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Symposium on 'Dietary management of disease'

Session 3 (Joint with the British Dietetic Association): Management of obesity
Glycaemic index, appetite and body weight

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Much interest has been focused on the relationship between glycaemic index and body-weight loss, some of which is fuelled by popular media. However, there is a number of potential mechanisms that could be triggered by reducing the glycaemic index of the carbohydrate consumed in the diet. For example, the effect of foods on the gastrointestinal tract and the effect on blood glucose both could lead to potential appetite effects. Acute meal studies seem to point to an effect of glycaemic index on appetite regulation. However, the results of longer-term studies of weight loss are not as clear. In the present review a possible reason for this variation in outcome from the weight-loss studies will be discussed. The present review focuses on the possibility that the fermentable fibre content of the low-glycaemic-index diet may be important in weight-loss efficacy. A novel receptor that binds SCFA, the products of carbohydrate fermentation, has recently been described on the enteroendocrine L-cell in the colon. This cell releases a number of anorectic hormones and could offer an explanation of the appetite suppressant effects of fermentable carbohydrates. It could also explain the variability in the results of glycaemic-index weight-loss studies.

Carbohydrate: Appetite: Obesity: Glycaemic index: Gut hormones

Overview

The aim of the present review is not to consider glycaemic index (GI) in general but to debate the relationship between this method of classifying carbohydrate and appetite regulation. The term GI was originally introduced to classify different sources of carbohydrate (CHO) by their effect on post-meal glycaemia[1]. The aim was to improve glycaemic control in individuals living with diabetes. The glycaemic response to foods and its role in glycaemic control in individuals living with diabetes has been reviewed a number of times[2], but relatively little consideration has been given to its potential impact on satiety and long-term regulation of body weight. In particular, there is no clear evidence on whether low-GI foods or diets may be used as part of a strategy to reduce food and energy intake and provide a tool to manage obesity. Several intervention studies have shown that low-GI-CHO diets have practically no impact on body weight compared with isoenergetic high-GI-CHO diets[3]. A review of several *ad libitum* studies has concluded that low-GI-CHO *v*. high-GI-CHO diets eaten *ad libitum* result in a lower body weight[3]. This conclusion is also borne out by a systematic review, which has demonstrated an impact on body weight with the introduction of a low-GI diet[4].

Glycaemic index of foods or diets

GI as originally defined is the indexing of the glycaemic response to 50 g available CHO from a test food to the same amount of available CHO from glucose[1]. In

Abbreviations: CHO, carbohydrate; FCHO, fermentable CHO; GI, glycaemic index; PYY, peptide YY.

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practice, it corresponds to the incremental area under the blood response curve measured over 2 h of a 50 g CHO portion of a test food expressed as percentage of the response to the same amount of CHO from a standard food consumed by the same subject[5]. There are now a number of systematic reviews on the effect of GI in the management of diabetes and CHD[2,5,6].

Glycaemic index and satiety

Two recent systematic reviews have been conducted in this area[3,4]. They have examined the world literature in the case of effects on energy homeostasis in both 'short-term' studies (duration of 1 d) and 'long-term' studies (several days or weeks duration). Of the twenty-five short-term studies selected by the quality criteria of the systematic review most of the studies were randomised and satiety was assessed either by a subjective method based on visual scales or by an objective method using a preload test meal design. This methodology consists of either measuring the spontaneous energy intakes when a meal is served *ad libitum* at a given time after the consumption of the test foods or by measuring the time of spontaneous request for food and the corresponding energy intakes when snacks are provided *ad libitum* on request. The foods or meals tested in the studies were pure CHO, CHO foods or mixed meals. The findings are summarised as follows:

1. human studies testing pure CHO: an early short-term satiating effect (within the first hour) has been observed with high-GI CHO. An inverse association has been reported between the blood glucose response and the subjective appetite and food intake in the 1 h after consumption of isovolumetric preloads of CHO[7]. At 1 h after their consumption high-GI CHO (glucose, polycose and sucrose) suppress food intake whereas low-GI CHO (amylose, amylopectin and a fructose–glucose mixture) do not. The early satiating effect of high-GI CHO has been confirmed by the results of several other studies showing that high-GI CHO such as glucose and sucrose suppress short-term food intake 60–90 min after consumption of the preload. A later short-term satiating effect has been reported with low-GI CHO such as fructose. In the studies in which both fructose and glucose have been tested fructose produces an increased satiety compared with glucose at 135 min after consumption. These findings are corroborated by the fact that most studies showing increased satiety after the consumption of low-GI foods but not high-GI foods have observed this effect at 2–6 h after the ingestion of the preload[7];
2. human studies testing CHO foods or mixed meals: the overall view is that whatever the method used to assess satiety (subjective or objective method) and despite the possible contribution of some confounding factors (e.g. fibres, palatability) more than half the reviewed studies support an increased short-term satiety with low-GI foods or meals compared with high-GI foods or meals[3]. A short-term study in children has demonstrated that a low-GI breakfast reduces intake at lunch[8].

