Research article

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Bacteria cause diabetes mellitus: an integrative review

Abstract

Diabetes Mellitus is a metabolic syndrome of multiple origins, resulting from a lack of insulin or the body's inability to use it effectively, characterized by persistently high blood sugar levels (hyperglycemia). Insulin is produced in the pancreas and maintains metabolism by breaking down glucose to provide energy for the body's functioning. Scientific research has shown that people with diabetes have an imbalance between the beneficial and pathogenic bacteria that make up the gut microbiota, which may be associated with the development of this metabolic disorder. The objective of this manuscript is to report on the bacteria that cause type 1 and type 2 diabetes mellitus. This is an integrative literature review study that aims to survey all references found on a given topic. This integrative literature review aimed to gather all available references on a given topic. The data collected from scientific review publications compiled from the Scientific Electronic Library Online, Latin American and Caribbean Literature in Health Sciences, the Ministry of Health's Virtual Health Library, and Google Scholar platforms. After selecting the articles, the following steps were followed: A. Reading all the abstracts of the articles found. B. Reading the selected articles in full after reading the abstracts. B. Interpretive reading and writing of the text. Only original qualitative review articles, available in full text, in Portuguese, English, and Spanish that addressed the proposed topic and period were used. The selection was based on the scientific accuracy of the review articles.

Keywords: antibiotic resistant, pathogens, infections, bacterial resistance

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Introduction

More than 39 million people worldwide could die directly from antibiotic-resistant infections in the next 25 years. Antibiotic resistance, recognized as a major public health challenge, is expected to get worse in the coming years. This occurs when bacteria or other pathogens undergo changes that prevent them from responding to antimicrobial treatments. From 1990 to 2021, more than one million people worldwide died each year due to antibiotic resistance, according to the authors. The researchers analyzed 22 pathogens, 84 combinations of pathogens and treatments, and 11 infectious syndromes in people of all ages in 204 countries and territories, based on data from 520 million people. Deaths in children under five directly caused by antibiotic resistance have decreased by more than 50% in three decades, as infection prevention and control have improved in infants and young children.[1–3]

However, deaths among adults aged 70 and older increased by more than 80% during this period, due to the rapid aging of the population and the greater vulnerability of the elderly to infections. In the coming decades, deaths caused by antibiotic resistance will rise even further. The number of direct victims could reach 1.9 million annually by 2050, a 67% increase compared to 2021, according to researchers' projections. According to scientists, antibiotic resistance could directly cause more than 39 million deaths worldwide and be associated with 169 million deaths between 2025 and 2050.[1–3]

The increase in bacterial resistance leads to the ineffectiveness of treatments. That were previously considered safe and effective, resulting in increased morbidity and mortality, especially in hospital-acquired and community-acquired infections. Furthermore, treatment costs increased considerably, with the need for more expensive medications and longer hospital stays. The impact is even greater in countries with fragile health systems, where resistance can become endemic, making common infections more difficult to treat (current statistics or hospital-based).[1–3]

The Endocrine System consists of various glands and tissues that secrete chemical substances responsible for regulating most biological functions. These substances, called hormones, bind to specific receptors. The endocrine glands and their hormones are transported exclusively through the circulatory system. Hormones play a crucial role in the growth, function, and regulation of various organs, including those responsible for male and female physical characteristics.[4–6]

The pancreas contains tubule-alveolar glands, with secretory cells arranged into short tubes or small sacs called acini. Each acinus is made up of pyramidal epithelial cells. In the acinus, some cells that secrete aggressively accumulate large vesicles of zymogen at the apex. The acinus cells produce pancreatic fluid, which contains digestive enzymes.[4]

The pancreas not only produces digestive enzymes but also acts as an endocrine gland. Among the acinar cells are clusters of endocrine cells known as pancreatic islets. These include at least three types: alpha or A cells, which produce the hormone glucagon; beta or B cells, which produce insulin; and delta or D cells, which produce a substance called somatostatin (Figure 1).[5,6]

Diabetes can lead to elevated blood sugar levels, and high levels can cause complications in the heart, arteries, eyes, kidneys, and nerves, potentially leading to death.[7,8]

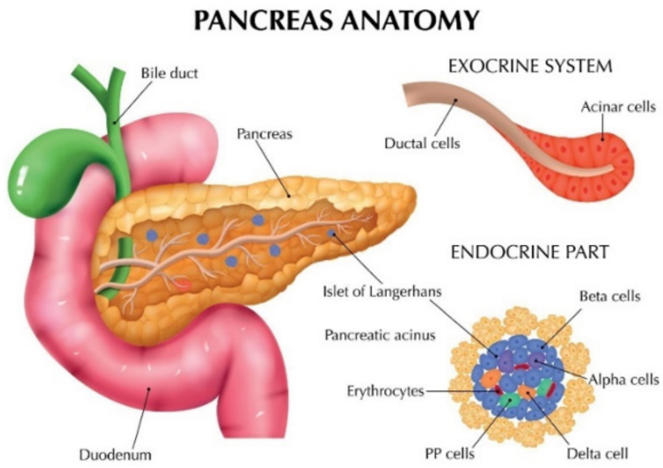

Figure 1 Pancreas Anatomy. This parenchymal organ is divided into five anatomical parts: head, uncinate process, neck, body, and tail. The pancreas's endocrine function is carried out by the pancreatic islets of Langerhans. These endocrine glands secrete hormones directly into the bloodstream and consist of three main cell types: alpha, beta, and delta.

Sources: VectorStock® and https://www.vectorstock.com/royalty-free-vector/pancreas-anatomy-infographic-vector-42854810

Types of diabetes

1. Diabetes mellitus type 1 is a chronic, non-transmissible, hereditary disease characterized by the destruction of pancreatic beta cells responsible for the production and secretion of insulin (Ministry of Health, 2025).[9]
2. Type 2 diabetes occurs when the body does not adequately use the insulin produced. Because type 2 diabetes is directly related to overweight, sedentary lifestyle, high triglycerides, high blood pressure, and poor eating habits, death (Ministry of Health, 2025).[8]
3. Type 3 Diabetes: This is a type of diabetes that is believed to be related to the brain's inability to respond to insulin. It mainly occurs in people with Alzheimer's.
4. Gestational diabetes is a type of diabetes that is diagnosed for the first time during pregnancy, caused by dysfunction in the production and lack of insulin in the body. It may be a genetic predisposition or a diet with excess fat and sugar.
5. Maturity Onset Diabetes of the Young (MODY) diabetes is a rare type of diabetes that occurs in young people, more common than type 1 diabetes and more similar to type 2 diabetes.
6. Latent autoimmune diabetes in adults is an autoimmune type of diabetes, but it occurs in adults.
7. Diabetes insipidus is a rare condition caused by the production of the antidiuretic hormone, which controls the speed at which urine is produced, or by alterations in the kidneys that fail to respond to this hormone.
8. Secondary diabetes is a type of diabetes caused by other health problems, which may result in type 1 or type 2 diabetes.
9. Type 5 diabetes is a type of diabetes caused by malnutrition, characterized by severe insulin deficiency, lack of insulin resistance, and poor metabolic control.[4-7]

Objective

The objective of this manuscript is to report on the bacteria that cause type 1 and type 2 diabetes mellitus.

