ELSEVIER

Trends in Food Science & Technology 22 (2011) 457–464

TRENDS IN FOOD SCIENCE & TECHNOLOGY

Review

Probiotics intake and metabolic syndrome: A proposal

Cristina Stewart B. Bogsan[a], Ana Carolina R. Florence[a], Natalia Perina[a], Ricardo C. Barbuti[b], Tomás Navarro-Rodriguez[b], Jaime N. Eisig[b] and Maricê N. Oliveira[a,*]

[a]Department of Biochemical Pharmaceutica Technology, Faculty of Pharmaceutical Sciences, University of São Paulo, Av. Prof. Lineu Prestes, 580, B16, 05508-000 SP, Brazil (Tel.: +55 11 3 fax: +55 11 38156386; e-mail: monolive usp.br)

[b]Department of Gastroenterology, Clinical Ho it Faulty of Medicine, University of S Paulo, B zil

Probiotics are practical tools to pro the odulat of microbiota. The ingestion of food ingred ts containing anti-inflammatory activity compon nts as probiot should be useful in obesity control and as d co-morbidities treatment. Metabolic syndrome is metabo dysfunction associated with visceral obesity and insulin resista in which the alterations in host–micro ota interactions play an important role. Besides diet and ph ical act ity, new strategies are necessary to control metabo syndr me, and as consequence improving quality of life. Thi ticle re ses the actual knowledge concerning p ic intake obesity as a proposal to control metab ic syn ome.

Introduction

Probiotics are l microorganisms that when administrate in adequate amounts confer health benefit on the host (FAO/WHO, 2002). The benefits include immunomodulation, antagonistic activity towards gastrointestinal pathogens, effects on cholesterol and lactose metabolism and antimutagenic and anticarcinogenic properties (Vasiljevic & Shah, 2008). Probiotics an a produce bacteriocins and organic acids and prom e the reduc n of pathogenic bacteria adherence to the e helial cells (tteland, Brunser, & Cruchet, 2006).

Obes is a chro di ase characterized by excessive accum ation o fat (Car alho, Dutra, & Araújo, 2009). Its etyn ogy is complex and multi factorial, resulting from in ract of gene environment, lifestyle and emotional ctors. Bes re esenting a risk factor for many chronic iseases, obesity s associated with dyslipidemia, diabetes, hyp ension and vascular hypertrophy left, which are coronary ri factors. Metabolic syndrome (MetS), also known as syndrome X, is a condition characterised by elevated waist circumference, elevated triglycerides, reduced HDL-ch sterol, elevated blood pressure and elevated glycaemia pro es. This syndrome is typically associated with being o rweight or obese and also relates to conditions leading to type 2 diabetes and cardiovascular diseases (Duvnjak & Duvnjak, 2009). The prevalence of obesity has increased dramatically worldwide, mainly in the past three decades, becoming a pandemic. Not only more and more adults become obese, but also children and adolescents (Hill, Wyatt, Reed, & Peters, 2003; Kalliomaki, Collado, *et al*., 2008; Kalliomaki, Salminen, *et al*., 2008).

There has been little long-term success in treating established obesity through changes in lifestyle. Perhaps, due to the large permanent changes in diet, physical activities are required to keep weight. An alternative strategy to address the obesity epidemic involves not only weight loss but promoting small changes to prevent the beginning of weight gain (Hill, 2009). In contrast, treating established obesity through lifestyle modification has proven to be extremely difficult (Tsai & Wadden, 2005), and those few who do succeed have made dramatic changes in their diet and physical activity patterns (Hill & Wyatt, 2002). Most people who achieve weight loss through lifestyle modification regain most of the weight lost over time (Tsai & Wadden, 2005).

Recently, there are evidences that alterations in human gut microbiota impacts on the development of obesity, owing mainly to differences found in obese, non-obese and type-2 diabetes microbiota (Cani & Delzenne, 2009a; Raoult, 2008). Moreover, reduction in quality of health and life justifies the production of functional foods, which may have

* Corresponding author.

0924-2244/$ - see front matter
doi:10.1016/j.tifs.2011.05.006

increased its acceptability when related to the aid in consumer weight loss and could influence appetite and satiety and reduce the risk of diabetes (Verschuren, 2002).

Concerning the higher increase in obesity in worldwide and the evidences that probiotic bacteria intake could be a useful tool in metabolic syndrome control, this review focus on the actual knowledge about intestinal microbiota, probiotic intake and obesity. The possible mechanisms involved in obesity and metabolic syndrome control due to probiotic intake are also discussed.

