Letter to the editor

Postprandial dysmetabolism: assessment and treatment

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We read with interest the Pappas et al review on postprandial dysmetabolism.¹ The authors describe evidence linking elevated levels of postprandial glucose and triglycerides (TGs) with cardiovascular (CV) morbidity and mortality as well as with polycystic ovary syndrome (PCOS), non-alcoholic fatty liver disease (NAFLD) and lack of hepatic receptors such as low density lipoprotein receptor (LDLR).¹ Specifically, it was reported that subjects with familial hypercholesterolemia have exaggerated and prolonged response to fatty meal loading.² The authors also discuss methods for screening and diagnosis of postprandial dysmetabolism as well as therapeutic options including lifestyle modifications, antidiabetic and hypolipidemic drugs.

The lack of a widely approved standardized protocol for the assessment of postprandial lipemia (PPL) impedes the early diagnosis and adequate treatment of this metabolic disorder.³ In this context, a previous meta-analysis proposed a 4 h time-point following an oral fat test meal containing 70-79 g of fat as the most representative method to measure PPL.⁴ Pappas et al.¹ mention the cut-off point of 175 mg/dl for non-fasting TGs as suggested by White et al⁵

in a sub-population of the Women's Health Study. An expert panel statement had earlier discussed the clinical implications and assessment of PPL proposing as optimal non-fasting TGs <180 mg/dl (2 mmol/l) and desirable postprandial TGs ≤220 mg/dl (2.5 mmol/l) at any time following a fat tolerance test (given after 8 h of fasting and consisting of 75 g fat, 25 g carbohydrates and 10 g protein).⁶ The same expert panel recommended that a fat tolerance test should be performed in individuals with non-fasting TGs 89-180 mg/dl (1-2 mmol/l).

Antidiabetic and hypolipidemic drugs have been reported to improve postprandial dysmetabolism as mentioned by Pappas et al.¹ Statins and insulin-sensitizer drugs (i.e. metformin and pioglitazone) were also shown to beneficially affect metabolic and menstrual abnormalities in PCOS.⁷ Moreover, it was found that gut peptides such as glucagon-like peptide-1 (GLP-1) and GLP-2 could be involved in homeostasis of PPL. Thus, the GLP-1 receptor agonists (GLP-1 RA) may be able to improve metabolic features in PCOS women but further research is needed in this field.⁸ With regard to NAFLD, statins and antidiabetic drugs [e.g. pioglitazone, dipeptidyl peptidase-4 (DPP-4) inhibitors, GLP-1 RA and sodium-glucose co-transporter 2 (SGLT2) inhibitors] are also likely to improve biochemical and/or histological features of NAFLD and its advanced form i.e. non-alcoholic steatohepatitis (NASH).⁹⁻¹³ Overall, combination therapy with both antidiabetic and hypolipidemic drugs may be a useful therapeutic tool for patients with postprandial dysmetabolism and PCOS or NAFLD.¹⁴⁻¹⁶

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Nevertheless, further investigations are required to firmly establish such associations.

DECLARATION OF INTEREST

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