CHAPTER 5

THE ROLE OF DIETARY PATTERNS IN CORONARY ARTERY DISEASE IN URBANISED BLACK SOUTH AFRICANS

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ABSTRACT

*Objective***:** To investigate the role of dietary intake as a risk factor in urbanised black South African CAD patients through the analysis of nutrients as well as of food group consumption patterns.

Design: Dietary habits, including use of ultra-processed foods, of urbanised CAD patients were compared with those of healthy volunteers from an urbanised reference population (PURE).

Setting: Urban areas in Soweto and North West Province, South Africa.

Subjects: Black CAD patients from Charlotte Maxeke Johannesburg Hospital and Chris Hani Baragwanath Hospital (n=91) and apparently healthy volunteers (n=534) from urban group of South African PURE study population at very low risk of developing CVD.

Results: Although the CAD patients consumed significantly higher protein, SFA and MUFA as percentage of energy, their diet was still considered prudent. In general, they had higher micronutrient intakes than the reference group. Both groups, however, met the DRIs for most micronutrients, except for calcium, vitamin C, magnesium and potassium. The CAD patients, furthermore, consumed more fruit and vegetables, dairy products, total meat products and eggs, as well as more ultra-processed foods than did the reference group.

Conclusion: The CAD patients' diets can be considered prudent, but are still not adequate in micronutrients owing to their low intake of foods such as fruit and vegetables and dairy products. Furthermore, they consumed more ultra-processed foods, providing evidence for a link between food processing and disease. The use of the analysis of food groups, including ultra-processed foods, greatly enhances the interpretation of nutrient data.

Key words: CAD, urbanisation, dietary patterns

INTRODUCTION

Ischaemic heart disease has been projected to be one of the three leading causes of the global burden of disease in 2030, and the 4th and 5th cause in middle- and low-income countries respectively[1]. South Africa is also experiencing an increase in CVD, besides still battling the burden of infectious diseases[2]. Coronary artery disease (CAD), specifically, was historically thought to be rare in black South Africans[3,4], but studies are now showing an increase in prevalence especially in urban areas[5,6]. Because CAD was previously rare in this population, little is known about the aetiology of CAD in black South Africans and it may differ from what is known in the Caucasian population[7,8]. Previous work shows, indeed, that the contribution of known risk factors to the development of CVD differs between ethnicities, as do specific cut-offs used to infer increased risk. Black South Africans have, for instance, been shown to have lower lipid and plasminogen activator inhibitor-1 (PAI-1) levels but a higher prevalence of hypertension[9,10], heightened vascular reactivity to stress[11,12] and higher fibrinogen levels[13,14] compared with other ethnicities in South Africa.

The increase in CVD risk and the rising prevalence of CAD in middle- and low-income countries has been ascribed to a worsening diet in populations as they transition from a rural to an urban lifestyle, among numerous other factors [15]. Traditionally, in black South Africans, the diet was low in fat and sugar and rich in fibre[16]. An increase in the intake of total fat and saturated fat has been observed, however, in the North West province of South Africa in the Transition and Health during Urbanisation in South Africans (THUSA) study[17], as well as in other developing countries[18]. Because of urbanisation, the diet now tends to be richer in animal products, refined grains, fats, salt and sugar, and lower in fibre[9].

The role of diet in the aetiology of CAD is complex, particularly within this context of urbanisation. It is difficult and challenging to evaluate the independent role of diet as other changes in lifestyle are happening at the same time, such as decreased physical activity, increased psycho-social stress and multiple other social and economic changes[19]. To evaluate dietary intake accurately is also challenging and complex. The main focus of investigation into dietary intake has changed over the last few decades from examining nutrient intakes only, to assessing food consumption patterns as well. Foods are biochemically complex and contain compounds that interact with each other, necessitating the investigation into the intake of food or food groups and dietary patterns. Very little information is available the role of dietary intakes in CAD development in populations undergoing urbanisation, especially with regard to indicators of dietary intake assessment other than nutrient intakes, such as food groups. Another facet often neglected or overlooked in the evaluation of dietary intakes and eating patterns is the issue of food

processing. Monteiro *et al.* have classified foods into three groups based on the extent and purpose of industrial processing used in their production[20]. In this classification, foods that are ready to eat or ready to heat with little or no preparation, and that are liable to be consumed as snacks or desserts or to replace home-prepared dishes, are classified as ultra-processed food products. According to the authors, diets that are high in ultra-processed foods are intrinsically nutritionally unbalanced and harmful to health, although studies linking ultra-processed foods to health outcomes are still lacking[21].

