Prevalence of and contributing factors to dyslipidaemia in low-income women aged 18-90 years in the peri-urban Vaal region

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Abstract

Objective: Determining the prevalence of dyslipidaemia and examining dietary and other contributing factors, namely hypertension, overweight and obesity, as well as abnormal blood lipid levels in women aged 18-90 years.

Design: Cross-sectional baseline survey study.

Setting: Peri-urban Vaal region.

Subjects: Seven hundred and twenty-two randomly selected black women in four purposively selected settlements in the Vaal region.

Outcome measures: Measurements included dietary intake (24-hour recall), anthropometric (weight and height), and blood pressure and biochemical indices (lipid profile) with venous blood samples. Data analyses included descriptive statistics, t-tests and regression analyses.

Results: A large percentage (34.3%) of the women (aged 18-90 years old) was dyslipidaemic. The majority of women in the nondyslipidaemic group were overweight (52.6%). 86.2% of the women in the dyslipidaemic group were obese. The total fat intake was 20% of total energy intake in both groups. No significant differences were observed between the dietary fat intake variables between the groups. Body mass index (BMI) (ß = 0.554, p-value = 0.000), age (ß = 0.419, p-value = 0.000), education (ß = 0.250, p-value = 0.000), total energy intake (ß = 0.105, p-value = 0.006) and total fat intake (ß = 0.092, p-value = 0.018) were predictors of dyslipidaemia in these women.

Conclusion: Dyslipidaemia was prevalent. High triglyceride and low high-density lipoprotein cholesterol levels were the most frequent abnormalities. Although positive associations existed between the prevalence of dyslipidaemia and the known risk factors of cardiovascular disease, such as ageing, hypertension, obesity and an abnormal lipid profile; BMI, age and education were the main predictors of dyslipidaemia.

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Introduction

Dyslipidaemia is classified as a significant risk factor for cardiovascular disease (CVD) and is characterised by having one or more of the following conditions: serum triglycerides (TGs) ≥ 2.26 mmol/l, total serum cholesterol (TC) ≥ 6.20 mmol/l, high-density lipoprotein cholesterol (HDL-C) < 1.03 mmol/l and low-density lipoprotein cholesterol (LDL-C) ≥ 3.36 mmol/l if the Framingham 10-year risk score is high, or LDL-C ≥ 3.36 mmol/l. if the Framingham 10-year risk score is intermediate, or LDL-C ≥ 4.14 mmol/l if the Framingham 10-year risk score is low.[1,2] CVD exists largely in developed countries, but in the past decade it has also become a problem in the developing world.[3] Globally, CVD was the main cause of death and disability in 2010.[1,4] This is also true in South Africa where CVD is responsible for 17% of all deaths. It is further estimated that 5.5 million South Africans who are older than 30 years are at risk of developing CVD. This may be because of raised total serum cholesterol levels (> 5.20 mmol/l),[5] as well as hypertension (blood pressure of 140/ 90 mmHg),[6] which are present in 22% and 27% of men and women who are older than 15 years respectively.[7] Dyslipidaemia, hypertension and obesity [body mass index (BMI) ≥ 30 kg/m^2][8] are well-known risk factors for CVD.[1] Total dietary fat, including saturated, polyunsaturated, monounsaturated and trans-fatty acids, dietary cholesterol, and saturated fatty acid (SFA) intakes (C14, C16 and C18, except in milk and coconut where SFA ranges from C4-C18)[4] are associated with obesity, undesirable serum cholesterol levels and coronary heart disease. However, it is important to consider both the quantity and quality of dietary fat. Therefore, it is essential that the dietary fat quality guidelines (which aim to reduce the incidence and prevalence of chronic lifestyle diseases) are followed.[9,10]

Although gender and CVD risk factors such as hypertension, metabolic syndrome, tobacco usage, overweight and obesity,

diabetes and abnormal blood lipid levels have been studied in recent years in other countries such as Latin America,[11] there is limited recent available information on dyslipidaemia, CVD and women in South Africa. This study was undertaken in low-income women of the Vaal region, a peri-urban area with an unemployment rate of 47.6%. 46.1% of the households live in poverty.[12] The main aim of this study was to determine the prevalence of dyslipidaemia and to examine dietary and other contributing factors, namely hypertension, overweight and obesity and abnormal blood lipid levels in women residing in the Vaal region.

