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Potential anti-obesogenic properties of non-digestible carbohydrates: specific focus on resistant dextrin

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Alterations in the composition and metabolic activity of the gut microbiota appear to contribute to the development of obesity and associated metabolic diseases. However, the extent of this relationship remains unknown. Modulating the gut microbiota with non-digestible carbohydrates (NDC) may exert anti-obesogenic effects through various metabolic pathways including changes to appetite regulation, glucose and lipid metabolism and inflammation. The NDC vary in physicochemical structure and this may govern their physical properties and fermentation by specific gut bacterial populations. Much research in this area has focused on established prebiotics, especially fructans (i.e. inulin and fructo-oligosaccharides); however, there is increasing interest in the metabolic effects of other NDC, such as resistant dextrin. Data presented in this review provide evidence from mechanistic and intervention studies that certain fermentable NDC, including resistant dextrin, are able to modulate the gut microbiota and may alter metabolic process associated with obesity, including appetite regulation, energy and lipid metabolism and inflammation. To confirm these effects and elucidate the responsible mechanisms, further well-controlled human intervention studies are required to investigate the impact of NDC on the composition and function of the gut microbiota and at the same time determine concomitant effects on host metabolism and physiology.

**Obesity: Prebiotics: Resistant dextrin: Metabolism: Non-digestible carbohydrates**

Obesity

Worldwide, the prevalence of overweight (BMI 25–29·9 kg/m^2) and obese (BMI >30·0 kg/m^2) individuals has increased from 857 million in 1980 to 2·1 billion in 2013[1]. In the UK population, it is estimated that 26 % of boys, 25 % of girls, 67 % of men and 57 % of women are currently overweight or obese[1]. Characterised by the accumulation of excess body fat, overweight and obesity are associated with a chronic low-grade systemic inflammation and other adverse metabolic effects. Consequently, the risk of pathologies including CVD, type 2 diabetes mellitus, chronic obstructive pulmonary disease, colon cancer, breast cancer, osteoarthritis, liver and gall bladder disease and reproductive dysfunction, are increased[2]. Overweight and obesity are also thought to increase the risk of common cognitive issues, such as anxiety and depression[3].

Population-based interventions to reduce the prevalence of overweight and obesity are now implemented as part of wider public health strategies in the majority of developed countries worldwide. In the UK, the Department of Health aims to achieve, by 2020, a sustained downward trend in the level of excess weight in children and a downward trend in the level of excess weight averaged across all adults[4]. Such interventions focus primarily on encouraging healthier food choices and increasing physical activity; however, they must compete with the

