Oldewage-Theron WH, Egal AA. Prevalence of and contributing factors to dyslipidaemia in low-income women aged 18-90 years in the peri-urban Vaal region. S Afr J Clin Nutr. 2013;26(1):23-28

http://www.sajcn.co.za/index.php/SAJCN/article/view/634/956

To the Editor:

I read with great interest the excellent article by Oldewage-Theron and Egal.[1]

Their key finding was that despite eating a diet of 71% carbohydrate to 20% fat to 9% protein, that approaches the macronutrient proportions advocated as healthy by South African dietary experts,[2,3] 34% of women in a peri-urban Vaal region had dyslipidaemia. Eighty-two per cent of the women with dyslipidaemia were obese, whereas 53% of the women without dyslipaemia were also overweight. More than 35% from both groups were hypertensive. The rates for diabetes were not reported.

The key difference in the measured blood parameters was a (paradoxically) lower serum cholesterol concentration in the dyslipidaemic group, in whom there was also a 30% lower serum high-density lipoprotein (HD) cholesterol concentration. There is clearly an error that was repeated in the bottom two lines of Table I. If it is presumed that the repeated line reports serum triglyceride [not low-density lipoprotein (LDL) cholesterol] concentrations, then this value was 1.94-fold higher in the dyslipidaemic group.

The authors did not report the prevalence of diabetes in this group, or glycated haemoglobin A_{1c} (HbA_{1c}) values, which would have given an idea of the extent to which insulin resistance was present in the community.

The point of this letter is that high blood triglyceride concentrations, together with low HDL cholesterol levels, and an increased number of small, dense LDL cholesterol particles, are a function of the carbohydrate, not the fat, content of the diet, as repeatedly shown.[4-8] Thus, the conclusion of this study should be that the dyslipidaemia present in so many subjects in this study, as well as their high rates of hypertension, obesity and likely diabetes, must be the result of a high-carbohydrate diet in those with varying degrees of insulin resistance.[9,10]

The authors do not make this connection, but consider other explanations for this finding, including "affluence and the consumption of energy-rich foods, resulting in obesity, a major contributing factor to dyslipidaemia and cardiovascular disease".

Instead, the findings of this important study should alert us to three alternate interpretations.

Firstly, the dyslipidaemia present in this population, and therefore perhaps also in a much wider group of South Africans, cannot be caused by a diet that is too high in fat, in line with the finding that this population did not have a high fat intake.

Secondly, the more likely explanation is that the high carbohydrate content of this group's diet was driving their high rates of obesity, dyslipidaemia and hypertension.

Thirdly, this would best be explained if there was a high prevalence of insulin resistance in this population, as seems probable, given the high rate of type 2 diabetes mellitus in similar South African populations.

If these point are true, reducing the carbohydrate, rather than the fat and protein, intakes, in this population would be more beneficial in improving their health[6,7,11] by reducing their high rates of obesity, dyslipidaemia, hypertension and probably type 2 diabetes mellitus.

Timothy Noakes,
University of Cape Town

References

1. Oldewage-Theron WH, Egal AA. Prevalence of and contributing factors to dyslipidaemia in low-income women aged 18-90years in the peri-urban Vaal region. S Afr J Clin Nutr. 2013;26(1):23-28.
2. Vorster HH. "Make starchy foods part of most meals": A food-based dietary guideline for South Africa. S Afr J Clin Nutr. 2013;26(3):S28-S35.
3. Vorster HH, Badham JB, Venter CS. An introduction to the revised food-based dietary guidelines for South Africa. S Afr J Clin Nutr. 2013;26(3):S5-S12.
4. Farquhar JW, Frank A, Gross RC, Reaven GM. Glucose, insulin, and triglyceride responses to high and low carbohydrate diets in man. J Clin Invest. 1966;45(10):1648-1656.
5. Petersen KF, Dufour S, Savage DB, et al. The role of skeletal muscle insulin resistance in the pathogenesis of the metabolic syndrome. Proc Natl Acad Sci USA. 2007;104(31):12587-1294.
6. Volek JS, Phinney SD, Forsythe CE, et al. Carbohydrate restriction has a more favorable impact on the metabolic syndrome than a low fat diet. Lipids. 2009;44(4):297-309.
7. Volek JS, Sharman MJ, Gomez AL, et al. An isoenergetic very low carbohydrate diet improves serum HDL cholesterol and triacylglycerol concentrations, the total cholesterol to HDL cholesterol ratio and postprandial pipemic responses compared with a low fat diet in normal weight, normolipidemic women. J Nutr. 2003;133(9):2756-2761.
8. Schwarz JM, Linfoot P, Dare D, Aghajanian K. Hepatic de novo lipogenesis in normoinsulinemic and hyperinsulinemic subjects consuming high-fat, low-carbohydrate and low-fat, high-carbohydrate isoenergetic diets. Am J Clin Nutr. 2003;77(1):43-50.
9. Reaven GM. The metabolic syndrome: requiescat in pace. Clin Chem. 2005;51(6):931-938.
10. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes. 1988;37(12):1595-1607.
11. Hu T, Mills KT, Yao L, et al. Effects of low-carbohydrate diets versus low-fat diets on metabolic risk factors: a meta-analysis of randomized controlled clinical trials. Am J Epidemiol. 2012;176 Suppl 7:S44-S54.

