiography.<sup>26</sup> Standard 2-D, M-Mode and Doppler measurement were conducted.<sup>27</sup> EAT thickness was assessed in the parasternal long axis view using a standardised method based on that of Iacobellis and Willens.<sup>28</sup> Epicardial fat was identified on the right ventricular free wall between the myocardium and the visceral layer of the pericardium; it was measured inner edge (visceral pericardium) to inner edge (RV free wall). EAT thickness was measured at end-systole in three cardiac cycles using the aortic annulus as a reference. A line was drawn between the aortic valve annulus and the right ventricular free wall (Figure 1). The point with maximal EAT thickness within one centimetre on either side of where this line intersected the RV free wall was identified. EAT thickness was measured perpendicular to the RV free wall at this point. Intra-observer and inter-observer variability were calculated from 21 randomly selected subjects with excellent agreement; inter-observer and intra-observer interclass correlation coefficients were both over 95%.

**Figure 1:** A) Parasternal long axis view showing epicardial adipose tissue (EAT), pericardial adipose tissue (PAT) and the right ventricle (RV). B) EAT measurement at end-systole with a reference line drawn from AV annulus and the RV free wall; EAT was measured at the point of maximal EAT thickness within 1cm of where the line met the RV free wall.



### Data analysis

The study data was analysed based on patients' self-reported ethnicity; patients were classified into two groups: NZE or Māori/Pacific. Māori and Pacific groups were combined due to low numbers. Patient characteristics were summarised and reported as mean (SD) for normally distributed variables and frequency (percentage) for categorical variables. Differences between study groups (NZE versus Māori/Pacific) were assessed by two tailed t-test, Chi-squared test or Fisher's exact test as appropriate.

The sample size was calculated based on previously available evidence showing moderate correlation between EAT and BMI;<sup>29,30</sup> our study had 80% power to detect moderate correlation ( $R^2=0.18$ ) between EAT and BMI in the simple regression model. The multiple regression analysis had 80% power to detect effect size ( $f^2$ ) of 0.18 based on previously available data regarding the expected correlation between tested variables and study outcomes after adjusting for other covariates. Alpha of 0.05 was used as the cut-off for significance.

Simple univariate regression was used to test the relation between EAT as a dependent variable and independent variables, including age, sex, ethnicity, BMI,

triglycerides, diabetes, hypertension and LV mass index. Relationships between EAT and other independent variables were tested in the overall study group as well as in each ethnic group separately. Multiple regression was used to further assess the relation between EAT and BMI; the overall model  $R^2$  is reported. For multiple regression models, age, sex, hypertension, diabetes, BMI were a priori selected predictors and were included into the base model irrespective of statistical significance. Other potential predictors included current smoking and LV echocardiographic parameters; predictors that were found to be significantly associated with study outcomes in the univariate analyses were considered as potential independent variables to be included in model. Backward elimination was then used to exclude statistically insignificant predictors from the final model. Testing for ethnicity as an effect modifier on the correlation between EAT and BMI was carried out by including BMI, ethnicity and an interaction term between ethnicity and BMI in a multiple regression model; further adjustment for other covariates was done in the way explained above for fitting the final multiple regression model. For all fitted regression models, model assumptions were assessed; model diagnostics to assess for linearity, normality of residuals, homoscedasticity,

multicollinearity, influential observations and leverage points were conducted. Testing for influential observations and leverage points did not result in the exclusion of any observations. Statistical analyses were conducted using R (R version 3.5.3, R Foundation for Statistical Computing, Vienna, Austria).

## Results

### Characteristics of the study population

Data were analysed for 205 patients: 164 (80%) NZE and 41 (20%) Māori/Pacific (28 Māori and 13 Pacific patients). All patients were undergoing open heart surgery with the majority (78%) having CABG surgery. Characteristics of the study population are presented in Table 1. Overall, the mean (SD) age of the study population was 67.9 (10.12) years and 74.6% of the study population were males. The study population mean (SD) BMI was 30 (5.72) kg/m<sup>2</sup> with Māori/Pacific patients having higher BMI compared to NZE. Māori/Pacific patients were younger and more likely to have diabetes mellitus type 2 in comparison to NZE patients (Table 1). Apart from left ventricular internal diameter in diastole and left ventricular mass index, which were both higher in Māori/Pacific compared to NZE, no significant differences in echocardiographic parameters were found between the two study groups (Table 1).

