Chapter

Gut Microbiota and Obesity: Prebiotic and Probiotic Effects

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Abstract

Malnutrition through excessive food consumption is a worldwide pandemic. Changes in lifestyle, diet and physical activity have resulted in an exponential increase in the number of obese people around the world. Multiple factors influence the development of this disease, and recently it has been suggested that gut microbiota (GM) plays an important role in nutrient absorption and energy regulation of individuals, thus affecting their nutritional status. It has been proven that gut microbiota is different in individuals with a normal nutritional status compared to those who are obese. Therefore, to study bacterial populations that make up the microbiota and to understand how prebiotics and probiotics affect the increase of these bacteria has become a promising alternative to treat obesity. This chapter looks at defining the established relationship between probiotics, prebiotics and gut microbiota that develop in obese people and people of normal weight, with the aim of providing future dietary recommendations to treat this medical condition.

Keywords: gut microbiota, prebiotics, probiotics, obesity

1. Introduction

Currently, obesity is defined as a medical condition characterised by an energy imbalance as a consequence of energy intake bigger than energy expenditure [1]. The US Centers for Disease Control and Prevention (CDC) defines it as 'excessive weight that is considered unhealthy for a given height'. It was not until 1997 that the World Health Organization (WHO) described obesity as an excessive accumulation of body fat that can be harmful to people's health [2].

One of the criteria to diagnose obesity is through the calculation of body mass index (BMI); the result, according to the WHO, must be equal to or bigger than 30 kg/m^2 [2].

However, the Asian population including Korea, given their increased risk of developing comorbidities, such as cardiovascular diseases and type 2 diabetes, agreed to an overweight category with a BMI between 23 and 24.9 kg/m^2 and obesity with a BMI > 25 kg/m^2 (**Table 1**) [3].

Worldwide obesity is a public health problem, and despite all the strategies designed and implemented to reverse the situation, these have not been effective. According to the World Health Organization, globally, in 2016, more than

	WHO (BMI)	Asia-Pacific (BMI)
Underweight	<18.5	<18.5
Normal	18.5–24.9	18.5–22.9
Overweight	25–29.9	23–24.9
Obese	≥30	≥25

Table 1.
Comparison of the World Health Organization (WHO) and Asia-Pacific body mass index (BMI) classifications in COPD patients [3].

1900 million adults were overweight, over 650 million were obese, and 41 million children under the age of 5 were overweight. Fifty-seven percent of the world's population lives in countries where being overweight and obesity cause more deaths than being underweight. Furthermore, each year at least 2.8 million people die worldwide due to obesity or being overweight, irrespective of whether the country is economically developed or not [1, 2].

Various studies have shown that patients with illnesses related to the gastrointestinal tract, such as type 2 diabetes, obesity and cardiovascular diseases, among others, present dysbiosis or an imbalance within the gut microbiota (GM).

A hypothesis of the positive influence of microbiota on fat storage exists, and several studies carried out in mice have proven the role of certain fatty acids in the diet in the prevention of obesity, an effect mediated through changes in the composition of gut microbiota. Studies in humans have also shown a relationship between a decrease in *Bacteroidetes* in obese individuals and a greater proportion of these in the faecal microbiota in subjects who have undergone hypocaloric diets and who have managed to reduce their weight [5, 7].

An important role of the regulation of gut microbiota has been proven as the ingestion in the diet of prebiotics and probiotics that modulate the growth of diverse families of bacteria that could influence the nutritional status of individuals.

2. Obesity as a multifactorial condition

Obesity is a medical condition of multifactorial causes, ranging from the influence of the nearby environment on the acquisition of predisposing habits, such as the consumption of foods high in refined carbohydrates and saturated fats and a sedentary lifestyle. Genetic predispositions are also among the principle causes [5]. On the other hand, socioeconomic development, transport, urban planning, environmental, agricultural, educational, processing, distribution and marketing policies on food influence dietary habits and preferences, as well as people's level of physical activity [5, 6].

Research concludes that obesity occurs with greater prevalence in women, older people (25 years and above), those with a lower school level and those with a low socioeconomic income. Geographic location also has an influence, but it occurs in an opposite way in men and women. There is greater obesity in women living in rural areas, but in men rurality is a protective factor since obesity is more prevalent in those living in urban areas. This is most likely explained by the higher levels of physical activity of men living in rural areas [6]. In terms of lifestyles, obese people have lower levels of physical activity, a bigger consumption of salt and low levels of sleep [7].

Another bibliographical review relates meal times with the prevalence of obesity. This study, which is based on an analysis of the circadian rhythm, a biological function regulated by the anterior hypothalamus, reveals clinical observations

and experiments on animals that relate the times of food intake to metabolism and body weight. It is proposed that eating at times when the body is prepared for rest increases body weight. At rest there are low levels of hormonal activity, a low body temperature and low activity of nutrient absorption which alters energy homeostasis and facilitates the accumulation of energy, increasing body weight [5, 8].