Methods

This is an integrative literature review study that aims to gather all references found on a given topic. The data were collected from scientific review publications compiled on the Scientific Electronic Library Online, Latin American and Caribbean Literature in Health Sciences, the Ministry of Health's Virtual Health Library, and Google Scholar platforms. After selecting the articles, the following steps were followed: A. Reading all the abstracts of the articles found. B. Reading the selected articles in full after reading the abstracts. B. Interpretive reading and writing of the text. Only original qualitative review articles, available in full text, in Portuguese, English, and Spanish that addressed the proposed topic and period were used. The selection was based on the scientific accuracy of the review articles.

Selections study

Diabetes mellitus

Carbohydrates such as starch are long chains of different molecules composed of simple sugars. Sucrose, lactose, carbohydrates, and other complex sugars must be broken down and converted into simple sugars by enzymes in the digestive tract before the body can absorb them. After the body absorbs simple sugars, it converts them into glucose, a source of energy for the body.[9-11]

Glucose is the sugar that is transported through the bloodstream and absorbed by cells. The body can also manufacture glucose from fats and proteins. Carbohydrates in the blood mean blood glucose. If the body does not produce enough insulin to transport glucose into cells, or if the cells stop responding normally to insulin a condition insulin resistance, the resulting high blood glucose levels, combined with an inadequate amount of glucose in the cells, cause the symptoms and complications of diabetes.[9,11]

Insulin, a hormone secreted by the pancreas organ at the back of the stomach that also produces digestive enzymes, regulates the amount of glucose in the blood. Glucose in the bloodstream stimulates the pancreas to produce insulin. Insulin helps transport blood glucose into cells, converting it to glycogen and into energy (ATP) that is used or stored as fat or glycogen.[12-14]

Diabetes is a disease caused by insufficient production or increased absorption of insulin, a hormone that regulates blood glucose and provides energy to the body. Insulin is a hormone that breaks down glucose molecules, converting them into energy to sustain our cells. Insulin, a hormone secreted by the pancreas, an organ at the back of the stomach that also produces digestive enzymes, controls the amount of glucose in the blood (Figure 2).[4]

Diabetes mellitus is a condition in which the body does not produce enough insulin or does not respond normally to insulin, causing blood glucose levels to become unusually high. Some types of sugar are simple, and others are complex. Sucrose consists of two simple sugars, glucose and fructose. Lactose is made up of glucose and galactose.[11,15-18]

Type 1 diabetes mellitus

The main cause of type 1 diabetes is the immune system's destruction of pancreatic beta cells, which are responsible for producing insulin. Therefore, people with type 1 diabetes do not

Citation: Santana MVO, Marchiori CH, Malheiros KP, et al. Bacteria cause diabetes mellitus: an integrative review. *MOJ Biol Med.* 2025;10(2):101–112. DOI: 10.15406/mojbm.2025.10.00249

produce adequate amounts of their own insulin, causing glucose to build up in the bloodstream. The onset of the disease may also be linked to genetic inheritance. Therefore, it is not associated with the patient's lifestyle or dietary habits, although these are risk factors for complications. [11,15-18]

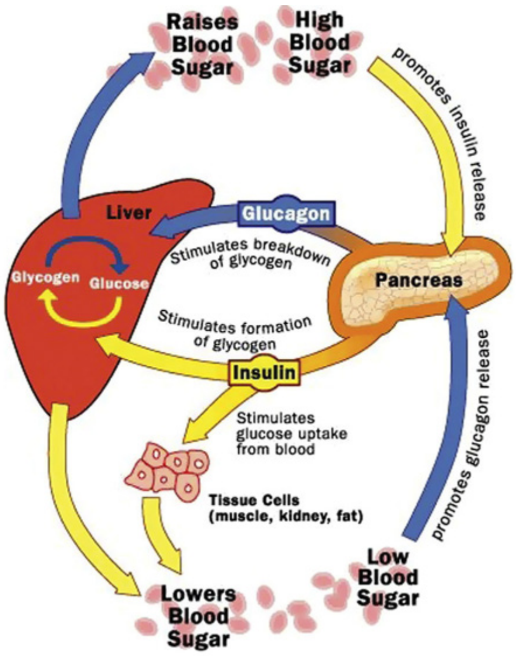

Figure 2 Mechanism of action. Insulin promotes glycogen synthesis, lipid synthesis, and protein. Insulin acts by directly binding to its receptors on the plasma membranes of the. Insulin's significant actions focus on storing excess energy in a fed state. Human insulin is a 51-residue peptide hormone produced by recombinant DNA technology by inserting the human insulin gene into.

Source: https://vamosarema.com/

The main symptoms of type 1 diabetes are:

1) Urge to urinate several times a day.
2) Frequent hunger.
3) Constant thirst.
4) Weight loss.
5) Weakness.
6) Fatigue.
7) Nervousness.
8) Mood swings.
9) Nausea and vomiting.[19]
10) It's believed that an environmental factor, possibly a viral infection or nutritional factor in childhood or adolescence, causes the immune system to destroy the pancreatic cells that produce insulin. A genetic predisposition makes some people more susceptible to an environmental factor. [11,15-18]

Self-care for type 1 diabetes is necessary to avoid complications. Check out:

a. Moderate alcohol consumption can cause hypoglycemia.
b. Avoid saunas and foot baths, which affect microcirculation, high temperatures, heat stroke, angiopathies, and other heart problems.
c. Increase eye care to prevent corneal cells, opportunistic infections, and diseases such as cataracts and glaucoma.
d. Manage stress.
e. People with diabetes are more likely to experience anxiety and depression, controlling hypoglycemia, taking insulin, and gaining weight.
f. Cut out smoking to reduce the risk of heart attack, fatty buildup in the arteries, which blocks circulation, and hypertension in diabetic patients.
g. Take care of your oral health: the blood of diabetics is more prone to bacterial development.
h. Type 1 diabetes has no cure. It is not possible to prevent type 1 diabetes, and there is no test.[19]

Hypoglycemia is a very low blood glucose level and is common in people with diabetes. To avoid hypoglycemia, as well as the complications of diabetes, the key is to maintain glucose levels within the target range established by the healthcare professional for each patient. This target range varies depending on age, general health conditions, and other risk factors, as well as situations such as pregnancy. During treatment, it is essential to maintain healthy habits and an active lifestyle, in addition to following the medication guidelines recommended by the healthcare professional to maintain the glucose target range, avoiding hypoglycemia and hyperglycemia.[8]

Insulin therapy for people with type 1 diabetes should mimic the physiological insulin secretion that occurs in individuals without diabetes. The preferred strategy is basal-bolus therapy, which should be instituted early, with Multiple Daily Insulin Injections (MDI) or with an insulin pump, continuous insulin infusion system. Intensive insulin therapy, whether MDI, is the fundamental therapy from the early stages of the disease, at all ages.[20,21]

Hyperglycemia is a metabolic disorder characterized by elevated blood glucose levels. Although often associated with diabetes mellitus, hyperglycemia can occur due to a variety of medical conditions, including pancreatic disease, hormonal problems, and medication side effects. Proper control of hyperglycemia is crucial, as elevated glucose levels can lead to serious complications, such as cardiovascular disease, neuropathy, and kidney problems. Hyperglycemia is a condition characterized by elevated blood glucose levels, typically above 130 mg/dL fasting or 180 mg/dL two hours after a meal.[22,23]

The symptoms of hyperglycemia, considered a physiological problem, are known to be caused by insulin resistance or variable insulin deficiency. This hyperglycemia causes classic symptoms such as polydipsia, polyuria, polyphagia, weight loss, and blurred vision. Prolonged hyperglycemia can lead to chronic complications, as well as a hyperosmolar hyperglycemic state or diabetic ketoacidosis.[22,23]