### EAT thickness and BMI

In the overall study population, a univariate regression model showed that higher BMI was significantly correlated with EAT thickness ( $\beta=0.44$ , 95% CI: 0.32-0.57;  $R^2=0.2$ ,  $p<0.001$ ). However, this relationship was ethnicity-specific: a significant association between EAT thickness and BMI was only seen in NZE ( $\beta=0.55$ , 95% CI: 0.40-0.69;  $R^2=0.26$ ,  $p<0.001$ ); no association was observed in Māori/Pacific patients ( $\beta=0.17$ , 95% CI: -0.08-0.42;  $R^2=0.05$ ,  $p=0.17$ ) (Figure 2). After adjusting for age, sex, diabetes, hypertension and ethnicity using a multivariate regression model, EAT thickness

was still significantly correlated with BMI in the overall study population ( $\beta=0.47$ , 95% CI: 0.34-0.60;  $R^2=0.21$ ,  $p<0.001$ ). In NZE, BMI remained significantly correlated with EAT thickness after adjustment for age, sex, diabetes and hypertension ( $\beta=0.56$ , 95% CI: 0.41-0.71;  $R^2=0.27$ ,  $p<0.001$ ).

Ethnicity modified the association between EAT and BMI with the interaction term showing statistical significance both before ( $\beta=-0.78$ , 95% CI: -1.39– -0.17;  $R^2=0.23$ ,  $p<0.05$ ) and after adjusting for age, sex, diabetes and hypertension ( $\beta=-0.82$ , 95% CI: -1.45– -0.19;  $R^2=0.24$ ,  $p<0.05$ ). BMI was still a significant predictor of EAT after including the interaction term in the final adjusted model ( $\beta=1.99$ , 95% CI: 1.18–2.8;  $R^2=0.23$ ,  $p<0.001$ ).

### EAT thickness and lipid parameters

Triglycerides were modestly, although statistically significantly, correlated with EAT thickness in the study population using a univariate regression model ( $\beta=0.24$ , 95% CI: 0.1–0.4;  $R^2=0.06$ ,  $p<0.001$ ). This association differed according to ethnicity, being significant in NZE ( $\beta=0.32$ , 95% CI: 0.16–0.48;  $R^2=0.1$ ,  $p<0.001$ ) and not significant in Māori/Pacific. Total cholesterol, HDL and LDL cholesterol were not significantly correlated with EAT thickness. Adjusting for age, sex, diabetes and the use of statins showed only modest modification of the association between EAT thickness and triglycerides ( $\beta=0.23$ , 95% CI: 0.08–0.38;  $R^2=0.06$ ,  $p<0.05$ ).

### EAT thickness and other covariates

EAT thickness showed no significant association with age, sex, diabetes, hypertension or current smoking. EAT thickness showed weak association with left ventricular posterior wall thickness (LVPW) ( $\beta=0.15$ , 95% CI: 0.01-0.3;  $R^2=0.02$ ,  $p<0.05$ ), which was found insignificant after adjusting for ethnicity ( $R^2=0.03$ ,  $p=0.08$ ). There was no significant association between EAT thickness and left ventricular internal diameter at end-diastole (LVIDD), interventricular septum thickness (IVS), LV mass index or LV ejection fraction.