A factor recently studied as a cause of obesity is gut microbiota, defined as the communities of living microorganisms that colonise the intestines. It is known that the gut microbiota contributes significantly to the two main functions of the gastrointestinal tract: nutrition and defence.

3. Human gut microbiota: role in people's health

The human gut is colonised by an enormous quantity of communities of living microorganisms, mainly bacteria that form the GM. As well as participating in processes of digestion and absorption of nutrients, the homeostasis of GM has been linked to beneficial health effects [9].

In the womb, the intestine of the human foetus is sterile, and it is after birth when the newborn acquires its gut microbiota. From birth, several factors influence the composition and initial colonisation of the GM. Natural labour favours the rapid settlement of bacteria that come from the vaginal and faecal microbiota of the mother, associated with a low risk of suffering from diseases and some types of malnutrition through excessive food consumption in the future. On the other hand, children born through caesarean section have a slower colonisation of bacteria, mainly from the hospital environment and healthcare professionals who are in contact with the child. It must be considered that the time taken to establish the GM is a risk factor for the colonisation of *Clostridium*. Despite the fact that GM is established from birth, its colonisation continues to vary, given that there are diverse factors that influence its composition: nutrition, exclusively or predominantly breastfeeding, age, geographic location and intake of supplements and medicines [9, 10].

In the first years of life, when nutrition is acquired through breastfeeding or formula milk, GM is mostly populated by *bifidobacteria*. This is highly adapted to processing oligosaccharides in milk. Later, reaching adulthood, the GM is made up of two main families of bacteria: *Firmicutes* and *Bacteroidetes*. *Firmicutes* are the family found in greater proportion, including more than 200 genders, and the most important are *Mycoplasma*, *Bacillus* and *Clostridium*. Once these are established in adulthood, they remain stable in the individual (**Figure 1**) [11–14].

Various studies support the fact that variations in people's health status are directly related to functions of the GM, highlighting its effect on the immune response, nutrition and metabolism [15].

The immune tissues of the gastrointestinal tract make up the most complex and largest proportion of the body's immune system, which is why alterations of GM are caused by disequilibrium of the immune system, such as the development of allergies and autoimmune diseases [16].

There is a complex interaction between the microbiota, the intestinal epithelium and the gastrointestinal immune system, with many metabolites and microbial components that have a direct influence on the host immunity. The production of metabolites from nutrients or the modification of the metabolites produced by the host has a direct effect on the immune cells and on the integrity and permeability of the intestinal epithelium. The enteric immune system is constantly evaluating and responding to the gut microbiota [17].

Nutritional and metabolic functions of GM involve the synthesis of certain vitamins such as K, B12, biotin, folic acid and pantothenic acid, as well as the

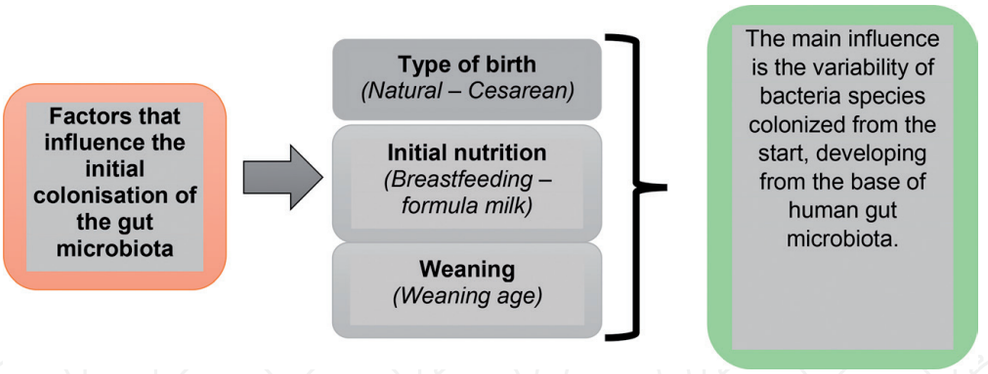

Figure 1.
Factors that influence the initial colonisation of gut microbiota.

fermentation of non-digestible carbohydrates, polysaccharides and oligosaccharides. It makes up an important source of energy for bacterial proliferation and produces short-chain fatty acids that the host can absorb, favouring the recovery and absorption of ions such as calcium, iron and magnesium [18].

As well as the principal functions of GM, recent studies have associated it with the development of obesity and chronic diseases. In order to avoid such conditions, it has been proposed that the role of prebiotics and probiotics in the maintenance of the nutritional status and prevention of diseases should be studied [4].

Undoubtedly, microorganisms present in the guts are fundamental throughout life as they have a direct relationship with health and illness. It is vital to take special care in the factors that make up the initial GM as it is responsible for the general status of future health of individuals.