Causes

I. Type 1 diabetes: The body does not produce enough insulin.
II. Type 2 diabetes: The body does not use insulin effectively.
III. Pancreatic diseases: Conditions that affect insulin production.
IV. Hormonal problems: Disorders that affect glucose regulation.[22,23]

Type 2 diabetes mellitus

The pancreas typically continues to produce insulin, sometimes even in higher amounts than normal, especially in the early stages of

Citation: Santana MVO, Marchiori CH, Malheiros KP, et al. Bacteria cause diabetes mellitus: an integrative review. *MOJ Biol Med.* 2025;10(2):101–112. DOI: 10.15406/mojbm.2025.10.00249

the disease. However, the body develops resistance to the effects of insulin; therefore, the existing insulin is insufficient to meet the body's needs. As type 2 diabetes progresses, the pancreas's ability to produce insulin decreases.[11,24-26]

These are metabolic changes marked by high and persistent blood glucose levels. It is a long-term, worsening condition that can lead to serious complications, with high rates of illness and death, and a major burden on the healthcare system and society. Type 2 diabetes results from the gradual loss of insulin production, often combined with insulin resistance. It is usually without symptoms, with a higher rate in people over 40 who are overweight, lead a sedentary lifestyle, eat unhealthily, and have a family history of diabetes.[11,27-29]

It is usually asymptomatic, and symptoms generally occur in adulthood after the age of 40, with slow symptom progression and the possibility of late complications, renal, ophthalmological, and neuropathic. It occurs primarily in people who are overweight, have a sedentary lifestyle, unhealthy eating habits, and a family history of diabetes. The diagnosis is often made during routine examinations in asymptomatic individuals, or an acute event, such as infections, cardiovascular events, myocardial infarction, or stroke, or surgery. Symptoms resulting from involvement of the peripheral nervous system, such as paresthesias of the extremities, foot ulcers, blurred vision, fatigue, erectile dysfunction, and recurrent genital candidiasis, may also suggest the diagnosis.[8,11,27]

Obesity is the main risk factor for developing type 2 diabetes, and 80% to 90% of people with type 2 diabetes are overweight or obese. Because obesity causes insulin resistance, people with obesity may require a large amount of insulin to maintain normal blood glucose levels. An elevated corticosteroid concentration is often due to the use of corticosteroid medications, such as prednisone, or Cushing's syndrome and gestational diabetes.[8,11,27]

Obesity is a complex disease with multifaceted origins; its development is influenced by several factors, including individual, collective, social, economic, cultural, and environmental causes. This means that obesity is not only related to personal attitudes and behaviors. Obesity has significant impacts on the healthcare system, and these consequences are not limited to economic costs. This list also includes social costs, such as decreased quality of life, lost productivity, premature mortality, and problems related to social interaction (Figure 3).[8,11,17,30,31]

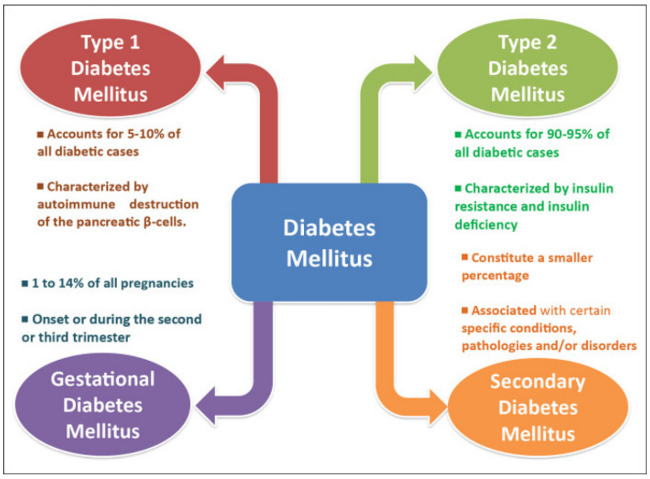

Figure 3 Classification of Diabetes Mellitus (Four types of diabetes mellitus): Type I diabetes mellitus; Type 2 diabetes mellitus; Gestational diabetes mellitus; Specific types of DM due to other causes. Diabetes mellitus, triggered by various reasons, such as monogenic diabetes syndromes, diseases that affect the pancreas or situations where diabetes is induced by drugs or chemicals.

Sources: Doi: 10.4103/ajm.ajm_53_20. PMID: 33437689 and PMCID: PMC7791288

A new treatment may eliminate the need for insulin in patients with type 1 diabetes. A study of an experimental therapy shows promising results and could reach the market in 2026.

Type 1 diabetes is a chronic disease in which the. As a result, it produces little or no insulin, the hormone that regulates glucose into cells. The lack of insulin causes blood sugar levels to rise. It usually appears during childhood and adolescence. Symptoms of type 1 diabetes include excessive thirst, frequent urination, hunger, fatigue, and blurred vision. Without treatment, weight loss and other complications can occur in childhood. A new experimental stem cell treatment has eliminated the need for pharmaceutical insulin in patients with difficult-to-control type 1 diabetes.[32]

The results, considered promising, include Zimislecel, a therapy that uses pluripotent stem cells transformed in the laboratory into pancreatic islet structures specialized in insulin production, then implanted into the patient's body [Researchers from Toronto General Hospital in Canada and the University of Pennsylvania in the United States]. Zimislecel cells are injected into a blood vessel supplying the liver. There, they settle and start producing insulin naturally, without the need for external injections. To prevent rejection, participants take immunosuppressants, similar to those used in organ transplants (Figure 4).[32]

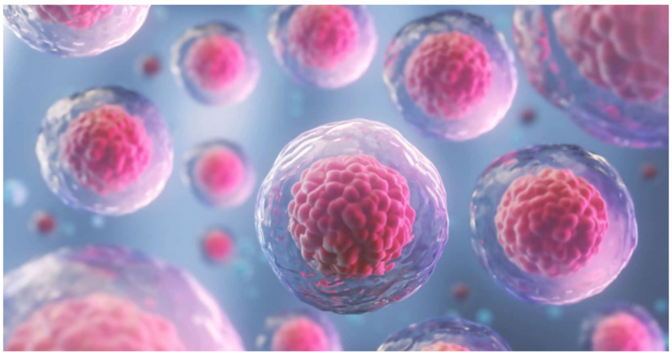

Figure 4 Zimislecel is a stem cell–derived islet therapy lab-grown insulin-producing cells that are infused into the body to help restore natural insulin production. The idea is to bring back the body's ability to regulate blood sugar, without relying entirely on external insulin.

Sources: Experiential Diabetes Education Group Connected in Motion is a registered Canadian Charity and https://www.connectedinmotion.ca/blog/vertexs-stem-cell-therapy-for-type-1-diabetes-at-ada-2025/

In addition to stopping insulin use, participants experienced

1. Zero episodes of severe hypoglycemia, a condition that can cause mental confusion, seizures, and even hospitalization.[32]
2. Improved glycemic control, with glycated hemoglobin levels below 7%, considered ideal for people with diabetes.
3. Longer duration of normal blood glucose levels and reduced glycemic variability, which reduces the risk of complications.
4. According to the researchers, the observed side effects were mild to moderate.[32]

Digital tools in diabetes. The role of telemedicine

A digital platform, data generates Big Data that, when processed using Cognitive Computing, produces algorithms capable of improving diabetes interventions based on the best scientific evidence.