The aim of this study is, therefore, to investigate the role of dietary intake through the analysis of nutrients as well as of food group consumption patterns, as a risk factor in urbanised black South African CAD patients. A previous investigation into the role of diet in this population with documented CAD looked at nutrient intake only and was performed in a much smaller study population (40 cases compared with 20 controls)[22]. This study aims, in addition, to include a comparison of food group consumption in a larger number of black CAD patients (n=91), with that of an apparently healthy reference group (n=534) at low risk for developing CVD within the next 10 years, from the South African Prospective Urban and Rural Epidemiological (PURE) study population.

MATERIALS AND METHODS

Ninety-one black patients (58 males, 33 females) with documented CAD were included in this study. Thirty-three of the CAD patients were participants in a case control study conducted at Charlotte Maxeke Johannesburg Hospital (study design previously reported)[23], and 58 patients attended the Chris Hani Baragwanath Hospital in Soweto, South Africa, as part of the Heart of Soweto Study. This is a large-scale study of emerging heart disease in Soweto, South Africa. The design of this study has been previously reported[24]. The patients from both these groups lived in Soweto and surrounding areas. Ethical approval for the Heart of Soweto study (M050550) and the case control study (No: 010102) was obtained from the Human Research Ethics Committee of the University of Witwatersrand, Johannesburg. The patients were included in the studies after signing informed consent (Annexure B). Details regarding the collection of socioeconomic information, anthropometric measurements, blood, and blood pressure readings have been reported previously for both groups of patients[23,24].

For the reference group, 535 apparently healthy volunteers (202 men and 332 women) from an urban community with a similar socio-economic and demographic background were selected from the South African PURE study cohort. The volunteers in the reference group

had a low risk (<5%) for developing heart disease within the next 10 years, according to the Reynolds Risk Score[25,26]. The PURE study was approved by the Ethics committee of the North-West University, South Africa (No. 04M10). The subjects signed informed consent before commencement of the study, after the study was explained to them in their home language (Annexure F). All data were treated confidentially and all analyses were performed with coded data. Details regarding the design of the PURE study, the gathering of socioeconomic information, anthropometric measurements and blood pressure readings and the collection of blood, as well as some dietary data have been reported previously[27-29]. All three studies fully conformed to the principles outlined in the Declaration of Helsinki.

A culturally sensitive quantified food frequency questionnaire (QFFQ) (Annexure D) was completed for both the reference group and the CAD patients. The QFFQ was completed at least three months post-diagnosis at the Charlotte Maxeke Johannesburg Hospital and at the time of diagnosis at Chris Hani Baragwanath Hospital. The QFFQ with good reproducibility[30] was previously developed[31] and validated in this population, using seven-day weighed records and biomarkers[32]. Portion sizes were estimated using food portion photographs[33], appropriate utensils and containers and examples of specific foods. Portion sizes were reported in household measurements and converted to weights using standard tables[34]. The QFFQ was completed for foods eaten over the previous 30 days. The food intake was coded according to the South African Food Composition Database System of the South African Medical Research Council and then used to calculate the nutrient and food group intake per day[35]. Mean and median intake of the food groups was calculated for the total population and not for consumers only.

Statistical analysis. Data were analysed using the SPSS (Statistical package for Social Sciences, version 20) software package. A p-value ≤ 0.05 was regarded as statistically significant. Normally distributed variables are reported as mean (95% confidence interval), and not normally distributed data as median [25^{th}–75^{th} percentile]. Data were log transformed to improve normality, after which t-tests were done, unless data were still not normally distributed, in which case Mann-Whitney U tests were done. Two binary logistic regression models for nutrients and food groups separately were used to determine whether there were any nutrients or food groups that significantly increased the odds of being a case or in the reference group. All nutrient and food group variables were standardised before being entered into the regression models. All nutrients and food groups were entered into the logistic regression and, with the use of the forward stepwise approach, the model that gave the lowest chi-square and highest p-value in the Hosmer and Lemeshow goodness of-fit-test, together with the best prediction ability (97.4% for nutrient model and 90% for food

group model) was selected to identify which nutrients most strongly associated with the CAD or reference group. Age and gender were included in both models. The two CAD patient groups were initially analysed separately and compared. No differences were found between the two groups and their results were combined in further analysis.