4. Microbiota and its role in obesity and chronic diseases

As already described, the gut microbiota is made up of a diversity of pathogens which highlights the presence of Gram-negative or Gram-positive bacteria. The equilibrium of this ecosystem provides us great health benefits, in the absorption and digestion of nutrients, as well as influencing immune function [4–18].

In recent years, the prevalence of non-transmittable chronic diseases has increased, which has led different researchers to address the different risk factors related to the appearance of those that have been mentioned and the direct risk between microbiota and diseases [18].

Studies linked to the establishment of microbiota with chronic diseases have described that this stability of original microbiota is affected by diverse factors among which the macro- or micronutrients and dietary patterns are mentioned [18].

When the balance of microbiota is altered by a factor, whether it is the use of antibiotics, stress or diet with an increased consumption of fats, carbohydrates and fibre, it will have an effect on health. It has also been mentioned that abundance or scarcity of food, environmental pollution, chronic stress and food such as dairy products, sugars, coffee, tea and alcohol, among others, affects the intestinal mucous membrane and, consequently, changes the stability and mode of action of the microbiota [19].

All these factors cause an alteration in microbiota where the equilibrium of two large families of bacteria is affected: *Bacteroidetes* and *Firmicutes*. In a study carried out on the composition of the microbiota of obese mice, it was shown that they had increased concentrations of *Firmicutes* by more than 50%, while those of *Bacteroidetes* decreased correlatively [20].

In order to demonstrate that the microbiota changes through time, a study was carried out where old teeth found in skeletons in different periods of history were analysed. It was found that microbial changes are linked to human evolution from the hunter-gatherer period to the industrial revolution, due to the increased consumption of processed foods. These conclusions support the idea that diet and the type of food alter human microbiota [18].

Turnbaugh et al. carried out studies on 'humanised' or sterile mice models to which human faecal matter was transplanted and the microbiota was analysed once they were fed with a Western diet rich in fats and sugars. They found a presence of greater adiposity and a reduced proportion of *Bacteroidetes* than *Firmicutes* in the faecal microbiota of these mice [19].

There is a direct relationship between microbiota and energy uptake and consequently a relationship with the onset of obesity. This has been supported by a study in obese children, which showed a microbiota rich in *Enterobacteriaceae*, low in *Bacteroidetes* and increased in *Firmicutes*. When the children were subjected to diets with low carbohydrate and fat content, the composition of the microbiota changed [18].

Other studies carried out in mice reflect the relationship between energy balance, diet and microbiota. The transplantation of the obese microbiota results in an increase of adiposity in the recipients, which shows that microbiota affects nutrient acquisition, energy storage and consequently the development of obesity [18, 22].

Jumpertz et al. carried out studies on the GM of obese and slim patients. They were given caloric diets, and their stools were analysed by measuring calories ingested and those eliminated in the faeces using a bomb calorimeter. This study concluded that there are changes in the GM where there are increases in energy storage, a decrease in the *Bacteroidetes* and an increase in the *Firmicutes* bacteria. All these changes could explain the variation of the uptake of energy in individuals and therefore a relationship with the predisposition to obtain metabolic disorders [18].

According to the above, the instability of the GM or changes in the original GM affects the metabolism of the whole organism. Recent studies have shown that the increase of bile acids in the intestine when comparing sterile rats with normal rats would show that the GM is not only related with obesity but also with a diverse range of metabolic diseases [21].

When transplanting GM into obese mice, the new term obesogenic microbiota was determined, associated with an increase in hepatic glucose production and promotion of triglyceride deposits. These studies show that there is an increase in TNF pro-inflammatory cytokines which can cause insulin resistance. This can be correlated with the appearance of DM-OB, since its presence has been proven to be directly related to the presence of elevated pro-inflammatory factors [22].

Toll-type receptors are receptors that recognise important patterns within immunity and inflammation processes; they are present in diabetic obese patients with metabolic syndrome. In publications made in the Latin American report, they show the role of GM with regard to the regulation of these diseases, since the mice deficient in Toll-like receptor 5 (TLR5), which recognises microbial patterns, show hyperphagia, become obese and develop characteristics indicative of metabolic syndrome. This is also proven when GM from these mice was transplanted into germ-free mice with the TLR5 gene—the receptor mice developed characteristics similar to the metabolic syndrome. In summary, Larsen et al. demonstrated that GM from diabetic patients produced a significant reduction in *Firmicutes* and *Clostridia* [22, 23].

When administering antibiotics to these types of obese patients, many *Firmicutes* were eliminated, which resulted in improved insulin and glucose intolerance.