1. **In Continuous Glucose Monitoring (CGM)3**, the sensor subcutaneously captures data and sends it to a smartphone app, which in turn forwards it to the cloud, where it can be integrated with other automatically generated or manually entered data.
2. **Telemedicine Communication Systems or Platforms** include phone calls, interactive voice response (IVR), short message services (SMS), two-way video, email, websites, and mobile technologies such as smartphones, tablets, and others.
3. **Retina Risk is a platform** for calculating and stratifying the risk of developing diabetic retinopathy, based on algorithms generated by studying retrospective retinopathy risk data. Digital therapies are diabetes care management platforms, either autonomous or mediated by healthcare professionals, based on websites, apps, and smartphones, resulting in better control and a significant reduction in HbA1c levels that is maintained over the medium term.
4. **Telemedicine can improve HbA1c** outcomes when it includes real-time engagement with the care team and the availability of live remote medical consultations for possible medication adjustments.[32,33]

Gut microbiota

The gut microbiota engages in several metabolic functions, including fermenting and absorbing undigested carbohydrates, absorbing electrolytes and minerals, regulating intestinal motility, and producing certain micronutrients. Because of its role in developing these functions, changes in the microbiota have been proposed as a possible cause of obesity. The microbiota mainly consists of bacteria, with smaller amounts of viruses, fungi, and eukaryotic cells. Several factors can influence the composition and diversity of the microbiota, including diet, age, genetics, antibiotic use, and lifestyle.[34-38]

Viruses such as enterovirus, cytomegalovirus, and hepatitis C virus have been linked to an increased risk of developing type 2 diabetes. These viruses can trigger an autoimmune response in the body, leading to the destruction of insulin-producing cells in the pancreas. Bacteria, such as *Helicobacter pylori* (Goodwin *et al.,* 1989) (Campylobacterales: Helicobacteraceae) and *Streptococcus* Rosenbach, 1884 (Lactobacillales: Streptococcaceae), have also been linked to type 2 diabetes, causing chronic inflammation and tissue damage. The species *H. pylori* is a pathogenic bacterium present in the stomach mucosa, which may cause gastritis, gastroduodenal ulcers, and gastric cancer.[39]

More recent studies analyzing the microbiome identify fungi present in human feces samples, mainly the species *Candida albicans* (Berkhout, 1923) (Saccharomycetales: Cryptococcaceae), *Cladosporium* Link (1816) (Capnodiales: Davidiellaceae), for the most abundant genus *Saccharomyces boulardii* Henri Boulard, Seguela, Bastide & Massot, 1923, and *Saccharomyces cerevisiae* Meyen ex Hansen, 1883 (Saccharomycetales: Saccharomycetaceae) (Figure 5).[40,41]

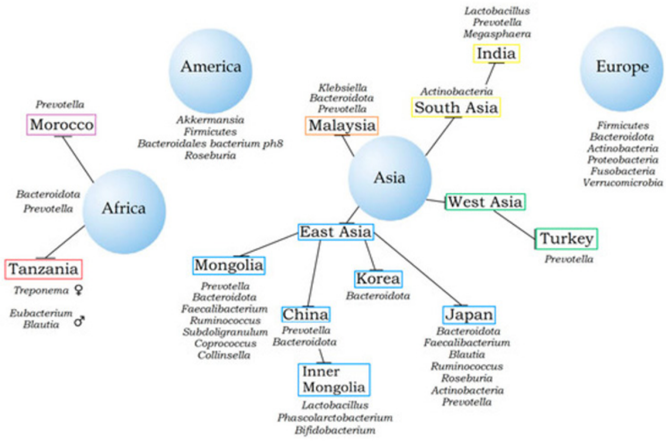

Figure 5 Features of the gut microbiota of different regional populations. In various parts of the world, the human gut microbiota is a complex community of microorganisms living in the digestive tract, composed of more than 1,500 species from different phyla, with 99% of the bacteria in the microbiota coming from approximately 30 to 40 species. In our body, the colon alone contains the largest population of the diverse human microbiota and can harbor up to 100 trillion bacteria.

The most common phyla in both humans and mice are the Firmicutes, which make up 60–80% and include over 200 "genera". *Clostridium* Prazmowski, 1880 (Eubacteriales: Clostridiaceae), *Lactobacillus* Beijerinck 1901 (1980) (Lactobacillales: Lactobacillaceae), *Ruminococcus* Sijpesteijn, 1948 (Oscillospirales: Ruminococcaceae). Bacteroidetes, accounting for 20-30% notably *Bacteroides* Castellani & Chalmers 1919 (Bacteroidales: Bacteroidaceae)*, Prevotella* Shah and Collins, 1990 (Bacteroidales: Prevotellaceae), and *Xylanibacter ueki et al.,* 2006 (Bacteroidales: Prevotellaceae*),* representing about 10% with *Bifidobacterium* being the main genus, are also present. Additionally*, Escherichia* Castellani & Chalmers, 1919 (Bacteroidales: Prevotellaceae), as members of Enterobacteriaceae, are found to a lesser extent.[42-45]

In metabolic functions, the microbiota

1. Barrier against pathogens.
2. Regulation of the immune system.
3. Nutrient metabolism.
4. Production of bioactive compounds.
5. Production of vitamins.
6. Promotes tissue development.
7. Stimulates the production of natural antibodies.
8. In the biotransformation of bile acid conjugates.
9. Digestion of plant polysaccharides (Figure 6).[42-43]

Vaginally delivered newborns are exposed to bacteria more quickly than babies born by cesarean section. This is because vaginal delivery provides direct contact with the mother's fecal flora. In the cesarean section, the source of pollution is the environment, thus delaying the establishment of the microbiota. However, microbiota production occurs soon after birth, which affects the physiology, development, and morphogenesis of the host and the maintenance of tissue and organ balance.[44,45]

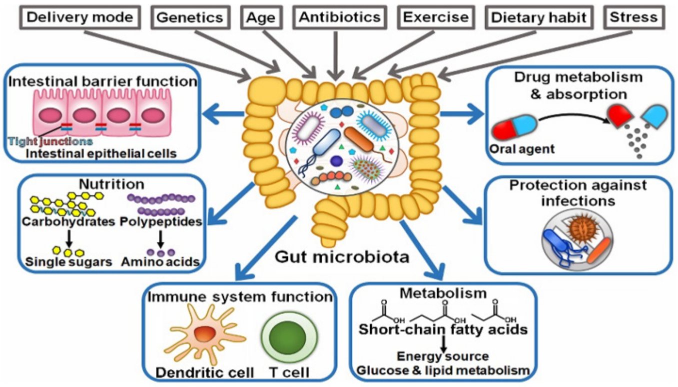

Figure 6 The gut microbiome refers to all the microorganisms living in the digestive system. Your gut flora, also known as gut microbiota or gut microbiome, consists of trillions of microorganisms, predominantly bacteria. Em nosso corpo, o cólon, por si só, contém a maior população da diversa microbiota humana. In our body, the colon alone contains the largest population of diverse human microbiota.