RESULTS

Table 1 provides the general characteristics of the reference group compared with the CAD patients. There were more men (65% as against 35%) in the CAD group and the median age of the CAD group was significantly higher. For this reason, age and gender were adjusted for in all further statistical comparisons. The reference group had significantly higher blood pressures (although still within the normal ranges) than the CAD group, probably because the CAD group was receiving treatment for hypertension. After adjusting for age and gender, the total cholesterol did not differ significantly between the two groups. However, the LDL-cholesterol and triglycerides of the CAD group were significantly higher than in the reference group and HDL-cholesterol was significantly lower in the CAD group. Additionally, C-reactive protein (CRP) and BMI were both significantly higher in the CAD group.

The median nutrient intakes of the PURE reference group and the CAD patients are compared in Table 2. After adjusting for age and gender there was no significant difference in total energy intake between the two groups. The CAD group consumed significantly higher protein, saturated fat and monounsaturated fat as percentage of energy, while the PURE reference group consumed significantly higher PUFA as percentage of energy and had a higher median alcohol intake. No differences were seen between the two groups in total fat or carbohydrate as percentage energy, or in total dietary fibre or cholesterol intake. On a micronutrient, level the CAD patients consumed significantly more of all micronutrients, except for magnesium, copper, vitamin A, riboflavin and vitamin B12, where no differences were seen. Although the intakes in the CAD patients were higher, both groups met the DRIs for most micronutrients, except for calcium, vitamin C, magnesium and potassium, which were below the recommended adequate intake (AI) or estimated average requirement (EAR) in both groups[36,37-39].

Table 3 gives the median intake of foods and food groups. After adjusting for age and gender, the CAD group consumed significantly higher amounts of fruit and vegetables (although still less than the recommended 400g), dairy products, total meat products and eggs, as well as more snacks and sweets and chocolates. The PURE reference group, on

the other hand, consumed significantly more plant-based oils, alcohol and carbonated cold drinks containing sugar. When classifying the foods and food groups into the ultra-processed food product group recommended by Monteiro *et al.*[20] the CAD group consumed significantly higher amounts (except for processed meats) of all foods falling into this category, except for carbonated cold drinks which was lower. When comparing the alcohol consumption of the two groups in Table 4, it is seen that the majority of the CAD population (67%) did not drink alcohol, 22% consumed alcohol moderately and only 11% were considered heavy drinkers. In the reference group, however, 57% of the group were classified as moderate or heavy drinkers.

Table 1: General characteristics of the PURE reference group compared with the coronary artery disease (CAD) patients

Parameter	**Reference group**	**CAD**	**p-value***
Gender (men/women)	202/332 (38%/62%)	58/33 (65%/35%)	<0.0001
Age (years)	44.0 [40 – 50]	56.5 [49.0 – 64.0]	<0.000001
Systolic blood pressure (mmHg)	129 (127; 130)	126(121;131)	0.006
Diastolic blood pressure (mmHg)	85.8(84.7; 86.9)	75.5(72.4;78.6)	<0.000001
Total cholesterol (mmol/L)	4.98(4.86;5.10)	5.39 (4.98;5.81)	0.151
LDL cholesterol (mmol/L)	3.13(3.03;3.23)	3.90(3.52;4.29)	<0.000001
HDL cholesterol (mmol/L)	1.46[1.12-1.97]	1.13[0.88-1.39]	<0.000001
Men	1.59[1.18-2.21]	1.08[0.88-1.39]	<0.000001
Women	1.41[1.08-1.84]	1.15[0.97-1.41]	0.02
Triglycerides (mmol/L)	1.06 [0.81-1.51]	1.50[1.1-2.47]	<0.00001
C-Reactive protein (mg/L)	2.32[0.73-8.29]	6.0[3.0-31.0] (47)	<0.000001
Body mass index (kg/m^2)	22.3[19.2-28.2]	28.9[25.0-32,0]	<0.000001
Men	19.5[18.1-21.8]	27.0[24.5-31.0]	<0.000001
Women	25.9[21.3-31.7]	31.0[26.6-36.3]	0.03

Normally distributed data reported as: mean (95% CI) and log transformed data reported as median [25th – 75th percentile]

*ANCOVA p-value adjusted for age and gender.