The exact mechanisms by which GM contributes to the development of obesity have not been fully elucidated, but it has been suggested that the main ways would

include increased lipoprotein lipase (LPL) activity, increased intestinal permeability and lipogenesis [20]. Faecal microbiota transplantation studies in both healthy individuals and rats to obese receptors have shown the favourable action of the GM of the emitters towards the obese individuals, reducing the levels of glycemia, preventing the expansion of fat and regulating inflammatory processes. Studies also suggest that the reverse mechanism, that is, transplantation of intestinal microbiota from obese to healthy individuals, can transmit the development of obesity, assigning GM as a predisposing factor for obesity [17].

Thanks to many randomised studies that have been carried out, it will be possible to identify the microbiota properties that are contributing to the obesity epidemic, diabetes and metabolic diseases. This will in turn allow scientists to extract information and characterise diseases with the aim to prevent or cure them [21].

5. Role of prebiotics and probiotics in maintaining nutritional status

Probiotics have been defined by the United Nations' Food and Agriculture Organization (FAO) and the WHO as 'living microorganisms that provide their host with health benefits when consumed in appropriate quantities' [4].

Prebiotics were defined by Gibson and colleagues in 2004 as 'ingredients that when selectively fermented, give rise to specific changes in gut microbiota composition and/or activity, which leads to many health benefits with regard to, for example, the wellbeing of individuals' [24].

According to the preceding analysis, it has been recently established that the microbiotics that colonise the human intestine can play an important role in causing obesity and developing other metabolic and immunological illnesses within their host. This statement is based on recent metagenomic studies that have determined that there are differences between obese individuals and those of a normal weight in terms of gut microbiota. These differences are associated with the presence of a microbial population in obese individuals, which has a greater capacity to recuperate dietary energy and/or favour the onset of chronic low-grade inflammation [25].

Some studies conducted on obese individuals in relation to their prebiotic consumption—capable of selectively stimulating a limited number of gut microbiota genres/species which results in health benefits—have demonstrated results that can be categorised by an increasing trend in satiation. This effect has been observed with inulin fibre [24].

The mechanisms implicated in satiation following the consumption of oligofructose and inulin have been associated with intestinal fermentation-induced hydrogen formation, which in turn has been correlated with a postprandial increase in plasma levels of peptides GLP-1 and PYY, which are involved in energy homeostasis through supressing the sensation of hunger (**Figure 2**) [24].

Studies carried out involving the use of probiotics to control body fat accumulation show promising results, but it is also necessary to carry out further, well-designed studies which incorporate randomised, double-blind trials with a placebo control, in order to demonstrate the efficacy of specific probiotic strains when preventing or treating the overweight and obese. In this sense, it is important to highlight that the results obtained in the trials would be specific to the strains analysed, meaning it is not possible to extrapolate the results or action mechanisms to a species or genre as a whole [4, 24].

Consequently, much remains to be done in this field. However, when putting everything in perspective, modulating the composition of gut microbiota using probiotics could be considered as a new avenue through which we can treat the overweight and obese [24].

Figure 2.
Mechanisms implicated in the satiety of obese individuals related to inulin consumption.

On the other hand, prebiotics promote the absorption of minerals such as calcium, magnesium, zinc and iron due to their ability to bind to them. This thereby prevents their absorption into the small intestine to reach the colon, where they are released and subsequently absorbed. Better calcium absorption is linked to prebiotic fermentation caused by gut microbiota, which produces short-chain fatty acids and lowers luminal pH, increasing the bioavailability and passive absorption of calcium through colonocytes. It has also been proven that calcium bioavailability is improved when released through hydrolysis of a compound called 'calcium phytate', which occurs due to action of bacterial phytases present in the beneficial microbiota. It is also improved when calcium becomes more soluble as a result of an increase in the volume of water in the colon, caused by the osmotic effect of prebiotics. This increase in calcium absorption is beneficial for bone health as it increases bone mass and delays the onset of osteoporosis. This is important when considering growth periods, during which peak bone mass is reached in postmenopausal women and in elderly people [25].

Prebiotics stimulate iron absorption into the colon, by increasing the soluble fraction in the cells. Zinc is necessary for the development and maturation of the skeleton, and prebiotics stimulate zinc bioavailability. Therefore, mineral is supplemented through the diet. Furthermore, prebiotics are attributed to another set of properties related to particular systemic disorders. Prebiotic carbohydrates (GOS, FOS, inulin) reduce blood pressure, as well as blood levels of glucose, cholesterol, triglycerides and phospholipids, as well as triglyceride and fatty acid synthesis in the liver, minimising the risk of developing diabetes, obesity and atherosclerosis [25, 26].

6. Influence of probiotics in the development of obesity

The development of obesity is not caused by one specific factor. On the contrary, it is the result of an interaction of genetic, environmental, social, lifestyle factors, etc., which transforms it into a multifactorial disorder, which explains how quickly it has become more prevalent throughout all age groups in developed countries and shows that traditional theories are not adequate to explain the complex phenomenon of gaining body weight [27].