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Abbreviations: AXOS, arabinoxylan-oligosaccharides; FFAR, free-fatty acid receptor; GLP-1, glucagon-like peptide-1; FOS, fructo-oligosaccharides; NDC, non-digestible carbohydrate; PYY, peptide YY.
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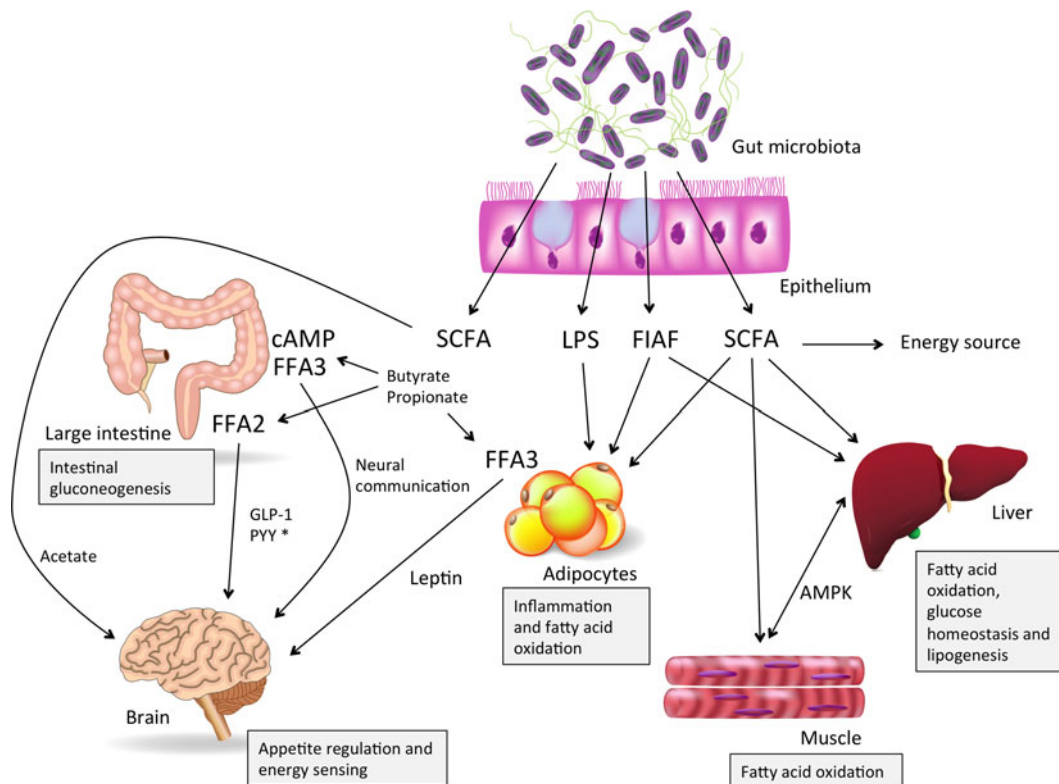
Fig. 1. (Colour online) The gut microbiota influences various regulatory processes associated with obesity through a multitude of metabolic pathways. SCFA may alter appetite regulation via the activation of free-fatty acid receptors 2 and 3 (FFAR2 and FFAR3) and release of glucagon-like peptide 1 (GLP-1), peptide YY (PYY) and leptin. *GLP-1 and PYY also impact on glucose homeostasis. Acetate may also influence appetite through a central homeostatic mechanism. SCFA act via cyclic AMP (cAMP) and FFAR3-dependent mechanisms to alter intestinal gluconeogenesis and subsequent host energy signalling. SCFA affect fatty acid oxidation in various tissues through increased AMP-activated protein kinase activity. Suppression of fasting-induced adipocyte factor (FIAF) may increase hepatic lipogenesis and lipoprotein lipase activity in adipocytes. Lipopolysaccharide (LPS) endotoxin release into the circulation increases pro-inflammatory cytokine secretion and resultant inflammation in adipocytes.

obesogenic environment in which most human subjects now live, with energy-dense foods and drinks being easily accessible and sedentary lifestyles commonplace[5]. Over the past few decades, there has been increasing interest in the potential weight management properties of functional foods and food ingredients, and specifically their ability to affect host metabolism and eating behaviours[6–10]. Evidence indicates that certain types of non-digestible carbohydrate (NDC) are able to selectively modulate the microbial inhabitants of the gastrointestinal tract (known as the gut microbiota) and may provide benefits for the prevention and treatment of obesity and associated diseases[11,12].

Gut microbiota and host metabolism

The gut microbiota comprises at least 10^{14} bacteria and more than 1000 different species and is the most densely populated bacterial ecosystem in the human body[13]. This complex bacterial community resides primarily in the large intestine, an organ supportive of bacterial growth, involving slow transit speeds, anaerobic

conditions, abundance of nutrients and a more neutral pH compared with that found in the upper regions of the gastrointestinal tract[14]. The gut microbiota plays a pivotal role in host physiology and metabolism, affecting localised and systemic processes and facilitating cross-talk between major organs of the human body[12]. Importantly, many metabolic parameters influenced by the gut microbiota are fundamental processes in the development of obesity and associated diseases, such as energy extraction from the diet, appetite regulation, glucose and lipid metabolism and inflammation (Fig. 1). The gut microbiota is also involved in the development and regulation of the immune system[15], synthesis of vitamins B[16], K[17] and folate[18], host absorption of minerals[19], and metabolism of bile acids[20] and foreign chemical compounds (xenobiotics)[21].