Table 2: Nutrient intakes of PURE reference group compared with those of the coronary artery disease (CAD) patients

Parameter	Reference group n = 534	CAD n=91	p-value*
Total energy (kJ)	8619[6238-11625]	8839[7082-11940]	0.438
Protein (% of TE)	12.5(12.3;12.6)	14.5(14.0;15.1)	0.000000
Plant protein (% of TE)	6.24(6.14;6.33)	6.28(5.98;6.57)	0.78
Animal protein (% of TE)	5.82(5.63;6.02)	7.93(7.28;8.58)	0.000000
Total fat (% of TE)	26.1(25.6;26.7)	25.0(23.8;26.3)	0.4103
SFA (% of TE)	6.47[5.14-7.82]	7.0[6.0-9.0]	0.0002
MUFA (% of TE)	7.17[5.70-8.68]	8.0[7.0-10.0]	0.0007
PUFA (% of TE)	7.45(7.24;7.65)	5.45(4.98;5.85)	0.000000
Carbohydrates (% of TE)	56.1(55.5;56.7)	57.0(55.4;58.6)	0.2598
Added sugar (% of TE)	7.53[5.09-10.1]	9.0[5.0-13.0]	0.143
Alcohol (g)	3.43[0.0-20.7]	0.00[0.0-2.63]	0.00013
Cholesterol (mg)	218[139-326]	267[172-361]	0.26
Total fibre (g)	21.5[14.4-30.6]	23.8[18.0-32.9]	0.069
Calcium (mg)	386[276-584]	474[326-666]	0.005
Iron (mg)	14.65(14.0;15.3)	18.5(16.8;20.2)	0.00016
Magnesium (mg)	338[232-453]	336.6[253.1-422.1]	0.65
Potassium (mg)	1932[1299-2557]	2372.1[1792-3080]	0.0003
Zinc (mg)	10.7[7.30-14.7]	15.04[11.3-19.0]	0.000000
Copper (µg)	1.32[0.92-1.78]	1.34[1.04-1.79]	0.202
Manganese (mg)	2193[1566-3301]	2564[1775-3704]	0.055
Vitamin A (RE)	787[480-1414]	893.4[672;1324]	0.145
Vitamin C (mg)	30.9[17.6-54.3]	38.4[26.3-72]	0.0002
Thiamine (mg)	1.57[1.09-2.34]	1.95[1.55-2.67]	0.00068
Riboflavin (mg)	1.39[0.93-1.95]	1.53[1.11-2.17]	0.213
Niacin (mg)	15.6[10.7-21.4]	22.7[18.5-31.0]	0.000000
Vitamin B6 (mg)	1.48[1.0-2.3]	3.88[2.68-5.13]	0.000000
Folate (µg)	377[272-548]	454.1[336.4-586.9]	0.026
Vitamin B12 (µg)	4.26[2.30-6.78]	4.23[2.60-6.44]	0.89
Pantothenic acid (mg)	4.36[2.96-5.98]	5.75[4.04-7.12]	0.00002

TE, total energy; Normally distributed data reported as: mean (95% CI) and log transformed data reported as median [25^{th} – 75^{th} percentile]; *ANCOVA p-value: adjusted for age and gender

Table 3: Food and food group intake of the PURE reference group compared with that of the coronary artery disease (CAD) group