Method

This was a cross-sectional baseline survey study. The study protocol was approved by the University of the Witwatersrand Medical Ethics Committee for Research on Human Beings (M080931, M070126, M080930, M030556). Informed consent was obtained from all the participants after the objectives and study procedures had been explained to them. The research was conducted according to the Declaration of Helsinki's and the South African Medical Research Council's guidelines for research on human beings.

A power calculation[13] was used to determine the sample size for a 95% confidence level and 3.75 confidence interval (CI), based on the total number of adult women (≥ 20 years) (n = 3 666 111) in the Vaal region.[14] A sample size of 683 was required for statistical representation. Four peri-urban settlements were purposively included, based on the criteria of having a low income (< R1 500 per household per month) and being predominantly Sotho-speaking.[12] The districts in each of the settlements were randomly selected. A residential map was used and the researchers selected every fourth household for inclusion in the sample. An extra 39 respondents (5.7%) were selected to provide for possible dropouts during the data collection period (2008-2011).

Ten postgraduate students from the Vaal University of Technology were recruited and trained as fieldworkers to facilitate completion of the questionnaires. A manual was developed to train the fieldworkers to ensure a high standard of research. The same fieldworkers worked with the recruited respondents from the four settlements to regulate the procedures. A registered dietitian and a public health nutritionist (registered in the United States of America) took the anthropometric measurements. A registered haematologist and qualified nursing sisters from a pathology laboratory drew blood, collected the blood samples and recorded the blood pressure measurements. Blood pressure measurements were taken according to the generic measurement principles of the 2011 *South African hypertension guidelines*.[6]

Measurements

The age, employment status and monthly household income of the respondents were recorded. A 24-hour recall questionnaire was administered over a weekend and two weekdays by the trained fieldworkers, using food models to obtain quantitative descriptive information on dietary intakes. The mean intake over the three days was calculated for the various dietary fat intake variables. Dietary intake data were analysed by a registered dietitian using the Foodfinder® version 3 software programme developed by the Medical Research Council and based on the South African food composition tables.[15] Daily intakes of the dietary fat variables were compared to recommended cut-off points for a healthy diet. These cut-off points were calculated as a percentage of total energy intake as follows: total fat intake 20-35%, SFA ≤ 10%, total polyunsaturated fatty acids (PUFA) 6-11%, n-6 PUFA 2.5-9%, n-3 PUFA 0.5-2%, trans-fatty acids (TFA) < 1%, and monounsaturated fatty acids (MUFA) the balance.[16]

Anthropometric measurements included body weight and height measured according to standard procedures[17] with a calibrated Philips® electronic scale [model HF350 (135 kg/100 g) with a two-point decimal precision], and a Scales® 2000 stadiometer, respectively. All measurements were taken twice and the average of the two measurements recorded. BMI was calculated using weight (kg) divided by height squared (m^2) and categorised according to the World Health Organization cut-off points.[18]

The participants were asked to fast and were reminded of this by short message service the day before the biochemical measurements were taken. After a fasting period of approximately 8-10 hours, a venous blood sample was drawn before 10h00 with a Vacutainer® needle with minimal use of tourniquets. Breakfast was served immediately after blood collection. The blood was placed on ice until separation within two hours of blood collection. Serum and plasma were stored at -80^{0}C for two weeks until analysis to prevent changes in fatty acid composition as a result of prolonged storage times.[19] All blood parameters were examined according to standard protocol in the biochemical analysis laboratory at the tertiary institution. Total serum cholesterol, HDL-C and TG were analysed by means of the colorimetric method on a Konelab™® analyser with a coefficient of variation (per cent coefficient of variation (CV) between runs of 1.2-2.8% for all analysed serum variables). In this study, the CV was lower than that reported elsewhere.[19] The Friedewald formula was used to calculate LDL-C.[20]