In recent decades and as a result of advances in science, research for 'nontraditional' etiological factors involved in an individual's excessive weight gain has begun [28]. One of these new factors is highlighted in a study on the role of probiotics and their influence on gut microbiota. It should be noted that even now few studies have been carried out on humans to elucidate the effect of these microorganisms on body

weight, and existing studies still show contradictory results; the available literature arises from findings found in animals (rats) [4].

However, several ways in which probiotics may influence adiposity and weight gain have been identified. These range from extracting calories from nutrients, generating specific metabolites, to modulating the behaviour of the brain-intestine axis [27].

These hypotheses arise from research suggesting that obese individuals have a different composition of gut microbiota to subjects of a normal weight. For example, in people with obesity, they have observed a decrease in members of phylum *Bacteroidetes* (*Prevotella* and *Bacteroides* genera) and an increase in members of phylum *Firmicutes* (*Clostridium*, *Enterococcus*, *Lactobacillus*). It has been suggested that the aforementioned microorganisms have a greater capacity to extract energy from the undigestible waste products of nutrients that pass through the large intestine. These alterations are normalised with both phyla, when the subjects lose weight (increase of *Bacteroidetes* and decrease of *Firmicutes*) [29]. It has been observed that obese and inactive people have greater adiposity and insulin resistance but also less diversity and microbial composition (**Figure 3**) [4].

A study conducted at Laval University in Canada evaluated the effect of *Lactobacillus rhamnosus* probiotic (LRP) supplementation on weight reduction, appetite control and eating behaviour in a group of obese men and women. The results showed that, in the group of women, the consumption of this microorganism provides beneficial effects on mood and behaviour linked to dietary intake, compared to the group of men and those who received the placebo. This opens up a series of possibilities for health professionals with regard to the implementation of successful obesity programmes, especially considering that many individuals experience great difficulties when trying to sustain their weight loss. This is mainly due to poor appetite control and not taking the necessary action to lead a healthy lifestyle. Based on the findings of this research, it has been suggested that some probiotic strains could be included as environmental factors with regard to body weight loss and maintenance [27].

Corroborating these findings, a multicentre, randomised, double-blind study conducted on 29 men and 14 women, using the microorganism *Lactobacillus gasseri* and administered through a probiotic yogurt for 12 weeks, demonstrated, in the individuals analysed, a 4.6% decrease in areas of abdominal and subcutaneous fat, as well as a decrease in BMI, waist and hip circumference [4].

Nevertheless, and contrary to these results, several investigations using a similar model maintain that administering yogurt with other species of probiotics such as

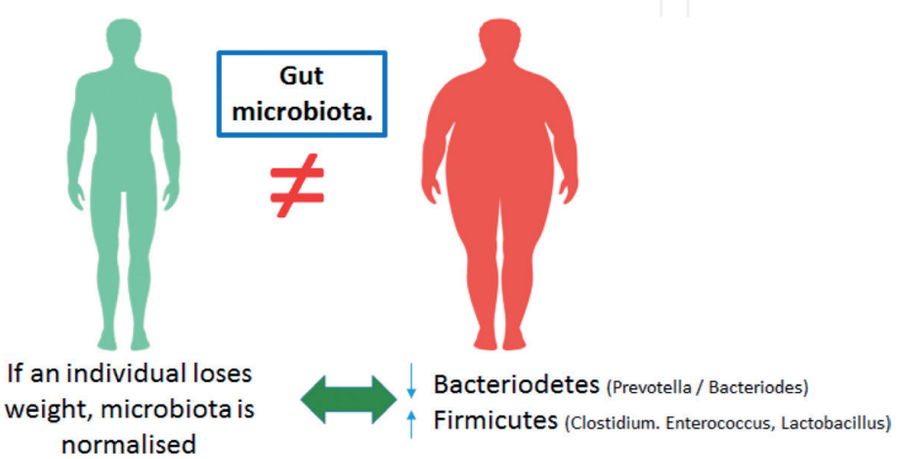

Figure 3.
Gut microbiota imbalance between healthy and obese individuals.

Bacillus lactis Bb 12 and *Lactobacillus acidophilus* La5 has no effect on body weight, BMI and serum lipid levels. This is also the case with *Streptococcus thermophilus* in fermented milk for 8 weeks [4].

As previously mentioned, the impact of consuming probiotics in relation to obesity and a variety of parameters has been widely documented. This is, thanks to studies on rats, used when administering different strains of the *Lactobacillus* and *Bifidobacterium* genera; various hypotheses have emerged from these investigations. More specifically, one of these arose from observing axenic mice (without gut microbiota). Despite consuming 30% more food than conventional animals of the same age and weight, they have 42% less total body fat. However, once the microbiota from conventional mice was transplanted into the digestive tract of the axenic mice, these latter experienced a 57% increase in total body fat. The mechanism associated with this increase is linked to the increased activity of the enzyme lipoprotein lipase, which inhibits the fasting-induced adipocyte factor (FIAF) hormone due to the effect of the gut microbiota. This explains why axenic mice do not show weight gain even when consuming a high-calorie diet. Various studies carried out on rats show that the 'antiobesity' effect is determined by differences between microbial species and strains [20].