Parameter (grams)	Reference group n = 534	CAD n = 91	p-value†
Starch group (Total)	637[466–958]	671[487-1021]	0.77
• Cooked porridge and starchy foods	500[354-841]	524[364-916]	0.93
• Breakfast cereals*	0[0-0]	0[0-3.57]	<0.000001
• Bread	103[58.6–157]	120[51.4-139]	0.54
Snacks: Sweet and savoury*	5.79[0-23.57]	19.6[5.36-46.2]	<0.000001
Fruit and Vegetable (Total)	169[103 – 279]	226[134-386]	0.0003
• Vegetable group (Total)	69.6[46.1-104]	103[77.1-171]	<0.000001
• Starchy vegetables	25.7[11.6-54.1]	3.57[0-17.1]	<0.000001
• Fruit group (Total)	63.6[24.3-119]	72.4[30-210]	0.01
Fruit, all fresh fruits	55.4[23.6–103]	53.6[22.9–124]	0.51
Dried fruit	0[0–0]	0[0–0]	0.02
Canned fruit and dried fruit (with sugar)*	0[0–0]	0[0-2.14]	0.2
Fruit juice*	0[0–0]	0[0-8.93]	<0.0001
Total legume group	4.93[0-21.4]	0[0–14.0]	0.23
Total nut and seed group	0[0-5.71]	1.0[0-9.0]	0.13
Dairy group (Total)	119[62.9-208]	191[82.1-309]	<0.000001
• Milk	117[62.7-206]	187[80.0-294]	<0.0001
• Cheese, all types*	0[0–0]	2.14[0.0-8.57]	<0.0001
Non dairy creamer	0[0–0]	0[0–0]	0.10
Ice cream, all types*	0[0–0]	0[0–4.46]	0.01
Eggs	14.9[0-29.7]	14.9[7.14-42.9]	0.02
Meat (total)	67.6[39.6-110]	95.7[63.6–146]	<0.0001
• Red meat group	28.7[24.1-33.1]]	27.3[23.3–31-.9]	<0.000001
• Chicken group	30.1[15-47.4]	40.7[23.7-68.6]	0.04
• Organ meats	12.9[3.57-22.9	2.14[0-8.71]	<0.000001
• Processed meat products*	13.4[7.57-24]	17.1[7.14-32.3]	0.33
Fish, all types	7.4[1.4-17.14]	11.4[4.29-22.1]	0.06
Plant fat-based spread, oil and dressings	16.4[10.7-26.14]	11[1.43-26.4]	<0.000001
Sugar and sugar products			
• Added sugar	22.1[12.5-38.0]	19.7[7.7-42.0]	0.62
• Chocolates and sweets*	2.86[0.0-7.29]	14.3[1.71-27.9]	0.000000
• Carbonated cold drink*	31.4[0.0-50.0]	7.14[0.0-71.4]	0.000000
Alcoholic drinks	107[0.0-714]	0.0[0.0-72.9]	<0.000001

Log transformed data reported as median [25th – 75th percentile]

†ANCOVA p-value: adjusted for age and gender

* Ultra-processed food products[20]

Table 4: Alcohol intake

Alcohol intake	PURE reference group			CAD		
	Men	Women	Total	Men	Women	Total
	n=202	n = 332	n=534	n =	n = 33	n=91
None	56	178	234(44%)	29	32	61 (67%)
Moderate*	50	76	126 (24%)	19	1	20 (22%)
Heavy†	96	78	174 (33%)	10	0	10 (11%)

*Moderate alcohol intake defined as ≤15g alcohol per day for women and ≤30g alcohol per day for men

†Heavy alcohol intake was defined as >15g alcohol per day for women and >30g alcohol per day for men

The nutrients and food groups were entered into two separate binary logistic regression models to determine whether there were any intakes of nutrients or food groups that were clearly associated with either the CAD or the reference group (Table 5). In the nutrient model, an increase of 1 standard deviation in niacin had the largest effect on increasing the odds of being in the CAD group, followed by potassium, zinc, manganese, and then age. One standard deviation increase in copper, PUFA as percentage energy, pantothenic acid and vitamin B12, increased the odds of being in the reference group.

In the food group model, male sex and increased age had the largest effect on increasing the odds of being in the CAD group. One standard deviation increase in the intake of meat, dairy, chocolates and sweets and fish significantly but modestly increased the odds of being in the CAD group, while alcohol consumption and plant-based spreads and oils significantly increased the odds of being in the reference group.