Blood pressure measurements were taken by the registered nursing sisters using a Tensoval® digital blood pressure monitor. Two measurements were taken and the average of the two measurements was used. Blood pressure was defined according to the *South African hypertension guidelines*.[6]

Statistical analyses

The total group was subdivided into a dyslipidaemic and a nondyslipidaemic group. The dyslipidaemic group was classified as having one or more of the following conditions:

- TG ≥ 2.26 mmol/l
- TC ≥ 6.20 mmol/l
- HDL-C < 1.03 mmol/l
- LDL-C ≥ 2.59 mmol/l if the Framingham 10-year risk score was high, or LDL-C ≥ 3.36 mmol/l if the Framingham 10-year risk score was intermediate, or LDL-C ≥ 4.14 mmol/l if the Framingham 10-year risk score was low.[1,2]

All the data were analysed on Stata® version 12.0. Descriptive statistics were determined for the dyslipidaemic and nondyslipidaemic

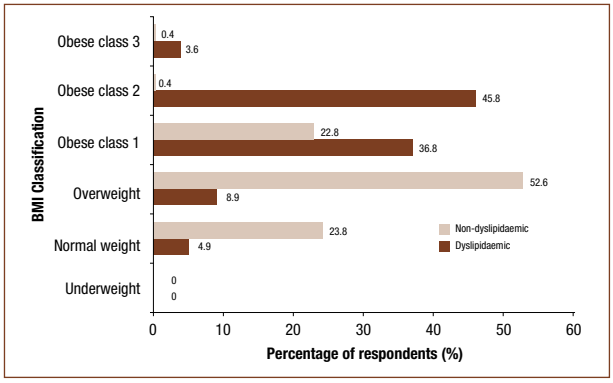
groups [means and standard deviations (SDs)], and the two-tailed independent t-test, including Levene's test for equality of means, were conducted to determine significant differences (p-value < 0.05) between the groups. Concomitantly, two-tailed bivariate Pearson correlations were carried out to determine the associations between serum lipid concentrations and dietary intake, and BMI and hypertension parameters at a 95% confidence level (p-value < 0.05). Regression analyses were performed in order to predict the risk factors that contributed to the prevalence of dyslipidaemia and to determine the variables with the greatest contribution.

Results

The results show that 34.3% (n = 247) of the women were dyslipidaemic. The mean ± SD age of the dyslipidaemic group of women was statistically significantly higher (p-value = 0.000) (55.4 ± 0.6 years) than that of the nondyslipidaemic group (41.2 ± 0.5 years). The same trend was observed for the monthly household income (see Table I).

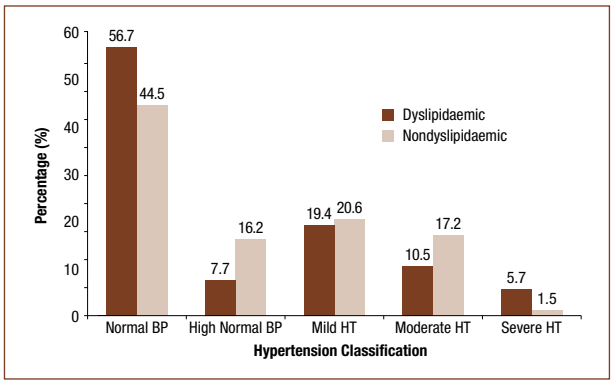
Significant differences in height (p-value = 0.000), weight (p-value = 0.000) and BMI (p-value = 0.001) were observed between the two groups. The dyslipidaemic group had the higher means for all these parameters. The results show that none of the women was underweight and that only 4.9% of the women with dyslipidaemia had normal weight compared with 23.9% of the women who were nondyslipidaemic (Figure 1). The majority of women in the nondyslipidaemic group were overweight (52.6%). However, the majority of women in the dyslipidaemic group (86.2%) were obese.

