

Letters

Response to 'Hypertriglyceridaemia: A practical approach for primary care'

My colleagues and I read with interest the recent article by Lan et al, 'Hypertriglyceridaemia: A practical approach for primary care'.¹ While comprehensive, we were concerned that carbohydrate restriction – the intervention with the strongest mechanistic rationale and clinical evidence for triglyceride reduction – received insufficient emphasis in the dietary recommendations.

Table 2's dietary guidance appropriately mentions restricting refined sugars and sugar-sweetened beverages, yet frames carbohydrate intake generically as '4–6 servings/day unless a lower-carbohydrate diet is indicated'. This understates the primary role of dietary carbohydrate in driving hepatic triglyceride synthesis. Parks demonstrated that carbohydrate intake, particularly fructose, directly stimulates hepatic de novo lipogenesis and very low-density lipoprotein production – the metabolic foundation of hypertriglyceridaemia.² Importantly, Forsythe et al showed triglyceride reduction occurs with carbohydrate restriction independent of weight loss, confirming this is a direct metabolic effect rather than merely a consequence of caloric reduction.³

The clinical trial evidence strongly supports prioritising carbohydrate restriction. Multiple systematic reviews demonstrate consistently superior triglyceride reduction with low-carbohydrate versus low-fat diets. The meta-analysis by Mansoor et al found an additional 0.26 mmol/L reduction favouring low-carbohydrate approaches,⁴ while Volek et al demonstrated 51% triglyceride reduction with very low-carbohydrate diets, in comparison to 19% with conventional

low-fat diets.⁵ These represent clinically meaningful differences for our patients.

For patients with insulin resistance and metabolic syndrome – the very populations most likely to present with hypertriglyceridaemia – carbohydrate restriction addresses the underlying metabolic dysfunction.⁶ Rather than treating triglycerides as an isolated lipid abnormality, therapeutic carbohydrate reduction improves the entire metabolic cluster: triglycerides, high-density lipoprotein cholesterol, glycaemic control, blood pressure and hepatic steatosis.

My colleagues and I suggest that clinical reviews and educational materials for general practitioners (GPs) should position carbohydrate restriction more prominently in hypertriglyceridaemia management. For mild-to-moderate elevation, eliminating refined carbohydrates and sugary beverages should be explicit first-line advice. For patients whose triglycerides remain elevated or who have concurrent metabolic syndrome, more comprehensive carbohydrate restriction (targeting <130 g or even <50 g daily for therapeutic ketosis) warrants consideration as primary dietary therapy, not merely as an optional approach.

Australian general practitioners need clear, evidence-based dietary guidance. The mechanistic rationale, trial evidence and clinical experience all support carbohydrate restriction as foundational therapy for hypertriglyceridaemia.

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Competing interests: PF is Chair of the Scientific Committee for the Australasian Society of Metabolic Health and operates a general practice with a special interest in metabolic health and low-carbohydrate dietary interventions. PF declares no financial conflicts of interest relevant to this correspondence.

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Reply

Thank you for your letter regarding our clinical article, 'Hypertriglyceridaemia: A practical approach for primary care'.¹ We agree that reducing carbohydrate intake is foundational in the management of hypertriglyceridaemia and that this

aspect could have been emphasised further. Within the limits of the word count, the article aimed to provide an overview of the overall management of hypertriglyceridaemia. This includes the identification of secondary causes such as diets high in carbohydrates and the importance of dietary modifications focusing on healthy eating patterns, which encompasses a reduction in simple carbohydrates. The importance of regulation of dietary carbohydrates was collectively emphasised in Tables 1 and 2, in Figure 1 and in the section on non-pharmacological treatment. As noted, prior studies have shown that carbohydrate-restricted diets lower triglyceride levels, with greatest reductions seen in very low-carbohydrate diets.²

Table 2 was reproduced from the American College of Cardiology guidelines for the management of persistent hypertriglyceridaemia³ and was also featured in a recent National Lipid Association (US) clinical perspective article.⁴ The quoted recommendation for ‘4–6 servings/day’ refers to grains (principally fibre-rich whole grains) rather than carbohydrates as a whole; we apologise if this was unclear. Nonetheless, we recognise that grains are a source of carbohydrates and that this should be considered in any dietary recommendations. Understanding and adhering to dietary modifications can be challenging for many patients, particularly diets that are low in both carbohydrates and fats.⁵ This highlights the need for personalised nutritional recommendations by a registered dietitian, which may enhance adherence and cardiometabolic care.

Overall, we agree that evidence-based dietary guidance for Australian general practitioners is essential and that carbohydrate restriction should be emphasised as foundational therapy for hypertriglyceridaemia. Comprehensive practical guidance on food consumption for the prevention of cardiovascular disease is provided in an earlier expert position statement.⁶

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