DISCUSSION

Although the prevalence of CAD is increasing in populations in developing countries, such as is being seen in the urbanising black South Africa population, the aetiology of CAD in these populations is largely unknown. Furthermore, the aetiology of CAD in these types of populations is exceedingly complex because urbanisation and improved economic status are associated with dietary changes as well as changes in various aspects of lifestyle, such as physical activity and psychosocial stress. This study investigated the role of diet in CAD by comparing the dietary intake (nutrients and food groups) of patients with documented CAD with that of an apparently healthy reference group from a similar urbanised setting.

Table 5: Use of logistic regression models to distinguish between dietary patterns of CAD patients and the healthy reference group

Variable*	P value	β (95%CI)
Nutrient Model	0.000	
Age	0.000	5.25 (2.49-11.1)
Potassium	0.000	67.3 (9.88-458)
Zinc	0.000	66.1 (9.16-477)
Copper	0.003	0.05(0.01-0.36)
Manganese	0.000	31.4(7.55-131)
Niacin	0.000	110.8(17.7-694)
Vitamin B12	0.013	0.29(0.11-0.77)
Pantothenic acid	0.003	0.16(0.05-0.53)
PUFA %E	0.000	0.09(0.04-0.22)
Food Group Model		
Gender	0.000	5.71(2.93-11.15)
Age	0.000	3.53(2.55-4.90
Dairy	0.000	1.59(1.24-2.05)
Total Meat group	0.000	1.93(1.43-2.61)
Fish group	0.007	1.42(1.10-1.83)
Plant based oils and spreads	0.007	0.51(0.35-0.74)
Chocolate and sweets	0.001	1.53(1.20-1.96)
Alcoholic drinks	0.000	0.22(0.11-0.43)

*Variables were standardised before being entered in the logistic regression models

A limitation of the study is the use of the Reynolds Risk score in selecting the reference group, since this score has not been developed for nor validated in the black African population. However, as no such risk score exists for black Africans, the Reynolds risk score was selected because, apart from the traditional Framingham risk score factors, it also includes CRP, increased levels of which are associated with increased poverty as well as with non-white ethnicities, as is the case in the PURE reference population[40]. The Reynolds Risk Score has been shown, furthermore, to reclassify African Americans into a risk category that is different from that of the Framingham Vascular Disease Risk Score, using CRP

testing[41]. It should also be noted that the risk score was not used to calculate absolute risk but purely to stratify the PURE population into CVD risk score categories, in order to select an appropriate reference group with a low risk (<5%) of developing CVD within the next 10 years. Therefore, although the possibility exists that the reference group may still develop CVD, since they were younger than the CAD group, the likelihood of this happening within the next 10 years is predicted to be small. The possibility cannot be excluded, however. Another limitation of this study is the possibility that the CAD patients from Charlotte Maxeke Johannesburg Hospital may have received dietary education in the days or weeks prior to the administration of the QFFQ, since their dietary intakes were recorded three months post-event. There were, however, no significant differences in intakes between the two CAD groups.

The results of this study indicated that there were some important differences in the diets of the CAD patients when compared with those of the reference group. The CAD patients consumed more proteins, SFA and MUFA as percentage energy than did the reference group, although these intakes were still well within the recommended guidelines for the prevention of CVD. They also had higher intakes of all micronutrients in comparison with the reference group. Additionally, the median micronutrient intakes of both groups were higher than the respective EAR or AI except for calcium, vitamin C, magnesium and potassium, which were lower in both groups. The CAD group, furthermore, consumed more fruit and vegetables (both groups consuming far less than the recommended 400 g per day, however), dairy, meat, eggs, snacks and sweets and ultra-processed foods, while the reference group consumed more plant-based oils, alcohol and carbonated drinks containing sugar. While there was no difference in total energy intake between the two groups, the CAD patients did, however, have a significantly higher mean BMI (29 kg/m^2) than the reference group (22 kg/m^2). Pearson correlations (not shown) indicated that the only nutrients that correlated, albeit weakly, with BMI, were total fat ($r=0.22$, $p<0.01$), SFA ($r=0.24$, $p<0.1$) and MUFA ($r=0.23$, $p<0.01$), expressed as percentage energy, intakes of which were higher in the CAD group. It is therefore possible that while the obesity observed in the CAD group may have contributed to the development of CAD, the obesity itself, rather than overt differences in nutrient intakes, is the result of other metabolic or lifestyle factors. It is known that adipose tissue is not simply a passive storehouse for fat but an endocrine organ that is capable of synthesising and releasing into the bloodstream an important variety of peptides and nonpetide compounds that may play a role in cardiovascular homeostasis[42].