The hypertension results (Table I) showed that the systolic (p-value = 0.014) and diastolic (p-value = 0.029) blood pressure differed significantly between the two groups, with higher measurements in the dyslipidaemic group. This is further clarified in Figure 2 which shows that despite the fact that the mean ± SD for both systolic and diastolic blood pressure was within the normal range for both groups, 39.3% and 35.6% of the nondyslipidaemic and dyslipidaemic groups suffered from hypertension respectively.



Obesity class 1: body mass index 30-34.99 kg/m², obesity class 2: body mass index 35-39.99 kg/m², obesity class 3: body mass index ≥ 40 kg/m² [18]

Figure 1: Body mass index classification of the dyslipidaemic (n = 247) and nondyslipidaemic (n = 475) groups of women



BP: blood pressure, HT: hypertension

Figure 2: Prevalence of hypertension between the dyslipidaemic (n = 247) and nondyslipidaemic (n = 475) groups of women

The results in Table 1 indicate significant differences (p = 0.000) between the mean serum parameters of the two groups. Although the mean ± SD values for total serum cholesterol and LDL-C indicated no risk of dyslipidaemia in both the groups, the mean ± SD for TG indicated risk in the women with dyslipidaemia. Of the women, 72.4% had TG levels > 2.26 mmol/l. The low mean

Table I: Descriptive statistics of socio-economic, biochemical and blood pressure variables that were measured

Variable	Unit of measure (normal range)	Nondyslipidaemic (n = 475, 65.7%)		Dyslipidaemic (n = 247, 34.3%)		Significant difference between groups (p-value)*
		Mean	SD	Mean	SD	
Age	years	41.20	0.46	55.39	0.61	0.000
Monthly household income	R	698.44	338.13	796.89	538.88	0.003
Height	m	1.47	0.06	1.51	0.07	0.000
Weight	kg	59.21	7.17	77.61	9.76	0.000
Body mass index	kg/m²	27.56	3.38	34.11	3.94	0.001
Systolic blood pressure	mmHg (< 140)[6]	127.27	20.94	131.74	24.04	0.014
Diastolic blood pressure	mmHg (< 90)[6]	83.98	14.04	86.12	11.60	0.029
Serum cholesterol	mmol/l (< 5.20)[5]	4.51	0.46	4.30	0.63	0.000
HDL-C	mmol/l (> 0.90)[5]	1.37	0.10	0.96	0.20	0.000
LDL-C	mmol/l (≥ 4.14)[5]	1.22	0.46	2.37	0.66	0.000
Serum triglycerides	mmol/l (< 1.70)[5]	1.22	0.46	2.37	0.66	0.000

* p-value = significant if < 0.05
HDL: high-density lipoprotein, LDL: low-density lipoprotein, R: rands, SD: standard deviation

± SD HDL-C indicated further risk in the women with dyslipidaemia. HDL-C levels of < 1.03 mmol/l were present in 64.3% of the women. Two per cent of the women with dyslipidaemia had TC levels < 6.20 mmol/l and only 0.4% had LDL-C levels of ≥ 3.36 mmol/l. This equates to a dyslipidaemia disease state. Only 0.4% and 1.2% of the women presented with two and three of the criteria, respectively. None of the women presented with all four of the criteria. The HDL to LDL ratio is, however, a better indicator of CVD risk than the individual levels of HDL-C and LDL-C. The observed mean ± SD HDL to LDL ratio of 2.37 ± 0.66 was significantly higher (p-value = 0.000) in the dyslipidaemic group than the 1.22 ± 0.46 in the nondyslipidaemic group, but the HDL to LDL ratio was much higher in both groups than the recommended > 0.4 ratio. Therefore, this indicates a low risk of CVD in this sample of women.[21] The total serum cholesterol to HDL-C ratio of 3.29 ± 0.34 for the nondyslipidaemic group was lower than the recommended 5:1 ratio[22] which indicates a low risk for CVD. However, the total serum cholesterol to HDL-C ratio of the dyslipidaemic group was 5.11 ± 4.5, thus confirming a risk for CVD.[22]