Dietary NDC escape digestion in the upper gastrointestinal tract and are available as substrates for fermentation by the gut microbiota. The main products of this fermentation process are SCFA, principally acetate, butyrate and propionate, which contribute energetic value to the host that may account for up to 10 % of overall energy intake depending on the amount of NDC consumed

in the diet and the composition of the gut microbiota[22]. Further to their role in extracting energy from the diet, SCFA are also important signalling molecules and exert many of their effects through activation of G-protein-couple receptors, free-fatty acid receptors 2 and 3 (FFAR2 and FFAR3), respectively, which are present in endocrine L cells, immune cells and adipocytes[23]. FFAR2 is equally sensitive to propionate, butyrate and acetate, whereas FFAR3 is sensitive in the order propionate ≥ butyrate > acetate[24,25]. Activation of FFAR2 on endocrine L cells by butyrate and propionate has been shown to induce the release of anorexigenic (appetite regulatory) hormones, glucagon-like peptide-1 (GLP-1) and peptide YY (PYY)[26,27]. Recent data suggest that SCFA-stimulated release of anorexigenic hormones in the colonic mucosa is not dependent on FFAR3 activation[28]; however, activation of FFAR3 on adipocytes may stimulate the release of leptin, another anorexigenic hormone[29]. A mechanistic investigation has shown that acetate may also suppress appetite, not through the activation of FFAR, but rather by crossing the blood–brain barrier and acting via a central homeostatic mechanism[30]. The findings of a recent study in mice have also highlighted the importance of butyrate and propionate in modulating intestinal gluconeogenesis, specifically intestinal gluconeogenesis gene expression, which has subsequent effects on glucose control and insulin sensitivity[31]. Butyrate and propionate appear to activate intestinal gluconeogenesis gene expression via separate mechanisms, with butyrate acting through a cyclic AMP-dependent mechanism, whereas propionate acts via a gut–brain neural circuit involving FFAR3. Importantly, in this study it was also shown that metabolic effects on glucose homeostasis were absent in mice deficient for intestinal gluconeogenesis. Together, these data highlight the importance of SCFA in the biochemical signalling between the gut and the brain (gut–brain axis)[32].

SCFA produced by the gut microbiota are also capable of elevating fatty acid oxidation in liver, muscle and brown adipose tissue through increases in AMP-activated protein kinase activity[33–35]. Furthermore, SCFA stimulated secretion of PYY and GLP-1, together with increases in hepatic AMP-activated protein kinase phosphorylation and activity, are thought to play an important role in glucose metabolism[36]. Gut microbial suppression of fasting-induced adipocyte factor may also act to increase hepatic lipogenesis and lipoprotein lipase activity in adipocytes, thus promoting adiposity[37]. The composition and metabolic activity of the gut microbiota also impact on local and systemic inflammation. Changes in gut barrier integrity[38], chylomicron formation[39] and alkaline phosphatase activity[40], all appear to contribute to lipopolysaccharide endotoxin release into the circulation, a condition known as metabolic endotoxemia[41]. Importantly, increases in lipopolysaccharide plasma concentrations promote the secretion of pro-inflammatory cytokines and inflammation in adipose tissue, which may contribute to the development of metabolic disorders and obesity[41]. For reviews on the interactions between the gut microbiota

and host metabolism, see Nicholson *et al.*[11] and Geurts *et al.*[42].

The composition and metabolic activity of the gut microbiota are influenced by a range of host characteristics, including genetic background[43], age[44], sex[45], diet[46], physical activity levels[47], medication usage[48], gastrointestinal surgery[49], geographical location[45] and delivery mode at birth[50]. Shifts in the composition of the gut microbiota have been, perhaps rather simplistically, categorised as either towards a state of 'dysbiosis', in which bacterial genera/species with potentially harmful or pathogenic effects predominate over those with positive properties, or towards a state of 'normobiosis', in which the opposite is true[14]. Obesity and associated metabolic diseases are associated with changes in the composition of the gut microbiota that may reflect a state of 'dysbiosis' and thus contribute to the pathogenesis of the condition[51,52].

Gut microbiota in lean *v.* obese individuals

Studies in rodents and human subjects provide evidence that obesity and diet-induced weight-gain are associated with an altered gut microbial composition at a range of taxonomic levels. The taxonomic classification is a hierarchal system used to classify living organisms, such as bacteria. Here compositional changes at higher levels (phylum) and lower levels (genus and species) will be discussed. The first evidence that obesity was associated with compositional shifts in the gut microbiota came from a study in mice, in which genetic obese (*ob/ob*) mice were found to have fewer Bacteroidetes and more Firmicutes than their lean counterparts[53]. However, findings of subsequent studies in this area have been inconclusive, particularly in the case of Bacteroidetes. Although obesity-related reductions in Bacteroidetes have been observed in some studies[52,54,55], another study found no difference[56] and in two studies numbers were elevated in obese compared with lean human subjects[57,58]. The majority of data suggest that Firmicutes are increased in obese *v.* lean individuals[54,58]. An increase in this phylum is supported by evidence that SCFA production is increased, and thus the capacity to extract energy from the diet is enhanced, in obese individuals[51].