Another possible explanation for the increased BMI observed in the CAD group may be the higher intake of ultra-processed food. The CAD group, for example, consumed seven times

more chocolates and sweets than the reference group. Although there is as yet no evidence that the ultra-processed food group is linked to obesity, higher intakes of the individual components have found to be associated with obesity[43]. This classification of foods brings added value to the assessment of dietary patterns, enabling them to be evaluated in a more holistic manner and providing a more complete picture of these patterns. The classification developed by Monteiro *et al.*[20] provides a tool for assessing the overall pattern of processed food production and consumption instead of focussing only on the effect of specific types of processed foods. This study is also one of the first to link ultra-processed foods to a health outcome. A previous study found that high consumption of ultra-processed foods was associated with the prevalence of metabolic syndrome in Brazilian adolescents[44]. The CAD group, although consuming higher amounts of some of the foods and nutrients linked to the protection against CAD, such as fruit and vegetables, calcium, potassium and vitamin C, also consumed more of the ultra-processed foods. The authors also allude to the fact that the inclusion of breads in the ultra-processed category has received criticism[20]. According to the definition of the ultra-processed group, bread is classified into this group. For this study, bread was not classified as an ultra-processed food, as in this population, bread forms an important part of the diet and the South African Department of Health's Food Fortification programme.

The logistic regression models indicated that the nutrients that had the strongest ability to increase the odds of being in the CAD group were niacin, potassium, zinc and manganese. The results of the nutrient model are supported by the food group model which indicated that intake of meat, dairy, chocolates and sweets, and fish increased the odds of being in the CAD group. The main food source of niacin is meat, for potassium it is meat and dairy, for zinc it is meat and fish and for manganese, cocoa and chocolate[35]. It is possible, therefore, that it is not the individual nutrients themselves that increase the odds of being in the CAD group, but that the associations were found because these nutrients are abundant in those food groups of which high intakes can increase the odds of being in the CAD group, through mechanisms other than acting as a good source of these nutrients.

The same picture emerges when looking at the nutrients and foods that increase the odds of being in the reference group. Copper, pantothenic acid and vitamin B12 are all abundant in organ meat, the intakes of which were significantly higher in the control group (Table 3). This is not reflected, however, in the food group logistic regression model, as organ meat was not entered separately in the model; only total meat was included. Plant-based spreads and oils are, furthermore, good sources of PUFA, and were both found to increase the odds of being in the reference group. Consumption of alcoholic drinks also significantly increased the odds

of being in the reference group. The protective effect of alcohol consumption on CVD, especially in moderate quantities, is well known[45,46] and these results indeed illustrate the difference in alcohol consumption between the two groups. The results of the logistic regression models illustrate the importance of including dietary intake measures other than nutrient intakes alone in order to better determine the relationship between dietary intake and disease.

In conclusion, the CAD patients consumed more SFA and MUFA as percentage energy, which may have contributed, at least in part, to the higher level of obesity observed in the CAD patients, probably contributing to the development of CVD through the deleterious effects of adipose tissue. Despite these intakes being higher than in the reference group, they were well within the recommended guidelines for the prevention of CVD. In addition, the CAD patients consumed more micronutrients than the reference group did, which, paradoxically, is thought to protect against the development of CVD. CAD patients, moreover, consumed more meat, dairy, fish and ultra-processed foods than the reference group did. The analysis of food groups, including ultra-processed foods, greatly enhances the interpretation of nutrient data and it is strongly recommended that nutrient intakes alone should not be used to determine the relationship between dietary intakes and disease. The CAD patients' diets can be considered prudent, but are still not adequate in micronutrients owing to their low intake of foods such as fruit and vegetables and dairy. Prevention programmes for the black African population in transition should be targeted at improving the adequacy of the diet by increasing the intake of foods rich in vital micronutrients known to protect against the development of CVD.

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