Despite a statistically significantly (p-value = 0.017) higher intake in the dyslipidaemic group, the mean ± SD energy intake of both groups was low when compared with the estimated energy requirements (see Table II). This was also true for total protein and dietary fibre intakes when compared with the estimated average requirements (EAR), although these intakes did not differ significantly between the groups. In both groups, the mean carbohydrate intakes were higher than the EAR. The dyslipidaemic group had a significantly higher intake (p-value = 0.007) compared to the nondyslipidaemic group. The total fat intake showed low intakes of 20% of total energy intake in both groups. According to the guidelines[7] for the prevention of chronic disease, SFA, PUFA, MUFA, TFA and linoleic acid intakes recorded percentages in line with the recommendations for both groups, whereas linolenic acid intake percentages (0.2%) were much lower than the recommended goal of 0.5-2% in both groups. The PUFA intake (5.4%) for the women with dyslipidaemia was lower than the recommended 6-11% of total energy intake, whereas the nondyslipidaemic women had borderline intake (6.11%). No significant differences were observed between the dietary fat intake variables between the groups.

The Pearson correlation analyses revealed that all the serum lipid levels significantly (p-value < 0.05) related to hypertension and age. A significant (p-value = 0.001) positive association existed between the prevalence of dyslipidaemia and age (r = 0.560, p-value = 0.000), BMI (r = 0.656, p-value = 0.000), employment (r = 0.260, p-value = 0.000) and income (r = 0.111, p-value = 0.003) at 99% confidence level, as well as total energy intake (r = 0.084, p-value = 0.024) at 95% confidence level. A significant negative association was observed between the prevalence of dyslipidaemia and education level (r = -0.291, p-value = 0.000) at 99% confidence level.

The linear regression model predicted that BMI (ß = 0.554, p-value = 0.000), age (ß = 0.419, p-value = 0.000), education (ß = 0.250, p-value = 0.000), total energy intake (ß = 0.105, p-value = 0.006) and total fat intake (ß = 0.092, p-value = 0.018) were predictors of

Table II: Analysis of 24-hour recall: daily mean intakes of the women (n = 722)

Dietary intake variable	Unit of measure	Nondyslipidaemic			Dyslipidaemic			Significant difference between groups (p-value)*	EAR[23]/FAO and WHO guidelines[16]
		Mean	SD	% of energy intakes	Mean	SD	% of energy intakes		
Total energy intake	kJ	4466.20	2074		5078.3	2164.40		0.017	8605 EER**
Total protein	g	39.04	24.43		41.95	27.21		0.145	46
Plant protein	g	19.01	10.18		19.66	8.59		0.394	
Animal protein	g	19.85	22.54		21.94	24.73		0.253	
Fat	g	28.77	26.71	20.0	30.26	29.30	20.0	0.493	20-35% total EI
Cholesterol	g	164.04	136.69		151.98	130.22		0.253	
SFA	g	8.65	9.24	7.3	9.45	11.30	7.0	0.313	< 10% total EI
MUFA	g	9.83	10.38		10.33	12.20		0.565	Balance [total fat - (SFA + TFA + PUFA)]
PUFA	g	7.26	9.14	6.1	7.32	7.04	5.4	0.919	6-11% total EI
TFA	g	0.43	1.31	0.4	0.37	0.94	0.2	0.567	< 1% total EI
Linoleic acid (n-6) C18:2	g	6.81	8.97	5.7	6.68	6.69	5.0	0.841	2.5-9% total EI
Linolenic acid (n-3) C18:3	g	0.22	0.21	0.19	0.22	0.23	0.16	0.877	0.5-2% total EI
Carbohydrate	g	163.77	77.31		179.80	70.58		0.007	100
Dietary fibre	g	10.40	6.39		10.84	6.08		0.365	25

