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Reply

EDITOR, — We appreciate the interest of Professor Read's team in our recent article in *Gut*.¹ Nevertheless, we would like to comment why we think our conclusions are valid, and also do not contradict the ones mentioned by Read *et al.*^{2,3}

Divergent rates of gastric emptying of different food components and their subsequent hyperaemic responses are well established.⁴ Indeed, in the cited paper by Moneta *et al.*,⁵ the hyperaemic effect of oral carbohydrates was the fastest one, whereas fat induced the most pronounced response in agreement with our hypothesis. These findings are not a contradiction to the unpublished studies by Qamar, where he tested the hyperaemic response to both liquid and solid oral meals. Indeed, in our own publication,¹ the response to oral liquid and solid food was tested and shown in Figure 1, giving similar amounts of responses with the exception that the maximal response was observed 15 minutes later for the solid meal, probably owing to slower gastric emptying. The aim of our studies, however, was to gain further insight into how different food components elicit different degrees of intestinal hyperaemia. To do so, we examined the major food components in an isocaloric and iso-osmotic manner by perfusing them into the duodenum to overcome the confounding effects of divergent rates of gastric emptying.

We cannot accept the hypothesis that differences between our results and those of Qamar *et al.*² were due to the addition of the water soluble non-absorbable marker polyethylene glycol (PEG). Non-absorbable polysaccharides such as pectin may delay gastric emptying and glucose absorption.⁵ Nevertheless, the concentrations given were higher than our PEG4000 concentration of 2 g/l. Interestingly, the authors of the cited study² used PEG4000 at a concentration of 5 g/l as an inert marker to examine the role of pectin.

Finally, animal studies have shown that PEG

at high osmolarities (3000 mosm/l) by itself increases splanchnic blood flow.⁴ Concentrations below 1500 mmol/l had no effect. In fact, postprandial osmolarities are much lower and in the range we tested with the 560 mM saline solution, where we found no change in superior mesenteric artery blood flow. As we added PEG at the same concentration in all our intraduodenal perfusion experiments, differences in hyperaemic responses to different food components cannot be caused by PEG.

We are unable to comment upon unpublished data and therefore hope that these studies will be published soon to help fruitful discussions.

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