There is not much research on the effect of probiotics and their relationship with obesity; however, the findings that are available indicate that the changes caused by gut microbiota could be useful as another strategy in the current quest to combat obesity. However, it is necessary to continue researching in this area, with more emphasis on investigations on humans and populations with a representative number of subjects so that results can be valid and extrapolated.

7. Discussion

Currently, obesity is a major public health problem worldwide. There is no specific cause that determines the development of this disease; on the contrary, it is the result of numerous factors that interact, including genetic, environmental, food, lifestyles, etc. [27]; however, the study of intestinal microbiota and its implication in obesity has aroused curiosity among scientists. The current information obtained comes mainly from the work done in rats and, exceptionally, in humans [17].

Although much of the research indicates that there are differences in the composition of the intestinal microbiota of obese and normal subjects, there is still controversy about it [4].

As indicated in this chapter, the development of these intestinal bacterial communities depends to a large extent on the host and can be modified by exogenous and endogenous influences. Ghosh et al. in 2011 talked about the direct relationship between food patterns and the microbiota composition [18].

In 2013, Devaraj et al. established the existing association in the imbalance of the microbiota where the composition of this is affected producing an increase of the *Firmicutes* genus and a decrease of the *Bacteroidetes*; this instability would affect the metabolism of the human being and as a consequence the appearance of various metabolic diseases among which obesity stands out [21]. In the same way, several publications continue confirming this situation, for example, the study of Jameel Barkat of the year 2018 relates the role of the microbiota in the regulation of energy balance, food absorption and its relationship with the appearance of various diseases: diabetes, metabolic syndrome and obesity, among others [30]. A study was conducted in 36 adults analysing the faecal bacterial composition, finding a decrease in *Firmicutes* and *Clostridia* in diabetic patients compared to the control group. This same research reaffirms that among the factors that alter the stability

and composition of the microbiota are the changes in the diet (high-fat diets) that impact on the relationship between *Firmicutes* and *Bacteroidetes* [31].

In order to demonstrate the aforementioned, it is convenient to point out two investigations carried out in 2015; Kasai et al. [32] conducted a study in Japanese population where they observed that bacterial diversity was significantly higher in obese subjects than in nonobese subjects. In the first group, there was a decrease of the *Bacteroidetes* species and increase of *Firmicutes* [31]. Angelakis et al. showed in a study performed in thin and obese subjects that the *Firmicutes* and *Actinobacteria* genera were the most predominant of the duodenal microbiota; however, in subjects with obesity, a higher proportion of anaerobic and lower proportions of aerobic genus were observed [33].

On the other hand, there are other authors such as Murugesan et al. that indicate in a study carried out in 190 Mexican children between 9 and 11 years old no significant differences between the different bacterial species of microbiota in the different evaluated subjects [34]. The same happens in Hu et al. In 2015, when 134 Korean adolescents from 13 to 16 years old were evaluated, no significant differences were found between the *Bacteroidetes*, *Firmicutes* and *Proteobacteria* populations [35].

It is widely recognised in the scientific community that diets with high-fat content favour the development of resistance to leptin, hyperphagia and therefore obesity. A study points out that the inclusion in the diet of the oligofructose prebiotic prevents the development of resistance to leptin and hyperphagia in rats; from this it is concluded that oligofructose reduces the energy of ingestion, and therefore it is suggested that it has the potential for the obesity treatment [36].

On the other hand, the use of probiotics is associated with multiple health benefits, which are highly endorsed by the scientific community. So far, the data that associate a specific type of microorganisms with human obesity are not conclusive, since they do not say whether it is the microbiota that plays a cause-effect function of obesity or whether it is the intestinal microbiota that is modulated in response to obesogenic diets and other factors related to the pathogenesis of this condition [28–30, 37].

Studies aimed at modulating the gut microbiota to prevent or control the obesity of the host, including the use of probiotics, show positive results [25, 30].

With the reviewed bibliographic evidence, it is crucial to highlight the importance of including the gut microbiota as one of the factors involved in the management of obesity; however, more conclusive information must be analysed about it evaluating what happens even in other specific groups such as the child population.

8. Conclusions

As has been discussed in this chapter, multiple factors influence the development of obesity, including diet, a sedentary lifestyle, selection and an inadequate intake of nutrients such as simple carbohydrates and saturated fats. It has also been observed that obese individuals and those of a normal weight have different compositions of gut microbiota, with some bacterial families more predominate than others, which in turn modulates an individual's energy balance in some way and promotes the development of obesity. It has been established that a dietary supply of prebiotics and probiotics, for which there is scientific evidence demonstrating their benefits, could serve as an additional tool to curb the rising trend of obesity in the population.