Effect of the consumption of low-glycaemic-index diets on food intake and body-weight regulation

Only a few studies have attempted to assess the effect of the lowering of glycaemic response on satiety and effects on body weight in the long term. A systematic review has shown inconsistent results, suggesting that the improved satiety observed in certain short-term studies with low-GI foods or meals does not lead to a long-term reduction in spontaneous energy intake and/or regulation of body weight[9]. This notion has been recently underlined by a study that has found no effect on appetite, energy intake and weight loss in volunteers randomised to a low-GI diet[10]. Furthermore, a long-term intervention study has failed to show the appetite regulation found in short-term studies of children exposed to a low-GI diet[11]. However, an overall systematic review does suggest an overall positive effect of low GI on weight loss[4].

Factors confounding the results

There are many factors that may be affecting the results of these long-term studies. The most obvious one is the heterogeneity in foods classified at different GI.

Carbohydrates: the complex group

From simple sugars such as glucose to complex polysaccharides such as amylopectin and cellulose the term 'dietary CHO' embraces a vast array of molecules. Moreover, CHO in unprocessed food have a physical structure such as the cell wall, whereas processed foods may present CHO for which this structure is lost. It is this diversity of not only chemical structure but also physical form that proves problematic in categorising CHO, even before physiological effects are discussed[5,12]. Even within two low-GI diets the interaction between the GI tract and the different CHO making up the diet can be very different despite the similar glucose profile. One major component is fermentable CHO (FCHO). The chemical make-up and the food structure in which a CHO is delivered to the small bowel determine the rate of absorption and the section of the GI tract at which products of CHO digestion are absorbed. Most simple CHO and starch are absorbed rapidly in the small bowel[13]. However, molecules with a 1–4-β link or retrograde starch cannot be digested by amylase so survive the small bowel; they are processed by the microbiota in the colon and the products of this fermentation are absorbed and have a metabolic effect[13]. For the purpose of the present review the focus will be on CHO that are malabsorbed in the small intestine and are fermented by the colonic microbiota. For ease, they will be termed FCHO.

Body composition and fermentable carbohydrates

The first observation that a diet high in FCHO may have an influence on body composition was made in a study of rats fed a high-resistant-starch diet[14]. By week 5 of this diet total body weight was found to be the same as that of the

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control group, but with smaller epididymal fat pads and adipocyte size. This observation was taken further by the demonstration that in rats on a diet high in resistant starch there is no reduction in whole-body weight but there is a reduction in adipose tissue, which is associated with a decrease in insulin and glucose response to a standard glucose load[15]. Studies in mice have confirmed these observations and have demonstrated that a diet high in resistant starch leads to decreased hepatic cellular lipid content along with an increase in adipose tissue insulin-stimulated glucose uptake[16]. This finding suggests that FCHO have a central role to play in adipocyte metabolism and body composition in animals.

In human subjects there are very few data. It has been demonstrated that low-GI diets that are high in FCHO have an influence on adipocyte metabolism. In individuals with CHD and in women with and without risk of coronary disease 3–4 weeks exposure to a low-GI diet increases whole-body insulin sensitivity and insulin-stimulated glucose uptake in the adipocyte[17,18]. This finding suggests that in human subjects low-GI diets with a high FCHO content could have a central metabolic role in body composition and adipocyte metabolism. There is one study that suggests that there is adipose tissue remodelling in human subjects following 5 weeks on a low-GI diet[19].

Obesity and fermentable carbohydrates

Obesity rates have greatly increased in the last 50 years. This period has also seen marked changes in diet. In particular, the quantity and quality of dietary CHO has changed considerably. While the total amount of CHO in a typical Western-style diet has moderately increased, the amount of FCHO has fallen dramatically. FCHO in the current Western diet are derived predominantly from cereal grains, and in many products are refined to increase their digestibility. Thus, contemporary diets contain much lower levels of FCHO compared with intake 50 years ago, which contained a much more diverse range of unrefined FCHO[20]. Recent epidemiological and experimental studies have demonstrated an inverse correlation between dietary fibre intake and body weight and adiposity[21–28]. As outlined earlier, animal experiments have consistently demonstrated that FCHO influence body fat composition, reducing intra-abdominal adipose tissue and hepatic lipid load and increasing insulin sensitivity. Recent studies have shown that large amounts of orally-administered FCHO in food-deprived animals result in a central nervous system neuronal activation pattern similar to that observed in fully-satiated animals compared with animals fed a low-FCHO diet. This neuronal activation pattern appears similar to that observed following infusion of the anorexigenic gut hormones peptide YY (PYY) and glucagon-like peptide-1. Animals fed high doses of FCHO have higher plasma concentrations of glucagon-like peptide-1 than those fed low-FCHO diets[29], suggesting that the changes in neural activation observed may be a result of changes in gut hormone release. The levels of FCHO used in these experiments are often between 7% and 30% of the total weight of the diet. High intake of FCHO has also been shown to improve insulin sensitivity in human subjects. A 1-year study has shown that children consuming a diet supplemented with the FCHO inulin (oligofructose) have greater weight loss than unsupplemented controls[30].