Authors' reply:

We appreciate the interest in the article, and hereby respond to the author's queries.

The authors did not report the diabetes rate because the focus of the article was dyslipidaemia. We acknowledge that diabetes could coexist with dyslipidaemia. We would gladly conduct a follow-up study in which fasting glucose and HBA_{1c} were reported, as well as the waist to height ratio, as an indication of the metabolic syndrome.

With regard to the comment on Table I, the authors re-analysed the data in Table I on 25 February 2014. When analysing the blood, the mean values were reported as mg/dl, originally. However, the journal requested that the values be converted to mmol/l. When using the conversion factors as 91.5 mg/dl ÷ 38.6 for LDL cholesterol, and 209.8 mg/dl ÷ 88.5 for triglycerides,[1] the new values resulted in identical figures for the last two variables in the table. Thus, this was not a typographical error, and the authors were just as surprised.

In relation to dislipidaemia, the authors agree with the fact that the dyslipidaemia in this study may have been the result of the high-carbohydrate diet. However, no significant correlation was observed between the prevalence of dyslipidaemia or any of the serum lipid parameters and carbohydrate intake. All of the macronutrient intakes were higher in the dyslipidaemic group. However, significant differences were not observed between the dyslipidaemic and non-dyslipidaemic group, except that total energy and carbohydrate intake was significantly higher in the dyslipidaemic group (Table II). A significant correlation existed between the prevalence of dyslipidaemia and total energy intake only. Furthermore, the linear regression model predicted that both total energy intake (ß 0.105, p-value 0.006) and total fat intake (ß 0.092, p-value 0.018), and not carbohydrates, were predictors of dyslipidaemia in these women. This finding was consistent with a study undertaken on Iranian women where higher intakes of energy were associated with increased total cholesterol and LDL cholesterol, and lower HDL cholesterol levels. Furthermore, a higher dietary energy density was associated with dyslipidaemia.[2] Bales and Kraus[3] reported that evidence from animal studies and few human trials confirmed that energy restriction had the potential to have a beneficial effect on the lipid profile. Carbohydrates contributed the most to total dietary energy intake, and thus it could be assumed that carbohydrate intake may have played a role, but this was not statistically substantiated. Furthermore, carbohydrate intake is not regarded as an independent risk factor in the aetiology of cardiovascular disease.[4,5]

With regard to the three alternate interpretations suggested by the author of the correspondence, the authors agree that the dyslipidaemia present in this population could not have been caused by a diet that was too high in fat, in line with the finding that the fat intake in this elderly sample was not high. We do not wish to speculate on whether or not the more likely explanation is that the high carbohydrate content of the diet compelled their high rates of obesity, dyslipidaemia and hypertension, but the statistically significant higher total energy intake, irrespective of carbohydrate intake, cannot be ignored (low as the total energy intake may have been in relation to the EAR/Food and Agricultural Organization of the United Nations and World Health Organization guidelines).[3]

The authors would also like to highlight the section in the recommendations of the article, in which the need for a longitudinal study was proposed to further investigate possible factors, including dietary ones, that could have contributed to the high prevalence rate of dyslipidaemia in this sample of elderly people.

Wilna Oldewage-Theron, PhD, Director; **Abdulkadir Egal,** PhD, Senior Researcher

Centre of Sustainable Livelihoods, Vaal University of Technology, Vanderbijlpark

References

1. Cholesterol and triglyceride conversion. Heart UK [homepage on the Internet]. c2014. Available from: http://heartuk.org.uk/fioles/uploads/dovuemnts/huk_fs_mfsP-cholestrigly_levelsconversion.pdf
2. Esmailzadeh A, Boroujeni HK, Azadbakht L. Consumptipn of energy-dense diets in relation to cardiometabolic abnormalities among Iranian women. Public Health Nutr. 2011;15(5):868-875.
3. Bales C, Kraus WE. Caloric restriction: implications for human cardiometabolic health. J Cardiopulm Rehabil Prev. 2013;33(4):201-208.
4. Institute of Medicine. Dietary carbohydrates: sugars and starches. Dietary reference intakes for energy, carbohydrates, fiber, fat, fatty acids, cholesterol, protein and amino acids. Washington: National Academic Press; 2002.
5. International Food Information Council Foundation. The science of sugars. Washington: IFIC; 2008.