However, more studies using randomised, double-blind, controlled trials are necessary to demonstrate the efficacy of prebiotics and probiotics for the prevention or treatment of overweight and obesity.

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Conflict of interest

The work team declares that it has no conflict of interest of any kind.

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References

[1] Fernando MB, Santiago MB. La desconcertante epidemia de obesidad. Revista Chilena de Nutricion. 2015;**42**(1):96-102. Disponible en: https://scielo.conicyt.cl/scielo.php?script=sci_arttext&pid=S0717-75182015000100013&lng=es [citado: October 01 2015]. DOI: 10.4067/S0717-75182015000100013

[2] WHO. Obesidad y sobrepeso [Internet]. 2018. Available from: http://www.who.int/es/news-room/fact-sheets/detail/obesity-and-overweight

[3] Lim JU et al. Comparison of world health organization and Asia-Pacific body mass index classifications in COPD patients. International Journal of Chronic Obstructive Pulmonary Disease. 2017;**12**:2465

[4] Prados-Bo A, Gómez-Martínez S, Nova E, Marcos A. El papel de los probióticos en el manejo de la obesidad. Nutrición Hospitalaria. 2015;**31**:10-18

[5] Escobar C, Ángeles-Castellanos M, Espitia Bautista EN, Buijs RM. Food during the night is a factor leading to obesity. Revista Mexicana de Trastornos Alimentarios. 2016;**7**(1):78-83

[6] Maira PM, Solar AP. Encuesta Nacional de Salud, ENS 2016-2017: Un aporte a la planificación sanitaria y políticas públicas en Chile. Margozzini y Passi. 2018;**43**(1):30-34

[7] Petermann F, Durán E, Labraña AM, Martínez MA, Leiva AM, Garrido-Méndez A, et al. Factores asociados al desarrollo de obesidad en Chile: Resultados de la Encuesta Nacional de Salud 2009-2010. Revista Médica de Chile. 2017;**145**(6):716-722. Available from: http://www.scielo.cl/scielo.php?script=sci_arttext&pid=S0034-98872017000600716&lng=en&nrm=iso&tlng=en

[8] Rodrigo C, Rut F, Patricio P. Circadian rhythms, eating patterns, and sleep: A focus on obesity. Revista Chilena de Nutricion. 2018;**45**(3):285-292. Disponible en: https://scielo.conicyt.cl/scielo.php?script=sci_arttext&pid=S0717-75182018000400285&lng=es [citado October 16 2018]. DOI: 10.4067/s0717-75182018000400285

[9] Icaza-Chávez ME. Microbiota intestinal en la salud y la enfermedad. Revista de Gastroenterología de México. 2013;**78**(4):240-248. DOI: 10.1016/j.rgmx.2013.04.004

[10] Holguín LD, García AM, Lemus K, Ramos A, Sierra GJ, Jiménez M. Microbiota intestinal y sus generalidades en el organismo del ser humano Intestinal microbiota and its generalities in the organism of the human being. Microbiota Biociencias. 2017;**1**(2):23-31

[11] Castañeda GC. Microbiota intestinal y salud infantil. Revista Cubana de Pediatría. 2017;**90**(1). Disponible en: http://www.revpediatria.sld.cu/index.php/ped/article/view/320) [citado October 8 2018]

[12] Koenig JE, Spor A, Scalfone N, et al. Succession of microbial consortia in the developing infant gut microbiome. Proceedings of the National Academy of Sciences of the United States of America. 2013;**108**(Suppl 1):4578-4585

[13] Rajilic-Stojanovic M, Heilig HG, Molenaar D, et al. Development and application of the human intestinal tract chip, a phylogenetic microarray: Analysis of universally conserved phylotypes in the abundant microbiota of young and elderly adults. Environmental Microbiology. 2009;**11**:1736-1751

[14] Suarez Diéguez T, Galván M, López-Rodríguez G, Olivo D, Olvera

Nájera M. El efecto de la dieta sobre la modulación de la microbiota en el desarrollo de la obesidad. Revista de Salud Pública y Nutrición. 2018;**17**(1):31-39

[15] Chan YK, Estaki M, Gibson DL. Clinical consequences of diet-induced dysbiosis. Annals of Nutrition & Metabolism. 2013;**63**(Suppl. 2):28-40

[16] Ley RE, Peterson DA, Gordon JI. Ecological and evolutionary forces shaping microbial diversity in the human intestine. Cell. 2006;**124**:837-848

[17] Castaner O, Goday A, Park YM, Lee SH, Magkos F, Shiow SATE, et al. The gut microbiome profile in obesity: A systematic review. International Journal of Endocrinology. 2018;(1):1-9