An abundance of *Bifidobacterium* spp. has been shown to be inversely correlated with obesity and increases in body weight[59]. In a recent study it was found that obese women have significantly fewer *Bifidobacterium* spp. and significantly more *Enterobacteriaceae, Staphylococcus* and *Escherichia coli* than normal weight women[60]. Importantly, *Bifidobacterium* spp. are associated with improved mucosal barrier function and reduced metabolic endotoxemia in mice and rats[61,62]. Obesity and diet-induced weight gain are also associated with reductions in *Clostridium* cluster XIVa[63], *Roseburia* spp.[63,64], *Faecalbacterium prauntitzii*[65] and *Akkersmansia mucciniphilia*[60,66]. *Faecalbacterium prausnitzii, A. mucciniphilia* and *Roseburia* spp. are of particular interest in the context of obesity. *F. prausnitzii* and

Table 1. Physicochemical structures of various fermentable non-digestible carbohydrates (NDC) of interest for obesity and associated diseases

NDC	Source(s) and production method	Unit length	Structure (type of units in **bold**)	Reference(s)
Inulin	Jerusalem artichoke, chicory root, banana, leeks, garlic, agave and onions	2–60	$\beta(2\rightarrow1)$ linked **fructose** and terminal **glucose**	van Loo et al.[80] and Paeschke & Aimutis[108]
FOS/oligofructose	Hydrolysed chicory root extract or enzymatic synthesis from sucrose	3–9	$\beta(2\rightarrow1)$ linked **fructose** and terminal **glucose**	Paeschke & Aimutis[108]
GOS/TOS	Enzymatic synthesis from lactose	2–8	Varies between manufacturers: **Galactose** linked by $\beta\ (1\rightarrow4)$, and/or $\beta\ (1\rightarrow6)$, and/or $\beta\ (1\rightarrow3)$	Paeschke & Aimutis[108]
Resistant dextrin	Dextrinisation and repolymerisation of maize or wheat starch	12–25	**Glucose** linked by α and $\beta\ (1\rightarrow2)$, $(1\rightarrow3)$, $(1\rightarrow4)$, $(1\rightarrow6)$	Le-franc-Millot et al.[109]
Chitin-glucan	Extracted from the cell wall of fungi	N/A	Poly N-acetyl-D- **glucosamine** and β $(1,3)$-D-**glucan**	Neyrinck et al.[64]
Arabinoxylans	Grain-based materials, most often wheat or maize	Depends on the source	$\beta(1\rightarrow4)$ linked **xylose** and side chains of **arabinose**	Paeschke & Aimutis[108] and Izydorczyk & Dexter,[110]
AXOS	Hydrolysed arabinoxylan	Up to 60; dependent on production conditions	Oligosaccharides from arabinoxylan (see earlier)	Paeschke & Aimutis[108] and Swennen et al.[111]

FOS, fructo-oligosaccharides; GOS, galacto-oligosaccharides; TOS, *trans*-galacto-oligosaccharides; AXOS, arabinoxylan-oligosaccharides.

Roseburia spp. are two main contributors to butyrate production in the large intestine[67]. *F. prausnitzii* has also been shown to exert anti-inflammatory effects in a mouse model of inflammation[68]. *A. muciniphila* is important for its role in mucin degradation and production of propionate and acetate[69]. This bacterium has also been shown to modulate expression of intestinal epithelial genes involved in establishing homeostasis in basal metabolism[70]. In addition to changes in the relative abundance of specific bacterial groups, obesity is also associated with changes in diversity of the gut microbiota. A study which utilised 16S rRNA gene surveying in thirty-one monozygotic twin pairs and twenty-three dizygotic twins found that obese twins had reduced phylogenic microbial diversity compared with their lean sibling[52]. Changes in the composition and diversity of the gut microbiota may be attributed to changes in host bodyweight and also differences in dietary intake. Accordingly, it is difficult to differentiate as to whether the changes to the microbiota are due to adiposity, as a result of dietary intake (such as high-fat intake) or a combination of the two. Furthermore, there is presently a lack of evidence to imply a causal relationship between the gut microbiota and obesity. Nevertheless, studies in mice provide some evidence that changes in the gut microbiota may contribute towards reduced host weight and adiposity[49]. Germ-free mice (mice raised under sterile conditions without any microbes of their own) administered with gut microbial samples from an obese human twin, gained significantly more body fat than those receiving the microbiota from a lean human twin, irrespective that all mice followed a standardised diet. Analysis of the faecal samples found metabolic changes similar to those found in obese human subjects, including an increase in branched-chain fatty acid production[71].

