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Impact of dietary fiber and fat on gut microbiota re-modeling and metabolic health

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A B S T R A C T

Background: Scientific evidence suggests that diet plays a role in obesity and its comorbidities, partly via its interactions with the individual's gut microbiota. Likewise, the individual's microbiota influences the efficacy of dietary interventions to reduce body weight. However, we require a better understanding of the key components of the gut microbiota that are responsive to specific diets and of their effects on energy balance in order to use this information in practice.

Scope and approach: This review provides an up-to-date description of the influence of dietary fibers and fat on gut microbiota and the mechanisms presumably mediating their effects on metabolic health. We also discuss the main knowledge gaps and the need to gain greater understanding of the role of diet-microbe interactions in obesity and the associated comorbidities.

Key findings and conclusions: Dietary fibers are major drivers of gut microbiota composition and function, stimulating the dominance of bacteria able to utilize these substrates as energy source, although effects vary depending on both the type of fiber and the individual's microbiota. However, the key bacteria and the primary and secondary metabolic pathways mediating specific fiber-induced effects on the metabolic phenotype remain unclear, and this information is necessary to personalize fiber-based interventions. The literature also shows that gut microbiota contributes to the adverse consequences of high-fat diets on the metabolic phenotype; however, little is known about the effects of dietary fat type. Further progress is expected from translational approaches integrating controlled dietary intervention human trials, combining functional omics technologies and physiological/clinical endpoints, and mechanistic studies in experimental models. This will ultimately help us to progress towards establishing informed microbiome-based dietary recommendations and interventions, which can contribute to tackling the obesity epidemic and its comorbidities.

1. Introduction

Obesity has reached pandemic dimensions affecting a vast number of people worldwide. In 2014, approximately 39% of adults (1.9 billion) were overweight and 13% of these (600 million) were obese. Moreover, 42 million children under the age of 5 were reported as overweight or obese in 2013 (World Health Organization, 2015). It is well known that obesity is not only associated with populations in high-income countries, but the prevalence is continuously growing in low- and mid-income countries, particularly in urban settings (World Health Organization, 2015). Obesity is a result of an unbalance between energy intake and expenditure, to which over-nutrition and a sedentary lifestyle are major contributors (Coppinger, Jeanes, Dabinett, Vogele, & Reeves, 2010). Obesity is associated with a state of chronic low-grade inflammation, which partly explains the insulin resistance phenotype observed in many obese individuals. In turn, insulin resistance is a component of the metabolic syndrome that often precedes the development of type 2 diabetes (T2D) and cardiovascular disease (CVD) (Jia, DeMarco, & Sowers, 2016). This metabolic inflammation is characterized by infiltration of macrophages and lymphocytes in peripheral tissues. This is accompanied by an increased production of pro-inflammatory cytokines, adipokines, acute-phase proteins and

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other immune mediators as a consequence of the activation of several signaling pathways, including the nuclear factor kappa B (NFκB)/Inhibitor of theκ kinase (IKK), c-jun N-terminal kinase (JNK), protein kinase R (PKR) and the Toll-Like receptors (TLRs) (Gregor & Hotamisligil, 2011). Adipose tissue from obese individuals is considered to be the main contributor to obesity-related metabolic inflammation, with the highest accumulation of infiltrating macrophages and tissue concentrations of cytokines, with similar events occurring in the liver and central nervous system, contributing to systemic insulin resistance (Johnson & Olefsky, 2013).

In the last decade, an increasing number of studies have reported that obesity is associated with alterations in gut microbiota structure, suggesting that specific microbial taxa could be contributing factors to the obesity epidemic, although results are not fully consistent across human observational studies (Sanz, Rastmanesh, & Agostoni, 2013). Animal studies have provided information about the mechanisms by which gut microbiota could play a role in obesity, including contribution to nutrient digestion and absorption and to regulation of immune and neuro-endocrine functions (Moya-Perez, Neef, & Sanz, 2015). Experimental models have also demonstrated that gut microbiota can transmit the obesity-associated metabolic phenotype of its original human host when transferred to a germ-free recipient, providing a first evidence of causality (Turnbaugh et al., 2006). Furthermore, a unique fecal transplantation study in humans has also demonstrated that the transference of feces from a lean donor into subjects with metabolic syndrome beneficially influence glucose metabolism, confirming the causal role of gut microbiota (Vrieze et al., 2010). Nonetheless, the role of gut microbiota in obesity seems largely dependent on diet-microbe interactions due to the fact that diet is a major modifiable factor influencing gut microbiota composition and function (De Filippis et al., 2015; Flint, Duncan, Scott, & Louis, 2015). Indeed, experimental models revealed that such interactions contribute to obesity, for example, by increasing lipid absorption or aggravating adipose tissue inflammation independently of adiposity in the context of diets rich in saturated lipids (Caesar, Tremaroli, Kovatcheva-Datchary, Cani, & Backhed, 2015; Semova et al., 2012). Furthermore, dietary reprogramming of microbiota ameliorates development of metabolic dysfunction despite susceptible genotypes (Ussar et al., 2015). Nevertheless, our understanding of how diet-microbe interactions influence energy balance, eating behavior and obesity in humans is still insufficient to transform this information into practical solutions to tackle obesity-associated disorders.