Sources: From www.frontiersin.org and https://guideofgreece.com/

Dysbiosis states are generally characterized by the loss or insufficient representation of beneficial species that are usually dominant, and an increased abundance of minority species that often include pathobionts or opportunistic pathogens. Changes can be specific to each niche and each disease and can lead to a global alteration in the structure of the microbiota, or the loss or acquisition of specific species (Figure 7).[42-46]

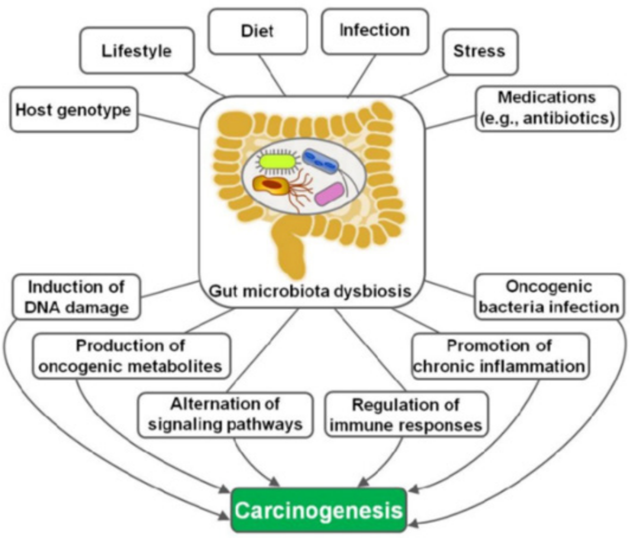

Figure 7 Host genotype, lifestyle, diet, infection, psychological stress, and medications are important factors leading to intestinal dysbiosis. Gut microbiota dysbiosis drives carcinogenesis via different mechanisms, including induction of DNA damage, production of tumor-promoting metabolites, regulation of diverse signaling pathways, manipulation of host immune responses, activation of chronic inflammation, and promotion of oncogenic bacterial infection.

Source: Doi: 10.7150/ijbs.68170

Healthy microbiota

1. A diet rich in fiber.
2. Consumption of fermented foods.
3. Use of probiotics and prebiotics.
4. Limit antibiotic use.
5. A varied and balanced diet.
6. Breastfeeding during infancy.
7. Physical activity.
8. Stress management.
9. Limit alcohol and tobacco consumption.[34-37]

Studies/Articles

The influence of intestinal microbiota on diabetes

Studies confirm that people with diabetes have a composition of intestinal microbiota healthy individuals.

Some differences include

1. Decreased number of beneficial bacteria, such as *Bifidobacterium* Orla-Jensen 1924 (Bifidobacteriales: Bifidobacteriaceae), and *Lactobacillus*.
2. Increase in the number of pathogenic bacteria, such as *Escherichia coli* (Escherich, 1885) (Enterobacteriales: Enterobacteriaceae and *Staphylococcus* Rosenbach, 1884 (Bacillales: Staphylococcaceae).
3. Alteration in the production of metabolites, such as fatty acids, from brown sugar.
4. Mechanisms that influence intestinal microbiota or diabetes (Figure 8).[9,47,48]

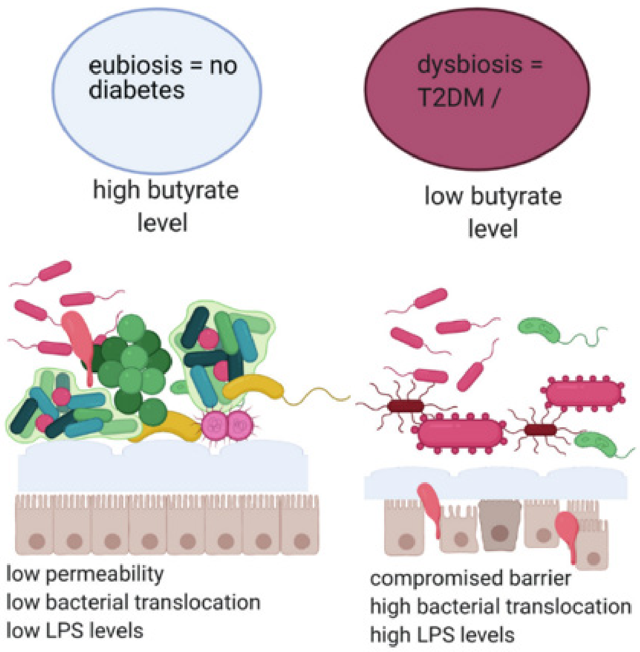

Figure 8 Microbiota and the intestinal barrier in healthy individuals and those with type 2 diabetes. A strong and growing body of evidence supports the role of gut microbiota in human metabolism. Altered glucose homeostasis is linked to changes in gut microbiota and the development of type 2 diabetes mellitus (T2DM) and its complications.

Source: Stachowska *et al.,* 2021

The intestinal microbiota can influence diabetes through several mechanisms, including

1. Regulation of blood glucose and insulin.
2. Modulation of the immune system and inflammation. Alteration of function and permeability of the intestinal barrier.
3. Implications for the treatment and prevention of diabetes.
4. Understanding the relationship between the intestinal microbiota and diabetes may have important implications for the treatment and prevention of the disease.[9,48]

Some possible strategies include

1. Administration of probiotics and prebiotics to restore the composition of the intestinal microbiota.
2. Diet modification to promote the growth of beneficial bacteria.
3. Development of new treatments that can manipulate the intestinal microbiota to control diabetes (Figure 9).[48,49]

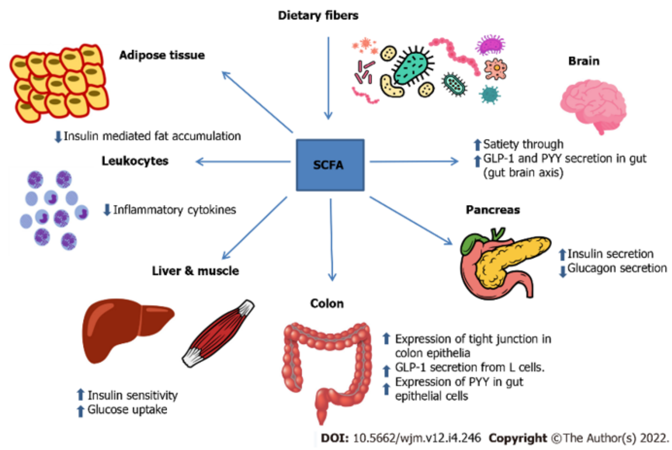

Figure 9 Role of gut microbiota and short-chain fatty acids in the pathophysiology of diabetes mellitus. SCFA: Short-chain fatty acids; GLP-1: Glucagon-like peptide-1; PYY: Peptide YY. The latest evidence linking Short-Chain Fatty Acids (SCFAs) to host metabolic health and Cardiovascular Disease (CVD) risk presents the evidence on possible biological mechanisms.

Sources: Doi: 10.1007/s13668-018-0248-8and Doi: 10.5662/wjm.v12.i4.246

Diabetes can accelerate the emergence of antibiotic-resistant bacteria

Published in the journal Science Advances, the research explained how the microbial environment in diabetic individuals favors resistance mutations and suggested new strategies to combat this growing health threat [Microbiologists at the University of North Carolina (UNC)].[50,51]

Staphylococcus aureus Rosenbach, 1884 (Bacillales: Staphylococcaceae), a leading cause of resistant infections, is the most common bacterium among. Findings from the diabetes study.[50-51] To investigate the relationship between diabetes and resistance, the researchers:

1. Used diabetic and non-diabetic mice infected with *S. aureus* and treated with rifampin, an antibiotic that rapidly triggers resistance.
2. After just five days of treatment, the results were revealing: In diabetic mice, rifampicin had little effect, and the bacteria developed resistance.
3. In non-diabetic mice, however, no resistance was detected.
4. The most striking finding was the rapid development of resistance, which occurred in just four days. However, when glucose was controlled with insulin, bacterial growth and resistance mutations decreased significantly (Figure 10).[50-52]

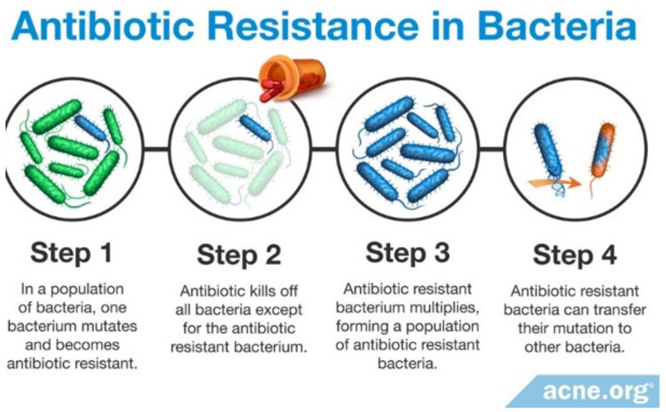

Figure 10 Mechanisms of antibiotic resistance. Bacterial resistance is the ability of a microorganism to grow in vitro in the presence of a given antimicrobial at concentrations higher than the highest concentration achieved by the drug in the bloodstream. Bacteria have two types of resistance: intrinsic resistance, which is their innate characteristic of being resistant to a given antimicrobial agent.