Furthermore, evidence that fermentable NDC improve weight management in rodents and human subjects provides further support that compositional changes in the gut microbiota may contribute to the development of obesity[64,66,72–77].

Physicochemical structure of non-digestible carbohydrates

Non-digestible oligosaccharides and non-digestible polysaccharides are two types of NDC. Oligosaccharides contain a small number (two to about ten) of monosaccharide units, connected by glycosidic linkages, whereas polysaccharides contain more than ten monosaccharide units[78]. Accordingly, the molecular weight of polysaccharides is much higher than that of oligosaccharides[79]. Non-digestible oligosaccharides include inulin-type fructans, which are found naturally in small quantities in certain foods, such as Jerusalem artichoke, chicory root, banana, leeks, garlic, agave and onions[80]. Non-digestible polysaccharides include dietary fibre and resistant starch, which are consumed in much higher amounts in the diet[81]. Further to differences in monosaccharide unit length, NDC vary considerably in other aspects of physicochemical structure, including type of monosaccharide unit and the position and type of linkages (Table 1). The physicochemical structure of the carbohydrate determines its physical (viscosity and solubility) and fermentable properties in the large intestine. The exact mechanisms by which gut bacteria break down carbohydrates are not known; however the process is dependent on bacterial enzymes that demonstrate specificity to structural configurations and the position and type of linkages[82].

Gut bacterial fermentation of non-digestible carbohydrates

Inulin, fructo-oligosaccharides (FOS) and galacto-oligosaccharides are NDC with established prebiotic effects and can be defined as 'selectively fermented dietary ingredients that result in specific changes in the composition and/or activity of the gastrointestinal microbiota, thus conferring benefit(s) upon host health'[83]. Favourable effects on gut microbial ecology include increased numbers of beneficial bacteria, reduced numbers of pathogenic bacteria (e.g. *E. coli, Staphylococcus aureus, Campylobacter jejuni, Clostridium difficile* and *Clostridium perfringens*), reductions in intestinal pH (promoting a more favourable environment for microbial growth), increased production of metabolic end-products (SCFA) and altered bacterial enzyme concentrations[84]. Much research in this area has focused on the ability of NDC to stimulate the proliferation of *Bifidobacterium* spp. and *Lactobacillus* spp.[85]. Although inulin, FOS and galacto-oligosaccharides have all been shown to increase the abundance of *Bifidobacterium* spp. in *in vitro* gut model systems and human studies, there is increasing interest in NDC that are able to increase the proliferation of other bacterial genera, including *Eubacterium, Faecalibacterium* and *Roseburia*[14]. FOS, resistant dextrin and arabinoxylan-oligosaccharides (AXOS) have shown the ability to increase these bacterial groups and further studies are required to determine whether other NDC also target these bacterial groups. In a recent study the administration of a resistant dextrin produced from wheat starch (14 g/d) to an *in vitro* gut model system was found to elevate levels of *C. cluster* XIVa and *Roseburia* spp., with a concomitant increase in butyrate production after 18 d[86]. Chitin-glucan, a soluble fibre extracted from the cell walls of fungi, has also been shown to increase the abundance of *C. cluster* XIVa and *Roseburia* spp., in mice fed a high-fat diet[63]. Furthermore, the intake of wheat arabinoxylans has been found to significantly increase levels of *Roseburia* spp., together with increases in *Bifidobacterium* spp. and Bacteroides/Prevotella in diet-induced obese mice[64]. However, it should be noted that AXOS, which are obtained by enzymatic treatment of arabinoxylans, have been shown in an *in vitro* model to exert opposing effects, with a significant reduction in *Roseburia* spp. and butyrate production, but a significant increase in propionate production[87]. Additional *in vitro* and *in vivo* studies are required to investigate the effects of resistant dextrin, AXOS and chitin-glucan, on the gut microbiota.