**Table 1:** Characteristics of the study population overall and by ethnicity.

	<b>NZE (n=164)</b>	<b>Māori/Pacific (n=41)</b>	<b>Total (n=205)</b>	<b>P-value</b>
Age (years), mean(SD)	69.1 (9.5)	63.5 (11.4)	67.9 (10.1)	<0.001 <sup>a</sup>
<b>Sex</b>				
Male, n (%)	120 (73.2)	33 (80.5)	153 (74.6)	0.34 <sup>b</sup>
Female, n (%)	44 (26.8)	8 (19.5)	52 (25.4)	
BMI (kg/m <sup>2</sup> ), mean (SD)	29.6 (5.5)	31.8 (6.2)	30.0 (5.7)	0.03 <sup>a</sup>
BSA (m <sup>2</sup> ), mean (SD)	2.0 (0.2)	2.0 (0.2)	2.0 (0.2)	0.08 <sup>a</sup>
Smoker, n (%)	15 (9.2)	3 (7.3)	18 (8.8)	0.21 <sup>c</sup>
Ex-smoker, n (%)	71 (43.3)	24 (58.5)	95 (46.3)	
Non-smoker, n (%)	78 (47.6)	14 (34.2)	92 (44.9)	
Type 2 diabetes, n (%)	32 (19.5)	14 (34.2)	46 (22.4)	0.045 <sup>b</sup>
HbA1c (mmol/mol), mean(SD)	40.6 (10.6)	42.7 (15.7)	41.0 (11.8)	0.12 <sup>a</sup>
Cholesterol (mmol/L), mean(SD)	4.6 (1.3)	4.9 (1.5)	4.7 (1.3)	0.11 <sup>a</sup>
Triglycerides (mmol/L), mean(SD)	1.6 (0.7)	1.6 (0.8)	1.6 (0.8)	0.80 <sup>a</sup>
Statin, n (%)	122 (74.4)	33 (80.5)	160 (75.6)	0.42 <sup>b</sup>
Hypertension, n (%)	95 (57.9)	25 (61.0)	120 (58.5)	0.72 <sup>b</sup>
SBP (mmHg), mean (SD)	135.9 (21.4)	137.1 (29.5)	136.2 (23.2)	0.97 <sup>a</sup>
DBP (mmHg), mean (SD)	75.3 (12.7)	78.2 (16.4)	75.9 (13.5)	0.37 <sup>a</sup>
<b>Echocardiography</b>				
IVS (cm), mean (SD)	1.2 (0.2)	1.2 (0.2)	1.2 (0.2)	0.16 <sup>a</sup>
LVPW (cm), mean (SD)	1.0 (0.2)	1.1 (0.2)	1.0 (0.2)	0.05 <sup>a</sup>
LVIDD (cm), mean (SD)	4.8 (0.6)	5.0 (0.9)	4.9 (0.7)	0.03 <sup>a</sup>
LVIDS (cm), mean (SD)	3.3 (0.7)	3.1 (1.0)	3.3 (0.8)	0.65 <sup>a</sup>
EF (%), mean (SD)	54.7 (9.9)	55.3 (12.2)	54.8 (10.4)	0.98 <sup>a</sup>
LV Mass Index (g/m <sup>2</sup> ), mean (SD)	98.8 (27.0)	114.8 (35.3)	101.9 (29.4)	0.003 <sup>a</sup>
<b>Surgery type</b>				
CABG only, n (%)	106 (64.6)	29 (70.7)	135 (65.9)	0.46 <sup>b</sup>
AV/MV surgery only, n (%)	34 (20.7)	7 (17.1)	41 (20.0)	0.6 <sup>b</sup>
Combined surgery, n (%)	24 (14.6)	5 (12.2)	29 (14.2)	0.69 <sup>b</sup>
Rheumatic heart disease, n (%)	2 (1.2)	2 (4.9)	4 (2.0)	0.18 <sup>c</sup>
EAT thickness (mm), mean (SD)	6.2 (2.2)	6.0 (1.8)	6.1 (2.1)	0.61 <sup>a</sup>

