The Editor
South African Journal of Clinical Nutrition

Dear Prof Labadarios

RESPONSE TO QUERY SAJCN #634

Thank you very much for sending us the letter regarding our article. We will respond to the query as follows in red:

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

Their key finding was that despite eating a diet (71:20:9% - carbohydrate:fat: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 percent of women with dyslipidaemia were obese whereas 53% of the women without dyslipaemia were also overweight. More than 35% of both groups were also hypertensive. Rates of diabetes were not reported.

The authors did not report diabetes rate as the focus was on dyslipidaemia. The authors acknowledge that diabetes could co-exist with dyslipidaemia.

The key difference in the measured blood parameters was a (paradoxically) lower serum cholesterol concentration in the dyslipidaemic group who also had 30% lower serum HDL-cholesterol concentrations. There is clearly an error in the bottom two lines of Table 1 that are repeated. If one presumes that the repeated line reports serum triglyceride (not LDL-cholesterol) concentrations, then this value was 1.94-fold higher in the dyslipidaemic group.

The authors re-analysed the data in Table 1 on 25 February 2014. See SPSS, version 22.0, output underneath.

T-TEST GROUPS=Dyslipidemia(1 2)

/MISSING=ANALYSIS

/VARIABLES=TC HDL LDL TRG

/CRITERIA=CI(.95).

T-Test

Notes

Output Created		25-FEB-2014 16:26:00
Comments		
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	N of Rows in Working Data File	722
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.
Syntax		T-TEST GROUPS=Dyslipidemia(1 2) /MISSING=ANALYSIS /VARIABLES=TC HDL LDL TRG /CRITERIA=CI(.95).
Resources	Processor Time	00:00:00.03
	Elapsed Time	00:00:00.05

Group Statistics

	Dyslipidemia	N	Mean	Std. Deviation	Std. Error Mean
TC	1	247	166.47175432240186	24.23664085909749 0	1.542140528435990
	2	475	174.30069413140086	17.81082305550018 0	.817216608423259
HDL	1	247	36.937887654338 47	7.656812125145233	.487191288819810
	2	475	53.072923601970 99	3.814872215234793	.175038341776101
LDL	1	247	91.540050068570 32	25.656432645253620	1.632479716448921
	2	475	47.060474689717 37	17.926798354464290	.822537920087801
TRG	1	247	209.81303511291787	58.805451815227300	3.741701296993543
	2	475	107.86427384634347	41.088856405365036	1.885286029227790

The mean values were reported as mg/dL originally as can be seen in the statistical analyses. However, the journal requested that the values be converted to mmol/L. When using the conversion factors as mg/dL ÷ 38.6 for cholesterol and mg/dL ÷ 88.5 for triglycerides (Heart UK. Cholesterol and triglyceride levels conversion. Available at: http://heartuk.org.uk/files/uploads/documents/huk_fs_mfsP_cholestrigly_leverlsconversion.pdf Accessed: 25 Feb 2014), the new values resulted in identical figures for the last two variables in the table. Thus this is not a typographical error and the authors were just as surprised when the conversion was done.

The authors did not report the prevalence of diabetes in this group or the glycated haemoglobin (HbA1c) values which would have given an idea of the extent to which insulin resistance is present in the community.

As stated earlier, the focus of this article was on dyslipidaemia. The authors would gladly do a follow-up study and article in which fasting glucose and HBA1c will be reported as well as the waist:height ratio as an indication of metabolic syndrome.

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 high carbohydrate diets 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 CVD".

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 prevalence of dyslipidaemia or any of the serum lipid parameters and carbohydrate intake. A significant correlation existed between the prevalence of dyslipidaemia and total energy intake only. Carbohydrates contributed the most to total dietary energy intake and it could thus be assumed that carbohydrate intakes may have a role, but this was not statistically proven.

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

First that 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 does not have a high fat intake.

The authors agree with this statement.

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

The authors would not want to speculate on this (see above explanation).

Third that this would best be explained if there is a high prevalence of insulin resistance in this population, as seems probable given the high rates of Type II diabetes mellitus in similar South African populations.

This was not reported in this study and the authors would rather do a follow-up study to measure the actual prevalence rate of diabetes mellitus and insulin resistance in this group of women.

If these points are true, reducing the carbohydrate rather than the fat and protein intakes of this population would be more beneficial for improving their health ([6;7;11]) by reducing their high rates of obesity, dyslipidaemia, hypertension and probably Type II diabetes mellitus.

The authors would like to draw the writer's attention to the recommendation section of the article in which they recommended a longitudinal study to further investigate the possible factors, including dietary factors that could contribute to the high prevalence rate of dyslipidaemia in this group of women.

The authors want to thank the writer for the interest shown in the article and for the constructive comments. We will consider all the comments in the design of our next study of this nature.

Yours sincerely

Wilna Oldewage-Theron & Abdulkadir Egal
Authors