Presently, there is a gap in our understanding of how the physicochemical structure of carbohydrates impacts on gut microbial fermentation. Ultimately, the aim should be to construct a detailed framework of evidence for the different structural properties on gut bacterial fermentation. This will allow for better targeting of specific gut microbial populations in selected regions of the large intestine, i.e. proximal or distal regions. Nevertheless, a number of studies are beginning to provide evidence for the fermentation characteristics of different structural configurations. *In vitro* studies have shown that molecular weight and the monosaccharide unit chain length affect fermentation by the gut microbiota. An *in vitro* study on wheat arabinoxylans found that low molecular mass NDC, with short monosaccharide unit chain length, were more selectively fermented by *Bifidobacterium* spp. and *Lactobacillus* spp.[88]. Moreover, longer chain NDC appear to be more slowly fermented than shorter chain NDC and therefore reach more distal regions of the colon[87,89]. To date, the majority of research has focused on selectivity of *Bifidobacterium* spp. and *Lactobacillus* spp., and therefore more work is required to determine the selectivity of other bacterial groups to NDC with differing molecular weight and monosaccharide chain length. The type of linkages in the NDC also appears to affect the rate of fermentation. NDC with more 1→6 linkages relative to 1→4 linkages are more slowly fermented in the gastrointestinal tract[90]. Accordingly, NDC with a high number of 1→4 linkages and large monosaccharide chain lengths, such as resistant dextrin, long chain AXOS/arabinoxylans and long-chain inulin, may be more slowly fermented and reach more distal regions of the gastrointestinal tract for bacterial fermentation, whereas FOS for example may be fermented primarily in proximal regions of the gastrointestinal tract. Rate of fermentation is an important consideration as this may impact on gas production and onset of side effects associated with high intakes of fructans, such as bloating and digestive discomfort[91]. Resistant dextrin, which may be more slowly fermented, is well tolerated in human subjects, even at high doses of up to 45 g/d[92].

Anti-obesogenic properties of non-digestible carbohydrates

The first studies to examine the potential anti-obesogenic properties of NDC were conducted in rodents[66,93]. While animal studies give some mechanistic insight into the potential benefits of NDC, it should be noted that the gut microbiota of mice and rats is very different from the gut microbiota of human subjects. The majority of intervention studies to date have investigated the effects of inulin-type fructans, including oligofructose and FOS. Kellow *et al.*[94] have recently published a comprehensive systematic review on the metabolic benefits of inulin, oligofructose, FOS or galacto-oligosaccharides in human subjects; however, the present review excluded other NDC that are fermentable by the gut microbiota but are yet to be confirmed as prebiotics. Here, data are presented from studies on the aforementioned NDC, but also from studies that have investigated the effects of other NDC that are not formally classified as prebiotics but may also exert favourable compositional and metabolic effects on the gut microbiota, including arabinoxylans, AXOS, chitin-glucan and resistant dextrin. Non-fermentable NDC were excluded from the present review in order to only provide data on NDC that may exert metabolic and physiological effects primarily via modulation of the gut microbiota.

A number of rodent studies have observed reductions in bodyweight and adiposity following long-term consumption of fructans[66,72]. In a recent study, oligofructose, with or without probiotic *Bifidobacterium animalis*, was given to adult diet-induced obese Sprague-Dawley rats[73]. Oligofructose, but not *Bifidobacterium animalis*, significantly increased the abundance of *Bifidobacterium* spp. and *Lactobacillus* spp. in the gut microbiota. Furthermore, improvements in body composition (reduced weight gain and fat mass) were found after the oligofructose intervention only. Glucose profiles were beneficially altered by the intake of both oligofructose and *Bifidobacterium animalis*. In another study, mice were fed a control diet, high-fat diet or a high-fat diet with wheat arabinoxylans[64]. The intake of wheat arabinoxylans was found to have various metabolic and physiological benefits, including decreased high-fat diet-induced adiposity, body weight gain, serum and hepatic cholesterol accumulation and insulin resistance. To date, only a limited number of studies have investigated the effects of NDC on weight management in human subjects, and there is very little known on the concomitant effects of NDC on gut microbial composition and body composition. A small number of studies have shown that long-term intake of fructans may result in small, but significant, reductions in body weight and adiposity[74,75]. Changes in body weight and adiposity were not observed in a recent study; however this could be due to a shorter treatment duration[95]. One study in obese women found that the intake of yacon syrup, a food naturally rich in fructans, significantly increased weight loss over a 120-d period[76]. However, it should be noted that all volunteers on this study were actively trying to lose weight. Resistant dextrin has also been demonstrated to have weight loss properties. In a study by Guérin-Deremaux *et al.*[77], the daily intake of 34 g resistant dextrin for 12 weeks was found to significantly reduce body weight and percentage body fat in overweight Chinese men.