Visceral obesity

Visceral obesity is characterized by excess fat storage in and around the abdomen; it is the prime cause of the metabolic abnormalities, is characterized as a chronic low-grade inflammation, in which the adipose tissue develops a main regulatory role and therefore represents an important target in the treatment of MetS (Matsuzawa, 2006). The development of obesity is a complex process involving genetic and environmental factors. Several genes are related in the determination of body weight, affecting appetite, energy, and metabolic functions (Cecil, Tavendale, Watt, Hetherington, & Palmer, 2008).

According to Hill (2006) and Jernas *et al.* (2006), visceral obesity results from disequilibrium in the energy balance - energy intake, energy expenditure, and energy storage. The excess energy is primarily stored in adipose tissue as triglycerides. Although, adipocytes are specifically designed to store energy and easily fill up with fat, the morphological changes associated with adipose tissue growth have no consequences for the organism as a whole.

The amount of liver fat is determined by the balance between fatty acid uptake, endogenous fatty acid synthesis, triglyceride synthesis, fatty acid oxidation, and triglyceride export. Changes in any of these parameters affect the amount of fat stored in liver. The excessive fat accumulation in adipose tissue, liver, and other organs strongly predisposes to the development of metabolic changes that increase overall morbidity risk. The metabolic abnormalities that often accompany obesity include hypertension, impaired glucose tolerance, insulin resistance leading to hyperinsulinemia and dyslipidemia (Stienstra, D'Orval, Kersten, & Muller, 2007).

Visceral obesity and inflammation

The link between obesity and inflammation was first established by Hotamisligil, Shargill, and Spiegelman (1993) with the positive correlation between adipose mass and expression of the pro-inflammatory cytokine tumor necrosis factor-α (TNFα). It was illustrated that increased plasma levels of several pro-inflammatory markers including cytokines and acute phase proteins like C-reactive protein (CRP) in obese individuals. Many of the inflammatory markers found in plasma of obese individuals appear to originate from adipose tissue suggesting that obesity is a state of chronic low-grade inflammation initiated by morphological changes in the adipose tissue (Trayhurn & Wood, 2005).

One consequence of the elevated inflammatory status is insulin resistance. Pro-inflammatory cytokines originating from fat have been shown to directly interfere with insulin signalling pathways (Greenberg and Obin, 2006). Besides, TNFα, adipose tissue produces a host of other adipokines with well-described effects on metabolism and inflammation. Resistin, adiponectin, leptin, monocyte chemoattractant protein-1 (MCP-1) and in a group of secreted proteins from adipose tissue with immunomodulating functions (Yu & Ginsberg, 2005). The production and secretion of these adipokines are altered during obesity, resulting in a more pro-inflammatory and thrombogenic secretion profile (Kadowaki & Yamauchi, 2005).

Although increased visceral fat depots (Matsuzawa, 2006) and adipocyte hypertrophy had been linked to a higher degree of adipose inflammation, until recently the exact pathways leading to pro-inflammatory state of adipose tissue in obese individuals remained unidentified. However, recently much attention has been diverted to the role of macrophages. Xu *et al.* (2003) and Weisberg *et al.* (2003) showed that diet-induced obesity is associated with infiltration of macrophages into white adipose tissue. Infiltrated macrophages, which are part of the stromal vascular fraction of adipose tissue, are subsequently responsible for the production of a wide variety of pro-inflammatory proteins including MCP-1, TNFα, and interleukin-6 (IL-6). The development of insulin resistance in adipocytes was initially linked to the infiltration of macrophages. However, if and how entry of macrophages into white adipose tissue (WAT) leads to systemic insulin resistance remains unclear, although it is increasingly believed that altered secretion of adipokines by WAT during obesity may represent an important piece of the puzzle. One of the other tissues that is affected by the enlargement and pro-inflammatory secretion profile of adipose tissue is the liver. Chronic activation of the master regulator of inflammation nuclear factor-κB (NF-κB) by cytokines has been directly linked to the development of insulin resistance in liver (Arkan *et al.*, 2005; Cai *et al.*, 2005). It has also been shown that adipose-specific over expression of MCP-1 increases hepatic triyglyceride content (Kanda *et al.*, 2006). Although steatosis is a common occurrence in obese individuals, the role of inflamed adipose tissue in development of steatosis needs further exploration. The molecular mechanisms underlying the development of steatosis and progression to steatohepatitis remain poorly understood.

Visceral obesity and gut microbiota

The physiologic processes that regulate weight and metabolism, including peripheral hunger and satiety signals, the central integration of this information, and the integrated gastrointestinal response to food intake, have received intense investigation, particularly during the past decade (Camilleri *et al.*, 2006; Murphy *et al.*,2006).

Recent evidence suggests that the trillions of bacteria that normally reside within the human gastrointestinal tract,