Source: Doi: 10.7476/9786557082331.0007 and https://fity.club/lists/suggestions/mechanisms-of-antibiotic-resistance/

The researchers now plan to expand their studies to human patients, including both diabetic and non-diabetic individuals, and to investigate resistance in other bacteria, such as *Enterococcus faecalis* (Orla-Jensen 1919), Schleifer & Kilpper-Bälz, 1984 (Lactobacillales: Enterococcaceae), and *Pseudomonas aeruginosa* Schroeter, 1872 (Pseudomonadales: Pseudomonadaceae).[50-51]

Changes in the microbiome translate into a greater risk of type 2 diabetes

These scientists have discovered specific viruses and gene variants within bacteria. In the study, which appears in Nature Medicine, the authors explain how they ed that species and strains of bacteria were linked to changes in gut microbiome function and a person's risk of type 2 diabetes [An interdisciplinary team of specialists from the Massachusetts Institute of Technology, Harvard University, and Brigham & Women's Hospital in Boston].[52,53]

These scientists have identified specific viruses and gene variants within bacteria. The research highlights phylogenetic diversity within species for strains of 27 species, such as *Agathobacter rectalis* Rosero *et al.,* 2016 (formerly *Eubacterium rectale*) (Eubacteriales: Lachnospiraceae), which explains differences between individuals in their risk of type 2 diabetes related to *E. rectale*. Our study presents strong microbial signatures across different groups. Many of these species have never been reported before (Figure 11)[7,53-55]

One important functional difference observed was the presence of a strain of *Prevotella copri S*hah and Collins 1990 (Bacteroidales: Prevotellaceae) Synonyms (*Xylanibacter ueki et al*., 2006), which can produce large amounts of Branched-Chain Amino Acids (BCAAs), was found more frequently in the gut microbiomes of patients with diabetes. Previous studies have shown that people with chronically high blood levels of BCAAs are at increased risk of obesity and type 2 diabetes (Figure 12).[53,54]

Citation: Santana MVO, Marchiori CH, Malheiros KP, et al. Bacteria cause diabetes mellitus: an integrative review. *MOJ Biol Med.* 2025;10(2):101‒112. DOI: 10.15406/mojbm.2025.10.00249

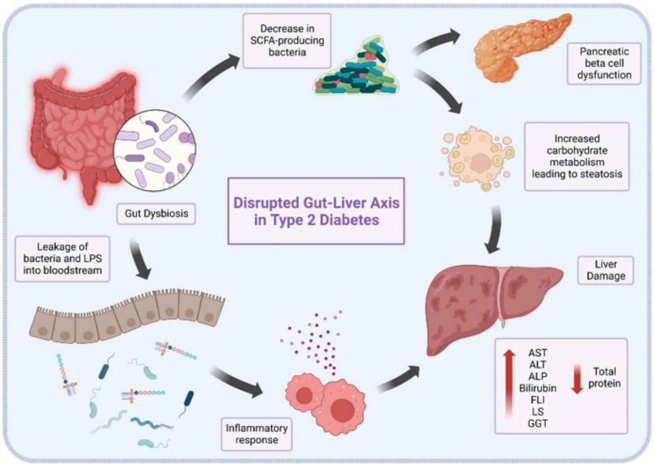

Figure 11 The role of gut dysbiosis in the pathogenesis and progression of type 2 diabetes-induced liver damage. Intestinal dysbiosis is an imbalance in the growth of microorganisms in the intestine. Imbalances in the intestinal flora, or gut microbiota, are primarily caused by a diet high in protein and fat and low in fiber, but can also be a consequence of certain medications or stress.

Source: Doi: 10.3389/fnut.2022.1052619

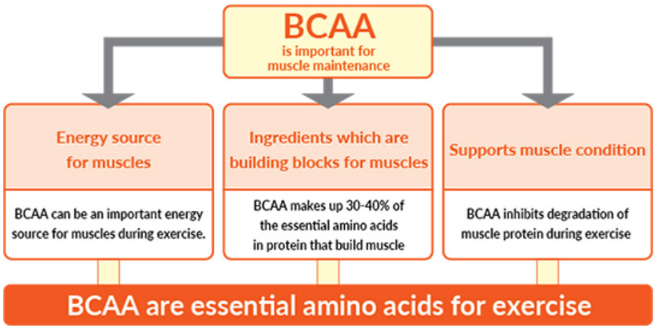

Figure 12 Branched-Chain Amino Acids (BCAA) is a general name for valine, leucine, and isoleucine. BCAAs are amino acids that suppress protein breakdown and are used as an efficient energy source during exercise. BCAAs are considered essential amino acids because the body cannot produce them on its own, so they must be obtained through dietary sources or supplements. They are commonly found in high-protein foods such as meat, dairy products, eggs, and legumes.

Sources: Otsuka Medical Devices and https://www.otsuka.co.jp/en/nutraceutical/about/nutrition/sports-nutrition/essential-

They discovered some bacteria that could cause type 2 diabetes

A new study has now suggested that bacteria may even be the cause of one of the most prevalent diseases of our time, type 2 diabetes. Researchers have found results confirming that prolonged exposure to a toxin produced by *S. aureus* causes all the specific symptoms of type 2 diabetes, including insulin resistance, glucose: intolerance, and systemic inflammation. "Type 2 diabetes was reproduced in cones simply by chronic exposure to the staph superantigen," [The team was led by Patrick Schlievert, PhD, professor and DEO of microbiology at IU Carver College of Medicine] (Figure 13).[56-58]

The findings that therapies targeting the removal of these staph bacteria or neutralizing the superantigens could potentially prevent or treat type 2 diabetes in the future, given the high number of cases. Obesity remains a known risk factor for developing type 2 diabetes; however, it is also a person's microbiome, the community of bacteria that lives on and influences our health. "What we're finding is that as people gain weight, they're more likely to be colonized by staph bacteria, and there are large numbers of these bacteria on the surface of their skin," [Patrick Schlievert].[56,58]

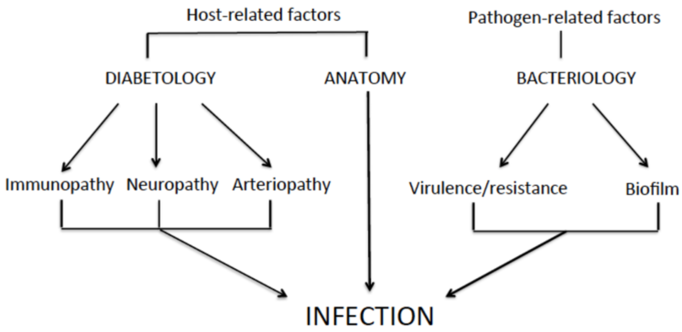

Figure 13 Interactions between metabolic, anatomical, and bacteriological factors in diabetic foot infection. Foot ulcers are common in diabetic patients. Its prevalence 15% and 25%. Infection of these ulcers is a frequent 40%–80%) complication representing a major cause of mortality and morbidity. The pathophysiology of diabetic foot infection is quite complex. The prevalence and severity are a consequence of host-related processes and pathogen-related factors virulence, antibiotic-resistance, and microbial organization.

