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Letters to the Editor

The Diabetic Diet

Dear Sir,

We enjoyed the review article by Dr. Jenkins and co-workers [1] and agree that it is time for diabetologists to start to consider food items not merely on the basis of their biochemical properties (nutrients) but, rather, for their physiological effects (glycaemic response) on metabolism. Bearing this in mind, we find that the recent authoritative dietary recommendations [2–3] have been overtaken, at least in part, by the new body of knowledge which is comprehensively reviewed in Dr. Jenkins' article.

In our opinion simple advice to diabetic patients to increase their carbohydrate consumption, while reducing their lipid intake, makes little sense. However, if interpreted, as in Dr. Jenkins' article, in the sense of an encouragement to substitute calories from butter and dairy products with beans and other legumes, it is appropriate. On the other hand, can an increase in carbohydrate intake, obtained by cutting down the consumption of olive oil and intensifying the use of bread, rice, and potatoes, still be considered a desirable goal for diabetic patients? We substituted 50 g of olive oil with an isocaloric amount of white bread in the diet (2000 Kcal, 40% carbohydrate) of six insulin-dependent diabetic patients. This led to a marked deterioration of blood glucose control (24 h monitoring by Biostator): the average daily blood glucose concentration rose from 8.44 ± 1.28 to 11.38 ± 3.50 mmol/l (mean $\pm$ SD) ($p < 0.05$) and so did the post-prandial blood glucose (7.99 ± 3.50 versus 12.43 ± 6.05 mmol/l, $p < 0.05$). Conversely, no substantial modification of serum lipoproteins was found (unpublished data).

Various dietary fats have different effects on serum lipoproteins. Monounsaturated and polyunsaturated lipids are not associated with hyperlipoproteinaemia even if consumed in large quantities [4]. Therefore to prevent atherosclerosis in diabetic patients by dietary means, it is advisable to reduce mainly saturated fats and cholesterol consumption to keep low the serum level of cholesterol and low densitiy lipoproteins which act as powerful risk factors in diabetic patients as in the general population [5].

Moreover, a high consumption of dietary fibre, but not carbohydrates, can improve the blood glucose control in diabetic patients [6] and reduce the serum level of the most atherogenic lipoproteins both in diabetic [6] and non-diabetic [7] individuals.

Therefore, if we want to proceed in the way indicated by Dr. Jenkins' review, we should characterize the diet for diabetic patients not simply by its nutrient composition but rather by the selection of food items known to normalize various metabolic parameters which are deranged in these patients.

The type of diet used in south Italy and in many rural areas of southern Europe not more than two decades ago probably has these characteristics. It is rich legumes, vegetables, fruit, fish, olive oil and spaghetti (which has a favourable glycaemic index!). We and others call this the 'Mediterranean diet' to refer to its geographical and cultural background. Using this diet, we have shown favourable metabolic effects both in diabetic and non-diabetic individuals [6, 7].

We believe that the 'Mediterranean diet' will prove to be well accepted in the long-term by diabetic people and, in general, by people who are concerned with health. This might further contribute in the correction of the metabolic abnormalities (by dietary means) which predispose to atherosclerosis.

Yours sincerely
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Very Low Density Lipoprotein Metabolism in Non-ketotic Diabetes Mellitus

Dear Sir,

Based on their observation that the mean pre-therapy values of very low density lipoprotein (VLDL) triglyceride turnover rates declined following dietary restriction and that this decline was accompanied by a fall in mean VLDL triglyceride levels without significant change in mean fractional catabolic rates, Ginsberg and Grundy [1] concluded that "the primary cause of hypertriglyceridaemia is excessive production of VLDL triglyceride." This conclusion raises important questions about the interpretation of tracer kinetic data, and the article serves to direct attention to the applicability of tracer kinetic studies, as currently employed, in the investigation of pathogenetic mechanisms.

Scrutiny of the pre- and post-therapy patterns of triglyceride kinetics in the individual subjects reported by these authors questions the validity of their assertion. Of the nine patients studied, seven demonstrated some atypical features that are at variance with the authors' interpretation of their findings, as exemplified by patients 4 and 9, who allegedly "had marked hypertriglyceridaemia without over-production. They thus appeared to have a defect in clearance of VLDL triglyceride."

A more fundamental issue is whether tracer kinetic studies conducted under different steady states [2–6] can identify defects in pathogenetic mechanisms. While there is no disagreement that at a particular steady state and turnover rate, the production and removal rates are equal, nonetheless, at two different steady states, differences in turnover rate are construed as signifying changes in net production (or removal), and accounting for alterations in the triglyceride concentration. A change in turnover rate from one steady state to the next, does not indicate an increase or decrease in net production, as any change in the "synthetic rate" is accompanied by an equal and compensatory alteration in the "removal rate". As a corollary, an alteration in the fractional catabolic rate does not provide an independent estimate of net triglyceride removal or clearance. In tracer studies of the three variables (triglyceride concentration, triglyceride turnover rate and fractional catabolic rate), only two are directly measured (the concentration, and either the fractional catabolic rate or the turnover rate), the third being derived from the other two [7]. Reference to the turnover rate and the fractional catabolic rate as though