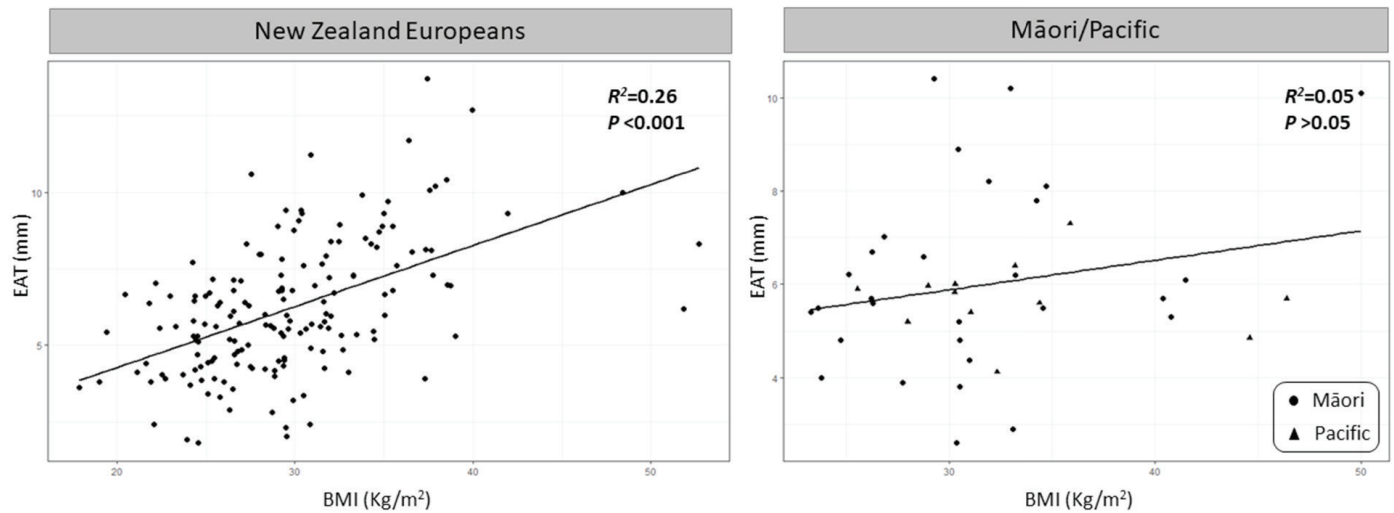
BMI, body mass index; BSA, body surface area; SBP, systolic blood pressure; DBP, diastolic blood pressure; IVS, interventricular septal thickness; LVPW, left ventricular posterior wall thickness; LVIDD, left ventricular internal diameter at end-diastole; LVIDS, left ventricular internal diameter at end-systole; EF, ejection fraction; CABG, coronary artery bypass grafting; AV, aortic valve; MV, mitral valve; EAT, epicardial adipose tissue.

<sup>a</sup> P-value, t-test between NZE and Māori/Pacific.