Sources: Doi.org/10.3390/toxins8070209, Doi: 10.1001/Jama.293.2.217, Doi: 10.1001/Jama.293.2.217 Doi: 10.2337/Dc05-2425, Doi: 10.1002/Dmrr.2234, Doi: 10.1186/S12916-014-0232-0

Superantigens are toxins produced by all strains of Staph bacteria that disrupt the immune system severe effects in many staph infections, such as toxic shock syndrome, sepsis, or endocarditis [Patrick Schlievert]. Researchers show that superantigens interact with fat cells and the immune system to induce chronic systemic inflammation, which leads to insulin resistance and other symptoms characteristic of type 2 diabetes [Patrick Schlievert].[56,58]

By examining the levels of staph colonization on the skin of four patients with type 2 diabetes, Schlievert's equipment estimate confirmed that exposure to these bacteria was proportional to the dose of superantigen that caused the symptoms of type 2 diabetes. "I believe we have a way to intervene here and change the course of diabetes. We are working on a vaccine against superantigens, and we believe that this type of vaccine could prevent the development of type 2 diabetes." [Patrick Schlievert]. The team is investigating the use of a gel containing topical glycerol monolaurate, which kills staph bacteria on contact, as an approach to eliminating staph bacteria from human skin.[56,58]

Gut bacteria could revolutionize the treatment of type 2 diabetes

Insulin resistance contributes to metabolic syndrome and type 2 diabetes. The gut microbiota processes nutrients. Insulin resistance also affects carbohydrate metabolism. It accounts for 10% of an individual's total energy intake and plays a key role in obesity, prediabetes, and type 2 diabetes. Their recent study, published in Nature emphasizes the importance of gut bacteria in diagnosing, preventing, and treating conditions like obesity, prediabetes, and type 2 diabetes.[9,59,60]

This groundbreaking work highlights the human microbiome, the trillions of bacteria inside us, and could usher in a new era of medical innovation. Increased insulin resistance is linked to the presence of Bacteroidales (Lachnospiraceae) bacteria in the microbiome, which contributes to this resistance. Treatment options include culturing bacteria in higher concentrations of Bacteroides bacteria [Hiroshi Ohno at the RIKEN Center].[9,61,62]

To better understand potential treatment options, researchers grew bacteria in the lab and tested them on obese children. The bacterium *Alistipes indistinctus* Nagai *et al.,* 2010 (*Bacteroidales: Rikenellaceae*), showed promising results by lowering blood sugar levels. This could lead to new therapies and diagnostic tools for human health. Lachnospiraceae bacteria and probiotics containing *A. indistinctus* might help improve glucose intolerance in people with prediabetes. However, before recommending probiotics for insulin resistance treatment, these findings need to be confirmed through human clinical trials [Hiroshi Ohno, RIKEN Center] (Figure 14).[61-63]

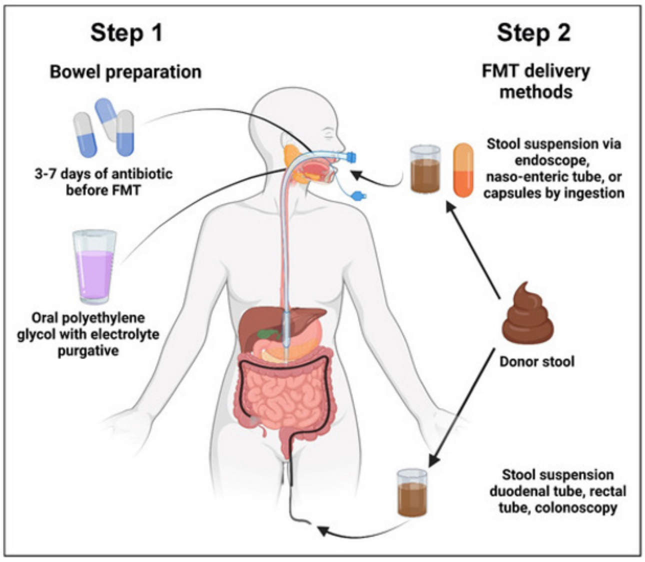

Figure 14 The two steps of fecal microbiota transplant. In Step 1, patients undergo bowel preparation with oral antibiotics followed by a laxative. At least 24 h after the last dose of oral antibiotics, the patient will receive the donor fecal material via capsule, naso-enteral tubes, or upper or lower gastrointestinal endoscopy.

Source: Doi.org/10.3390/cancers14030691

Fecal transplants, which involve introducing feces and bacteria from healthy donors into the recipient's gut, are commonly used to treat severe infections. Researchers are investigating how this knowledge can be applied to treat obesity, Crohn's disease, allergies, and other conditions. The potential use of intestinal bacteria to fight obesity and diabetes represents a promising avenue for future medical progress.[61,62]

Obesity is a complex disease with multifactorial origins: its onset is influenced by several causes, which can be individual, collective, social, economic, cultural, and environmental. This means that obesity is not only related to personal attitudes and behaviors. Obesity has significant impacts on the healthcare system, and these consequences are not limited to economic costs. This list also includes social costs, such as decreased quality of life, lost productivity, premature mortality, and problems related to social interactions.[64]

Crohn's disease is a syndrome that affects the digestive system, and its main symptoms are abdominal pain associated with diarrhea, fever, weight loss, and weakness due to difficulty absorbing nutrients. It is also an autoimmune disease that is a risk factor for bowel cancer.[65]

Ultra-processed foods harm good intestinal bacteria

Ultra-processed foods harm good gut bacteria, weaken the intestinal barrier, and increase the risk of diseases such as diabetes, heart problems, and depression. These foods reduce microbial diversity, promote pro-inflammatory bacteria, and decrease the production of short-chain fatty acids, which are vital for gastrointestinal health. Ultra-processed foods (UPFs) are industrial products made up of five or more ingredients, including sugars, refined oils, fats, salt, preservatives, and artificial additives. They often lack dietary fiber, which is essential for digestive health and microbiota.