[18] Ghosh S, Dai C, Brown K, Rajendiran E, Makarenko S, Baker J, et al. Colonic microbiota alters host susceptibility to infectious colitis by modulating inflammation, redox status, and ion transporter gene expression. American Journal of Physiology. Gastrointestinal and Liver Physiology. 2011;**301**(1):G39-G49. Available from: http://www.physiology.org/doi/10.1152/ajpgi.00509.2010

[19] Turnbaugh PJ, Hamady M, Yatsunenko T, Cantarel BL, Duncan A, Ley RE, et al. A core gut microbiome in obese and lean twins. Nature. 2009;**457**:480-484

[20] Gotteland M. El papel de la microbiota intestinal en el desarrollo de la obesidad y de la diabetes de tipo-2. Revista Chilena Endocrinol Diabetes. 2013;**6**(4):155-162

[21] Devaraj S, Hemarajata P, Versalovic J, Dra Lau L. La microbiota intestinal humana y el metabolismo corporal: Implicaciones con la obesidad y la diabete. Acta Bioquím Clín Latinoam. 2013;**47**(2):421-434

[22] Ariza-Andraca R, García-Ronquillo M. Cirugía y cirujanos El microbioma humano. Su papel en la salud y en algunas enfermedades. 2016;**84**:31-35. Available from: www.amc.org.mx

[23] Musso G, Gambino R, Cassader M. Interactions between gut microbiota and host metabolism predisposing to obesity and diabetes. Annual Review of Medicine. 2011;**62**:361-380

[24] Corzo N, Alonso JL, Azpiroz F, Calvo MA, Cirici M, Leis R, et al. Prebióticos; Concepto, propiedades y efectos beneficiosos. Nutrición Hospitalaria. 2015;**31**:99-118

[25] Rodríguez JM, Sobrino OJ, Marcos A, Collado MC, Pérez-Martínez G, Martínez-Cuesta MC, et al. ¿Existe Una Relación Entre La Microbiota Intestinal, El Consumo De Probióticos Y La Modulación Del Peso Corporal? Nutrición Hospitalaria. 2013;**28**(Suppl. 1):3-12

[26] Pamela M, Jerusa B, Martín G. La microbiota intestinal: Un nuevo actor en el desarrollo de la obesidad. Revista Médica de Chile. 2010;**138**(8):1020-1027. Disponible en: https://scielo.conicyt.cl/scielo.php?script=sci_arttext&pid=S0034-98872010000800013&lng=es [citado: October 08 2018]

[27] Gérard P. Gut microbiota and obesity. Cellular and Molecular Life Sciences. 2016;**73**(1):147-162

[28] Sanchez M, Darimont C, Panahi S, Drapeau V, Marette A, Taylor VH, et al. Effects of a diet-based weight-reducing program with probiotic supplementation on satiety efficiency, eating behaviour traits, and psychosocial behaviours in obese individuals. Nutrients. 2017;**9**(3):1-17

[29] Martínez-Cuesta MC, Peláez C, Requena T. Probioticos En la Salud humana. Grupo de Biología Funcional de Bacterias Lácticas. 2012

[30] Lone JB, Koh WY, Parray HA, Paek WK, Lim J, Rather IA, et al. Gut microbiome: Microflora association with obesity and obesity-related comorbidities. Microbial Pathogenesis. 2018;**124**:266-271

[31] Ley RE, Turnbaugh PJ, Klein S, Gordon JI. Microbial ecology: Human gut microbes associated with obesity. Nature. 2006;**444**:1022-1023. DOI: 10.1038/4441022a.nuevo

[32] Kasai C, Sugimoto K, Moritani I, et al. Comparison of the gut microbiota composition between obese and non-obese individuals in a Japanese population, as analyzed by terminal restriction fragment length polymorphism and next-generation sequencing. BMC Gastroenterology. 2015;**15**(1):100. DOI: 10.1186/s12876-015-0330-2

[33] Angelakis E, Armougom F, Carrière F, et al. A metagenomic investigation of the duodenal microbiota reveals links with obesity. PLoS ONE. 2015;**10**(9):e0137784. DOI: 10.1371/journal.pone.0137784

[34] Murugesan S, Ulloa-Martínez M, Martínez-Rojano H, et al. Study of the diversity and short-chain fatty acids production by the bacterial community in overweight and obese Mexican children. European Journal of Clinical Microbiology & Infectious Diseases. 2015;**34**(7):1337-1346. DOI: 10.1007/s10096-015-2355-4

[35] Hu HJ, Park SG, Jang HB, Choi MG, Park KH, Kang JH, et al. Obesity alters the microbial community profile in Korean adolescents. PLoS ONE. 2015;**10**(7):e0134333

[36] Abreu-Abreu AT. Prebióticos, probióticos y simbióticos. Revista de Gastroenterología de México. 2012;**77**:26-28

[37] Baothman OA, Zamzami MA, Taher I, Abubaker J, Abu-Farha M. The role of gut microbiota in the development of obesity and diabetes. Lipids in Health and Disease. 2016;**15**(1):108