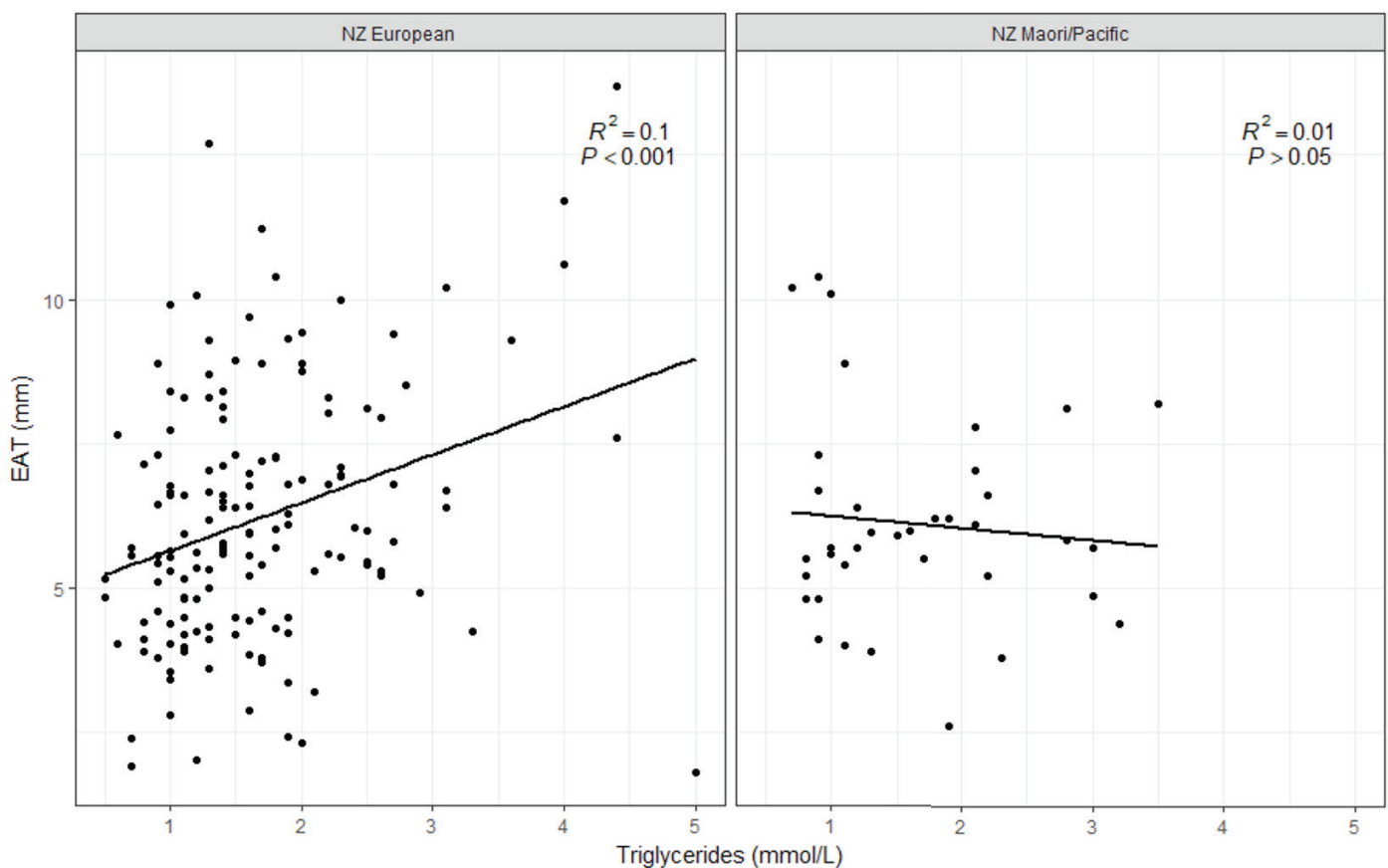
<sup>b</sup> P-value, Chi-square between NZE and Māori/Pacific.

<sup>c</sup> P-value Fisher's exact test between NZE and Māori/Pacific.

**Figure 2:** Association between EAT thickness (mm) and BMI (kg/m<sup>2</sup>) by ethnicity. Line represents univariate linear regression.



**Figure 3:** Association between EAT thickness (mm) and triglycerides (mmol/L) by ethnicity. Line represents univariate linear regression.



## Discussion

Our study showed important ethnic differences in the relationship between BMI and EAT thickness in a sample of high cardiovascular risk patients. Specifically, EAT correlated with BMI and triglycerides in NZE but not Māori/Pacific patients. This study extends on research into the disparity in cardiovascular disease risk between NZE and Māori/Pacific people that demonstrates differences in demographics as one of the underlying causes of higher cardiovascular disease risk in Māori/Pacific;<sup>31</sup> Māori/Pacific patients in our study had higher BMI, lower age and a higher prevalence of type 2 diabetes. Further to these commonly described differences, we demonstrated a moderate, significant association between EAT thickness and BMI in NZE, that was not significant among Māori/Pacific people. The use of BMI as an indicator for cardiovascular disease risk among Māori/Pacific may be misleading and contribute to the disparate outcomes in Māori/Pacific people.

EAT is considered a visceral fat depot; thus, its thickness was hypothesised to correlate with BMI (as a measure of obesity).<sup>10,12,24</sup> However, the association between EAT and BMI is inconsistent in previous research; some studies reported moderate to strong associations<sup>29,32,33</sup> while others showed either a weak or an insignificant association.<sup>24,34,35</sup> In addition, unlike other visceral fat depots, EAT adipocyte size is not related to BMI.<sup>36</sup> In a meta-analysis discussing the association between EAT and measures of obesity, a significant moderate relationship between EAT and BMI was highlighted,<sup>24</sup> which we confirmed in NZE.