* p-value is significant if < 0.050

** Estimated energy requirements were calculated based on mean ± standard deviation age for the total group (46 ± 12 years), mean ± standard deviation height, weight and body mass index of 1.5 ± 0.6 m, 66 ± 12 kg and 30 ± 5 kg/m^2 respectively, and moderate activity levels. The estimated energy requirements of 8 605 kJ were calculated as 448 – [7.95 x age (46)] + physical activity level for low active (1.5) x [11.4 x weight (66) x height (1.5)] x 4.18 kJ.[23]

EAR: estimated average requirement, EER: estimated energy requirement, EI: energy intake, FAO: Food and Agriculture Organization of the United Nations, MUFA: monounsaturated fatty acids, PUFA: polyunsaturated fatty acids, SD: standard deviation, SFA: saturated fatty acids, TFA: trans-fatty acids, WHO: World Health Organization

dyslipidaemia in these women. No other variables were significant. The logistic regression analysis model showed that it was more likely that dyslipidaemia would be prevalent with a higher BMI [odds ratio (OR) = 1.61, 95% CI: 1.47-1.75, p-value = 0.000], the prevalence of hypertension as observed by diastolic blood pressure [OR = 1.05, 95% CI: 1.02-1.09, p-value = 0.001) and age (OR = 1.23, 95% CI: 1.19-1.29, p-value = 0.000). The ORs for education, total energy and fat intakes were less than one and not significant.

Discussion

Despite the lack of national data on the prevalence of dyslipidaemia, CVD risk factors are prominent in the South African population.[24] Seventeen per cent of all deaths in South Africa in 2000 were due to CVD. CVD also contributed to more deaths in women than in men.[25] Despite abnormally low LDL-C in the nondyslipidaemic group and high TG and low levels of HDL-C in the dyslipidaemic group, respectively, the means of most of the lipid parameters were within the normal range for both the groups, even though dyslipidaemia was prevalent in 34.2% of these women. The main factors contributing to dyslipidaemia in this study were high TG and HDL-C levels. Interestingly, in the group of nondyslipidaemia women, the mean ± SD HDL-C value of 1.37 ± 0.10 mmol/l was above the cut-off point of 1.03 mmol/l. Although this indicates normal levels, this level was not close to the 1.55 mmol/l that is considered to be protective against CVD, thus becoming a negative risk factor.[1,26] So, even in the nondyslipidaemia group, the observed HDL-C serum levels did not reach the gold standard of 1.55 mmol/l whereby HDL-C is no longer a risk factor for CVD. However, the main prognostic lipid value is considered to be the total serum cholesterol to HDL-C ratio which is known to correlate with the development of acute coronary events.[1,27] In this study, the total serum cholesterol to HDL-C ratio of the dyslipidaemic group confirmed the risk for CVD.[22]

Maritz[24] indicated that a number of smaller randomly selected studies that were undertaken from 1982-1996 in South Africa found that 25% of black women were hypertensive and hypercholesterolaemic, especially older women. This could be the result of a higher prevalence of obesity in older women.[24] In this study, only 2% of the women presented with hypercholesterolaemia which was lower than the national prevalence of 32% in black women.[28] Research also found that the mean total serum cholesterol concentrations increased with age, peaking in those from 55-64 years old.[11] Total serum cholesterol levels were positively significantly associated with hypertension and age in this group of women with dyslipidaemia. This is consistent with findings in Eastern Poland.[29]