Fermentation and appetite regulation

Over the last few years the role of the gut microbiota in adipose tissue development and energy homeostasis has been championed[31–34]. In the main, the observations have been made using a germ-free mouse model. It has been demonstrated that colonisation of the large bowel of germ-free animals by microbiota from conventionally-raised animals results in a 60% increase in body fat content, suppression of fasting-induced adipocyte factor leading to an increased activation of lipoprotein lipase and increased storage of lipid in adipocytes[31]. Through a series of eloquent experiments the same investigators have suggested that obese human subjects[32]and animals[33,34] display an increase in firmicutes in the microbiota that increase fermentation and so enhance energy uptake in the form of SCFA.

In many ways these observation are contradictory to those reported with increasing intake of FCHO in animals and human subjects. The consensus of these studies suggests that supplementing animal feed with FCHO leads to an increase in gut microbiota species that increase fermentation and so increase SCFA production in the large bowel[35–37]. Indeed, the animals seem to have a lower body fat content and when fed obesity-inducing diet they are protected against weight gain[29,38]. The adipocytes of these animals tend to be smaller and more insulin sensitive. Recent data from a human study show that long-term feeding of FCHO results in weight loss and an enhanced release of the anorectic gut hormone glucagon-like peptide-1[39]. Thus, it could be suggested that another factor comes into play.

SCFA, GPR43, L-cells and appetite regulation

GPR43 is a SCFA receptor. In 2003 it was demonstrated that SCFA act as ligands for GPR43[40] (formerly orphaned G-protein coupled receptor). The GPR43 receptors are present on the luminal side of the L-cell in the rodent and human colon[41,42]. GPR43 has a particularly high affinity for the SCFA propionate[43], which is produced in the colon by the fermentation of CHO. SCFA produced during fermentation in the colon may be responsible for the changes in body composition and insulin sensitivity observed following FCHO administration in animals. In particular, colonic production of the SCFA propionate appears to play a critical role. Propionate is believed to have direct effects on L-cells in the colon and systemic effects at the level of the adipocyte[43,44].

The L-cell, the most abundant endocrine cell in the intestine, synthesises and releases the anorexigenic gut hormones glucagon-like peptide-1, PYY and oxyntomodulin. These three hormones are released in the physiological response to food and have been shown to inhibit food intake in animals and human subjects[45]. Bariatric

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surgery is thought to cause sustained weight loss by increasing endogenous anorectic gut hormone secretion[46]. Propionate induces activation of GPR43 on enteroendocrine L-cells and stimulates PYY[41] release *in vitro*. In animals FCHO ingestion stimulates L-cells to release glucagon-like peptide-1 and PYY, resulting in a reduction in food intake[47]. This effect is replicated by propionate in several mammalian species, suggesting that propionate production is at least in part responsible for mediating the effects of FCHO on appetite and gut hormone release. GPR43 receptors have recently been shown to be present on PYY3–36-producing enteroendocrine L-cells in the human colon[48]. Few human studies have demonstrated the effect of FCHO on appetite[49,50], which could be because the high concentration of SCFA is needed to trigger a gut hormone response. The normal SCFA concentration in the colon is 100 mM and the release of PYY occurs at 300 mM[51], which may explain the differences between the consistent animal observations that feed containing >7% (w/w) of a FCHO produces high concentrations of PYY, reduction in body weight, reduction in adipose tissue and improved insulin sensitivity. In human studies levels are often ≤15 g/d (<1% total weight of food consumed). Levels of 300 mM are only likely to be reached following high FCHO intake similar to those of man's ancestors of 100 g/d[51]. This factor has been highlighted in a recent study in which weight loss has been reported following a high intake of fructans (21 g/d), which is associated with an increase in the gut hormone PYY[52].

Discussion

There is convincing evidence from acute meal studies that the GI of meals is related to subjective measures of appetite regulation and at free meal intake following a standard meal. However, the findings from longer-term studies are unclear. The reason may be related to the amount of FCHO in the low-GI diet. In animal studies there seems to be clear evidence that FCHO added to feed increases PYY release and leads to a reduction in body weight. These observations may be a result of the enhanced production of SCFA from fermentation stimulating the release of PYY from the enteroendocrine L-cell through GPR43. In human subjects the results from studies are mixed, perhaps indicating that the amount of FCHO used is not sufficient to trigger an appetite response. One recent paper suggests that large intakes of FCHO may have a role to play in body weight control[52]. The design of the low-GI diets and the amount of FCHO may be critical to their role in appetite and weight loss.

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