Ethnic differences in visceral adipose tissue (VAT) as well as in the association between VAT and BMI have been previously highlighted.<sup>37–40</sup> In a multi-ethnic study, the association between visceral fat, BMI and waist circumference was investigated in a sample of African American, Hispanic and White men and women; the linear relationship slope between BMI and VAT was lowest in African Americans in comparison to Whites and Hispanics.<sup>41</sup> The impact of ethnicity on the association between EAT in particular and BMI has only been investigated in a limited number of studies. Ethnic differences in cardiovascular fat volumes

(EAT and pericardial adipose tissue assessed by electron-beam CT scanning) as well as the association between EAT and measures of adiposity were found between Black and White midlife women in the US.<sup>42</sup> Similarly, ethnic differences in cardiac fat volume, assessed by echocardiography, and its association with BMI was shown in middle aged men of different ethnicities.<sup>43</sup> Along the same lines, a significant difference in the epicardial and pericardial fat thickness, assessed by echocardiography, between African American and non-Hispanic White men was reported.<sup>44</sup> Our findings are in agreement with previously reported findings showing ethnic differences in EAT thickness and its relationship with anthropometric measures.

Additionally, our findings build on the previously reported differences in body composition between Māori and Pacific people in New Zealand and NZE. In New Zealand, Māori were reported to be leaner in comparison to NZE for similar BMI.<sup>44,45</sup> In their study, Rush et al analysed the association between BMI, body fatness, fat distribution as well as other anthropometric and metabolic variables in a population including Māori, Pacific Island, European and Asian Indian adults in New Zealand, finding ethnic differences in body fatness and distribution.<sup>46</sup> This has led to a call to move away from simple BMI measurements as an assessment of adiposity as part of such screening programmes as the B4 School Check.<sup>47</sup>

While a moderate association was previously shown between triglycerides and EAT thickness,<sup>30,48–50</sup> we observed a weak relationship that was only significant in NZE. Although our findings are in agreement with other studies reporting weak or insignificant association between triglycerides and EAT,<sup>34,51</sup> Several other confounding variables may have affected the strength of the relationship between triglycerides and EAT. Our analysis was adjusted for the intake of statins; however, the duration of treatment was not verified before having the lipid profile assessed.

The findings of our study should be interpreted within its limitations. The study was a retrospective analysis of participants, involving the extraction of clinical and demographic data from patients' records.

Combining two separate groups (Māori and Pacific) may have led to obscuring important differences between these groups, and larger studies should ideally attempt to analyse groups separately. However, there was no obvious difference between the two groups (Figure 2). Data regarding anthropometric measurements such as waist circumference, waist to hip ratio as well as quantitative assessment of visceral adiposity and body composition were not available due to the nature of the study. While visceral fat has been previously shown to be correlated with dietary factors<sup>52–54</sup> as well as lifestyle factors,<sup>55,56</sup> the association between these factors and EAT has not been previously studied. Studying the impact of these factors on the association between EAT and BMI in different ethnic groups in New Zealand would necessitate further research including participants at a wide range of cardiovascular risk. Our sample was recruited from patients having open heart surgery, mostly CABG, which represents a special high-risk population. A dedicated formula to calculate the visceral fat from height and weight in Māori/Pacific is not available, to the best of the authors' knowledge; using available formulae which are not ethnicity specific could have led to inaccurate calculations as

differences in body composition between Māori/Pacific and NZE have been previously reported.<sup>44,45</sup> Finally, our echocardiographic measurement of EAT has recognised limitations, with volumetric assessment by MRI or CT likely to provide a more accurate assessment of total epicardial adipose burden. However, echocardiography has the advantage of widespread availability, and EAT measurement is highly reproducible.

Our study is the first to investigate the differences in this relationship between NZE and Māori/Pacific people. The findings of our study corroborate those of other studies that have investigated differences in the relationship between BMI and EAT among other ethnic groups. Importantly, there are inequalities in cardiovascular risk reported among many of the ethnic groups where BMI is not correlated with EAT thickness. We also have shown that the association between BMI and EAT thickness was ethnicity specific; BMI was associated with EAT thickness in NZE patients, but not in Māori/Pacific patients. Our findings, in addition to previous research, indicate that the same level of BMI can carry different connotations of risk in different ethnic groups, with BMI likely being an inconsistent measure of obesity in Māori/Pacific patients.

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#### Competing interests:

Nil.

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