The intake of fructans in rodent studies has been shown to increase blood concentrations of GLP-1 and PYY, and reduce concentrations of orexigenic hormone ghrelin[75,96,97]. Moreover, the intake of fructans has been found to activate neural receptors in the brain associated with food intake[72]. In a recent murine study, the intake of galacto-oligosaccharides was also found to increase circulating GLP-1 and PYY concentrations. Furthermore, reductions in dietary energy intake and fat-pad weight were observed[98]. In a number of human studies, intake of fructans has been shown to concomitantly increase satiety and alter anorexigenic hormone profiles[95,99,100]. However, not all studies have observed changes in satiety after the intake of fructans[75,101,102]. This is especially true in acute/short-term studies, in which the intervention phase is most likely insufficient in length for the gut microbiota to adapt to the increased fructan intake. The daily intake of either 14, 18 or 24 g resistant dextrin over a period of 9 weeks, has been found to increase satiety and reduce energy intake in a group of overweight males[103]. Importantly, the magnitudes of the responses were dose dependent, highlighting

the importance of dose escalation studies when investigating the effects of NDC. In another study, the intake of 50 g resistant dextrin at breakfast was shown to decrease circulating levels of ghrelin throughout the rest of the day[96]. Although a number of studies have shown changes in circulating hormones, the mechanistic basis for this is still to be confirmed. Many of the authors propose a mechanism dependent on SCFA production; however, for this to be determined, future studies should investigate the effects of NDC on gut microbial composition, metabolic output (SCFA), circulating hormone profiles and measures of satiety. An important consideration when interpreting results from intervention studies is that of the viscosity of the NDC being investigated. Importantly, viscous NDC may affect host metabolism via mechanisms independent of changes to the gut microbiota. Specifically, the intake of highly viscous food ingredients increases stomach distension and delays gastric emptying, thus inducing the release PYY, GLP-1 and cholecystokinin. Release of these anorexogenic hormones contributes to the activation of the ileal brake feedback mechanism, subsequently leading to a reduction in hunger and food intake[104].

A number of studies have also shown that NDC have the potential to affect inflammation and glucose metabolism. A recent human intervention study found that the intake of inulin by obese women increased faecal numbers of *Bifidobacterium* spp., and significantly reduced plasma concentrations of lipopolysaccharide[105]. In another recent study, the intake of *trans*-galacto-oligosaccharides was also found to increase the abundance of faecal *Bifidobacterium* spp. and had a beneficial effect on fasted blood measures of insulin and cholesterol[106]. In mice, the intake of wheat arabonixylans was shown to decrease serum measures of serum and hepatic cholesterol accumulation and insulin resistance[64]. In obese rats, the intake of oligofructose was found to improve glycaemia and reduce insulin levels[73]. Furthermore, in another human study the daily intake of 34 g resistant dextrin for 12 weeks was shown to improve insulin resistance and determinants of metabolic syndrome in a group of overweight men[107]. This included reductions in blood glucose and insulin, increases in HDL cholesterol and reductions in total cholesterol and LDL cholesterol.

Conclusion

Data presented in this review provide preliminary evidence that various fermentable NDC, including resistant dextrin, may alter metabolic processes associated with obesity, including appetite regulation, energy and lipid metabolism and inflammation. To confirm these effects and elucidate the responsible mechanisms, there is a need for well-controlled human intervention studies to investigate the impact of NDC on the composition and function of the gut microbiota and determine concomitant effects on host metabolism and physiology. Furthermore, mechanistic studies are required to determine the influence of carbohydrate physicochemical

structure on the fermentation and metabolic activity of gut bacterial populations. Ultimately, the aim should be to establish a detailed framework of evidence for bacterial fermentation by a range of NDC, as this will enable more effective targeting of specific gut bacterial populations associated with obesity and metabolic diseases.

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Conflicts of interest

L. G. D. is currently employed by Roquette.

Authorship

M. R. H. conceived and wrote the manuscript. L. G. D., I. R., G. R. G. and O. B. K. critically reviewed the manuscript and approved the final version.

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