Invited Commentary

Weight gain and insulin sensitivity: a role for the glycaemic index and dietary fibre?

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Since its first description by Jenkins et al.\(^{(1)}\) in the 1980s, the glycaemic index (GI) has been used as a dietary tool to enhance the glycaemic control of people living with diabetes. The management of glycaemia is relevant today more than ever, with an estimated 2.9 million people currently affected by type 2 diabetes in the UK\(^{(2)}\). However, the GI as a concept has dipped in and out of scientific fashion. By its nature, the GI is highly complex in that it hinges on a number of physico-chemical properties of carbohydrates, such as the chemical structure of the carbohydrate, the surrounding food matrix and the processing it undergoes\(^{(3)}\). The fact that GI measurements are confounded by other food components within the diet serves as an additional layer of complexity\(^{(4)}\). Consequently, the concept of the GI has been met, at times, with scepticism and some have found it challenging to embrace. However, one could argue that the proof is in the eating. There are now two high-quality systematic reviews that demonstrate the clinical utility of the GI in the management of type 2 diabetes\(^{(5,6)}\), and a 24-week randomised controlled trial demonstrating a 0.5% fall in glycosylated H\(_{b}(7)\). There is also a Cochrane systematic review which suggests that the GI may play a role in promoting weight loss\(^{(8)}\). Therefore, the literature, as it currently stands, presents a convincing case for the clinical application of the GI in the management of body weight and glucose homeostasis.

In this issue of the British Journal of Nutrition, Lagerpusch et al.\(^{(9)}\) observed that in the dynamic phase of weight gain, a high-fibre, low-GI diet reduced daytime measurements of intermittent glucose when compared with an energy-matched low-fibre, high-GI diet. Furthermore, the deterioration in insulin sensitivity induced by refeeding was attenuated, though the effects of the two dietary interventions were resolved at the end of the refeeding phase. The authors claim that there is public health relevance to this, as many adults demonstrate short-term weight cycling, i.e. repeated cycles of weight loss\(^{(9)}\). Therefore, as it currently stands, presents a surprising finding of the study was the absence of an improvement in insulin sensitivity following a low-v. high-GI diet. However, in this study, the total amount of fibre was matched in both the high- and low-GI groups. This raises the intriguing question of whether the dietary deconstruction seen in most nutritional interventions, in line with the current reductionist scientific approach, is actually detrimental. By controlling for every aspect of the diet, could we actually be missing important physiological effects that occur from dietary manipulations which often go hand-in-hand, like GI and dietary fibre?

The mechanisms underlying the beneficial effects of high-fibre, low-GI diets on insulin sensitivity are complex and multifactorial. In the gastrointestinal tract, low-GI diets with a high fibre content slow gastric emptying, reduce digesta transit rate and alter the luminal environment, all of which will ultimately delay glucose absorption and result in an ameliorated insulin response\(^{(10)}\). High-fibre, low-GI foods may also influence insulin sensitivity via mechanisms independent of actions within the upper gastrointestinal tract. For example, high-fibre, low-GI diets may promote insulin sensitivity by improving metabolic flexibility, i.e. the ability of an organism to modify fuel oxidation in response to changes in nutrient availability. Metabolic flexibility enables an efficient transition from lipid oxidation and high rates of fatty acid uptake during the fasted state, to suppression of lipid oxidation and increased glucose uptake and utilisation in response to insulin stimulation\(^{(10)}\). In the 1990s, it was demonstrated that exposure to a low-GI diet over a 4-week period increases...
whole-body insulin sensitivity and suppresses circulating NEFA levels\textsuperscript{(21,22)}, an effect probably due to increased insulin-stimulated NEFA uptake at the level of the adipocyte\textsuperscript{(22)}. In support of this, an elegant human study by Robertson \textit{et al.}\textsuperscript{(23)} subsequently demonstrated that consumption of a fermentable fibre improved insulin sensitivity and reduced adipose tissue lipolysis. This coincided with a rise in the plasma levels of SCFA, end products of bacterial fibre fermentation in the distal gut. Research into the metabolic effects of SCFA has been gaining momentum since the identification of the G-protein-coupled SCFA receptors GPR41 and GPR43. The expression profile of these receptors in the adipose tissue, colon, liver and pancreas provides a plausible network by which these products of fermentation may have an impact upon insulin sensitivity and metabolic flexibility\textsuperscript{(24)}. Evidence from GPR43 knockout mice suggests that SCFA may directly suppress lipolysis from adipocytes and lower circulating NEFA levels \textit{in vivo}, effects associated with improved insulin sensitivity\textsuperscript{(25)}. Furthermore, SCFA are thought to promote the release of gut hormones such as glucagon-like peptide 1, an incretin which enhances first-phase insulin release, through the activation of GPR43.

The paper by Lagerpusch \textit{et al.} attempts to further our understanding by bringing to light some interesting observations about complex high-fibre, low-GI diets and their ability to beneficially influence glucose homeostasis in the dynamic stages of weight regain, a relatively unexplored metabolic state. Important strengths of this study include the strictly controlled nutrition regimen and the comprehensive assessment of insulin sensitivity. However, this paper raises as many questions as it answers. With beneficial effects on glucose homeostasis being apparent only during the active weight-gain period and not seen at the end of the refeeding protocol, are the benefits of a high-fibre, low-GI diet largely acute? Similarly, does the lack of deterioration in any insulin sensitivity measurement between baseline and post-refeeding suggest that weight cycling is not detrimental to health, at least in terms of glucose metabolism? Are these findings in lean, healthy, young men directly applicable to higher-risk populations with existing glycaemic impairment? Lastly, can the methodological approach be taken a step further to unpick the mechanisms underlying the insulin-sensitising effects? The real strength of this paper is to act as a stimulus for further investigation into the complex physiological effects of a high-fibre, low-GI diet, the mechanisms underlying these effects and how they may be exploited to improve the management of glycaemia.

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References