Hypertension is one of the major and most costly contributors to CVD in the South African population. It amounted to an expenditure of R4-5 billion in 1991 and constituted 7.5% of the direct total healthcare expenditure in the country.[6] Hypertension is the most commonly found CVD risk factor in the USA and Cuba and strongly correlates with CVD in black people.[30] In South Africa, the estimated hypertension prevalence rate for women was 41.4% in 2008.[31] In this study, the mean systolic and diastolic blood pressure was significantly higher in the dyslipidaemic group, although hypertension was more prevalent in the nondyslipidaemic women, than in those with dyslipidaemia. However, this was not significant. The higher prevalence of obesity and the more advanced age of the dyslipidaemic group may have contributed to higher blood pressure as age and obesity are well-known risk factors of hypertension.[32]

Although none of the women in this study presented with underweight, significantly more nondyslipidaemic women presented with normal weight compared to the women with dyslipidaemia. The majority of the nondyslipidaemic women were overweight, whereas the majority of the women with dyslipidaemia were obese. This high rate of obesity is consistent with the 41% obesity prevalence rate for women in South Africa.[31] The BMI positively and significantly correlated with age in this study. The older the participants were, the higher their BMI. Ageing is a proven risk factor for CVD in both men and women,[30] and in Latin America, it was found that with every additional five years of age in the elderly, the CVD prevalence increased by 11%.[33]

The main sources of dietary fat were flesh foods (meat, poultry and fish) with a mean ± SD food variety score (FVS) of 3.3 ± 2.6 for a seven-day period, followed by fats and oils (mainly sunflower oil) with a mean ± SD FVS of 1.5 ± 1.2, and dairy products (milk, cheese and sour milk) with a mean ± SD FVS of 1.3 ± 1.5.[34] Although the total dietary fat intake for both groups was low, the dietary fatty acid intakes of the women in this study met the recommended guidelines,[16] except for low PUFA intakes in the women with dyslipidaemia. The essential linoleic and alpha-linolenic fatty acids have a positive effect on blood lipid concentrations and play an important role in the working of the nervous system.[35] The main sources of linoleic acid in the diet include vegetable oils such as sunflower oil, soybean and maize oil, and nuts and seeds. Generally, these are more available than the food sources of linolenic acid, including walnuts, linseed and rapeseed oil. This was also true in this study in which the diet of both the groups almost met the guidelines for linoleic acid, but contained a very low intake of linolenic acid, thereby not meeting the recommended guideline. In this study, sunflower oil was the main contributor to linoleic acid.

BMI, age and education, in this order, were strong predictors of dyslipidaemia. Total energy fat intakes showed a weaker relationship. The results also confirmed that dyslipidaemia correlated with a higher BMI, hypertension and age. Thus, the findings of this study are consistent with those of other studies in which BMI,[36,30] hypertension,[30] age[30] and education[36] were linked with dyslipidaemia, and thus CVD risk. Overweight and obesity, a higher BMI (≥ 25 kg/m^2), and hypertension and age, are well-known risk factors for CVD. A higher socio-economic status, as measured by years of education and income, has been found to be positively and statistically significantly linked with obesity in women in South Africa.[37] In this study, a positive relationship was shown to exist between dyslipidaemia and employment. Therefore, education could lead to affluence and the consumption of energy-rich foods, resulting in obesity, a major contributing factor to dyslipidaemia and CVD.

Conclusion and recommendations

High TG and low HDL-C levels were the most frequent abnormalities in the women with dyslipidaemia in this study. Although positive

associations existed between the prevalence of dyslipidaemia and the known risk factors of CVD; BMI, age and education were the main predictors of dyslipidaemia. Furthermore, dyslipidaemia was more likely to be prevalent with a higher BMI, hypertension and more advanced age in this group of women.

A well-designed, longitudinal study of a statistically representative sample should be implemented in the Vaal region to determine the presence of the behavioural (diet, smoking and physical activity) and metabolic (hypertension, obesity, blood lipid profile, diabetes, metabolic syndrome and C-reactive protein) factors that contribute to CVD. Furthermore, CVD risk factors should be continually monitored and used as tools to develop appropriate actions and to design more successful strategies for prevention, early detection and treatment.

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