This review discusses the most recent data regarding the potential role of dietary fiber and fat in remodeling gut microbiota composition and function and, thereby, in programming metabolic health. It also addresses the main limitations that must be overcome to progress our understanding of the microbiome's role in the chain of events causing obesity. Only on gaining a better understanding of the above, will we be able to speed up the translation of this information into informed microbiome-based dietary interventions and recommendations.

2. Impact of dietary fiber on human physiology

2.1. Dietary fiber: role in metabolic health and as main fuel for gut microbiota

Dietary fiber is generally defined as non-digestible carbohydrates plus lignin, which include structurally different components including non-starch polysaccharides, resistant oligosaccharides (e.g. fructo-oligosaccharides [FOS], galacto-oligosaccharides [GOS]) and resistant starch (EFSA NDA Panel, 2010). Prebiotics are defined as dietary fibers that modify the composition and/or metabolic activity of gut microbiota, thereby conferring a benefit to the host (Gibson, 2004; Gibson, Probert, Loo, Rastall, & Roberfroid, 2004). According to this definition, a wide variety of food ingredients can be classified as prebiotics such as GOS, FOS and longer inulin-derived fructans, xylo-oligosaccharides (XOS) and arabinoxylan oligosaccharides (AXOS); however this is based mainly on their impact on gut microbiota rather than on robust evidence of their effects on health-related endpoints (Hutkins et al., 2016). Dietary fiber is not digested by human enzymes and thus it reaches proximal colonic regions, where it constitutes the main energy source for obligate anaerobic bacteria, whose fermentative activity leads to the generation of organic acids (lactic, succinic acid) and short-chain fatty acids (SCFA) (acetate, propionate and butyrate). Consequently, the quantity and quality of fiber is considered to be one of the main dietary determinants of gut microbiota composition and function (Scott, Gratz, Sheridan, Flint, & Duncan, 2013). The current recommendations on dietary fiber intake (25 g per day for adults) are based on their well-known role in regulating bowel habits (frequency of defecation), including native chicory inulin considered to be prebiotic (Hutkins et al., 2016). In addition, there is evidence for a role of dietary fiber and some prebiotics (inulin and oligofructose) in the reduction of dietary glycemic responses and glycemic load, with favorable effects on metabolic risk factors. Furthermore, consumption of fiber-rich diets with fiber intake above recommendations is associated with a reduced risk of coronary heart disease and type 2 diabetes as well as improved weight maintenance (Bes-Rastrollo, Martinez-Gonzalez, Sanchez-Villegas, de la Fuente Arrillaga, & Martinez, 2006; EFSA NDA Panel, 2010; Liu et al., 2000; Ludwig et al., 1999; Ye, Chacko, Chou, Kugizaki, & Liu, 2012). Dietary fiber is thought to positively influence metabolic health through multiple mechanisms, although effects cannot be generalized as they vary depending on the type of fiber. The mechanisms of action include direct effects related to its physicochemical and structural properties (e.g. indigestibility, viscosity, etc.) and indirect effects mediated by the individual's gut microbiota. For example, compared to digestible carbohydrates, insoluble and soluble fibers reach distal portion of colon with no major degradation by human enzymes leading to a significant reduction in postprandial glycemic responses due to their slower digestion (EFSA, 2014). Consequently, consumption of fiber improves the glucose metabolism as a whole, which have direct impact on satiety and tip the balance towards oxidation instead storage metabolism (reviewed in (Koh-Banerjee & Rimm, 2003)). Moreover, dietary fiber is considered to be very useful for weight loss/maintenance aims given its low energetics estimated to be ~1.91 kcal/g (8 kJ/g) in comparison with other macronutrients as digestible carbohydrates, (~4.06 kcal/g), proteins (~4.06 kcal/g), and fat (~8.84 kcal/g) (Menezes et al., 2016). Soluble viscous fibers may also exert beneficial metabolic effects by their ability to form gels that delay gastric emptying, inhibit nutrient absorption and bile acid (BA) binding; altogether this may contribute to a decreased postprandial glycemic response and a reduction in body cholesterol stores due to increased synthesis of new BAs from cholesterol in the liver (Dikeman & Fahey, 2006). In addition, dietary fiber is thought to mediate other effects (e.g. satiety and anti-inflammatory effects) through activation of the fermentative activity of gut bacteria, and the generation of potentially beneficial metabolites (e.g. SCFAs), as explained in greater detail in section 3.

2.2. Evidence of the influence of dietary fiber on gut microbiota from observational studies

The role of non-digestible carbohydrates in the gut microbiota is well exemplified by the differences in the infant's gut microbiota