Common industrial processes, such as the Maillard reaction, extrusion, and hydrogenation, improve the flavor, color, and texture of products, but at the cost of generating pro-oxidants and potentially carcinogenic compounds. In addition, these processes can cause lipid oxidation and the formation of free radicals, Low-Density Lipoprotein (LDL) cholesterol. Regular consumption of UPFs promotes the growth of pro-inflammatory bacteria and reduces microbial diversity, α-diversity, a key indicator of gut health. It also reduces the production of Short-Chain Fatty Acids (SCFAs), such as butyrate, which protect the intestinal mucosa and regulate inflammation (Figure 15).[66,67]

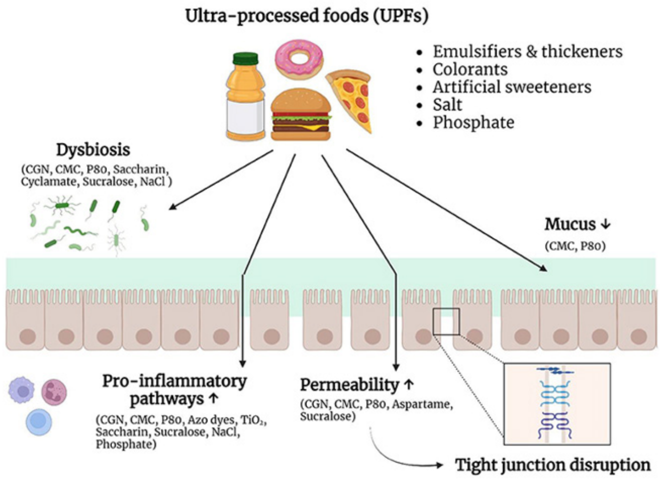

Figure 15 Ultra-Processed Foods (UPFs) combination of substances derived from foods and food additives. Non-nutritive ingredients and food additives are able to interact with the intestinal barrier and therefore play a role in the pathogenesis of Inflammatory Bowel Disease (IBD). Different food additives can induce dysbiosis, stimulate pro-inflammatory pathways, increase the epithelial permeability, or alter the mucus layer. The immune system encounters an increased bacterial exposure, which can result in chronic intestinal inflammation. CGN, Carrageenan; CMC, Carboxymethylcellulose; P80, Polysorbate-80; NaCl, Sodium Chloride; TiO2 and titanium dioxide.

Sources: Created with Biorender.com and Doi: 10.3389/fmed.2022.1058373

This leads to a state of systemic inflammation and oxidative stress, which increases the risk of chronic diseases. People who consume large amounts of UPFs have increased levels of harmful bacteria, such as genus *Granulicatella adiacens* Collins & Lawson, 2000 (Lachnospirales: Lachnospiraceae), and family Carnobacteriaceae, and a reduced presence of beneficial species, genus Blautia Liu et al., 2008 (Lachnospirales: Lachnospiraceae), *Lachnospira* Bryant and Small 1956 (*Lachnospirales: Lachnospiraceae*), and *Roseburia* Stanton & Savage 1983 (Lachnospirales: Lachnospiraceae).

Artificial emulsifiers also block the proliferation of anti-inflammatory bacteria, increasing intestinal permeability and allowing bacteria to enter the bloodstream. This phenomenon is associated with metabolic and cardiovascular disorders. Harmful health consequences of dysbiosis caused by ultra-processed foods.[67]

Artificial emulsifiers also block the proliferation of anti-inflammatory bacteria, increasing intestinal permeability and allowing bacteria to enter the bloodstream[67] This phenomenon is associated with metabolic and cardiovascular disorders. Harmful health consequences of dysbiosis caused by ultra-processed foods:

1. **1. Type 2 diabetes:** The altered microbiota favors an imbalance between Firmicutes and Bacteroidetes bacteria, which increases the risk of type 2 diabetes low presence of *Akkermansia muciniphila* Derrien *et al.,* 2004 (Verrucomicrobiales: Akkermansiaceae), a bacterium essential for regulating insulin and body weight, is common in individuals who consume high levels of UPFs[34,66]
2. **Inflammatory bowel disease**: Both ulcerative colitis and Crohn's disease are associated with intestinal dysbiosis, a reduction in anti-inflammatory bacteria, and increased permeability of the intestinal mucosa. Changes in the production of bile acids, SCFAs, and tryptophan metabolism are also observed.
3. **3. Colorectal cancer**: The Western dietary pattern rich in UPFs is associated with a higher incidence of Colorectal Cancer (CRC). An increase in bacteria such as *Bacteroides fragilis* Elsaghir, Redd, 2023 (Bacteroidales: Bacteroidaceae), *Fusobacterium nucleatum* Knorr, 1922 (Fusobacteriales: Fusobacteriaceae), and *Parvimonas micra* Tindall (2006) (Eubacteriales: Peptoniphilaceae), which can generate toxins and Reactive Oxygen Species (ROS), capable of damaging DNA, has been identified.
4. **Neurological diseases:** Direct connection between the intestinal microbiota and the brain, known as the gut-brain axis. Microbial alterations are implicated in diseases such as Parkinson's, Alzheimer's, and severe depression, where a decrease in beneficial bacteria such as Phylum Firmicutes and Phylum Bacteroidota Garrity and Holt 202, and Actinobacteria Goodfellow 2012 (Phylum Bacteroidotais) observed. [9,67]

Scientists at the Weizmann institute have sequenced the DNA of pathogens and their hosts at a unique level. This could explain why only a few subspecies cause serious illness

Like people, bacteria are independent entities. The social type of *Salmonella* can contain proteins that control cellular systems. One researcher has identified differences between the proteins of 2,500 subspecies of *Salmonella*. The researcher identified differences between the proteins [published in the Proceedings of the National Academy of Sci/Doi: 10.1073/pnas.221881212] [Roi Avraham of the Weizmann Institute of Science] reveals what makes some bacteria particularly virulent (Figure 16).[68-70]

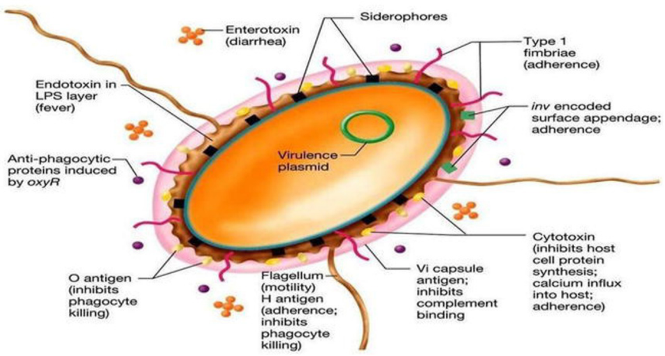

Figure 16 *Salmonella* Lignières, 1900 (Enterobacterales: Enterobacteriacea) virulence factors. Structures of a typical Gram-negative bacterial cell. Presence of capsule, cell wall, cell membrane, cytoplasm, nucleoid, fimbriae, flagella, ribosomes, and plasmid.

Sources: Madigan and Martinko, 2007, https://www.researchgate.net/publication/41584156, Adapted from Tortora *et al.,* 2012, and Faria, A. M., 2013

By examining the molecular processes, genetic material, and RNA of individual cells, a barcode scanner is needed to identify each species. Each mutant bacterial species expresses the "matched sequence" of the host-bacteria "pair" at a single cell level [Dr. Noa Bossel Ben-Moshe]. Twenty-five (25) mutant Salmonella species that infect immune cells called macrophages lack a protein that suppresses the host's immune system. The new function has not yet been discovered, and the desired functions will allow us to uncover beneficial bacteria.[69,70]

Basic research has focused on developing methods to combat bacterial resistance to antibiotics, reduce bacterial virulence, and enhance the host cell's immune response. Study allows us to study Dr. Camilla Ciolli Mattioli, Neta Blumberger, Dr. Gili Rosenberg, Aryeh Solomon, and Dr. Dotan Hoffman from the Department of Immunology and Regenerative Biology at the Weizmann Institute.[69,70]

Integrating recent studies on SCFA-mediated GLP-1 stimulation or LPS-induced inflammation models to support the studies (Initial study and recent study)

Between 2023 and 2025, Anvisa received communications from various sources regarding the use of glucagon-like peptide-1 (GLP-1) receptor agonist medications, advertised as "slimming pens," which indicated risks of unsupervised use and a strong aesthetic appeal, detrimental to the recommendations, indications, risks, and benefits approved in the package insert.[71]

One postulated link is the bacterial fermentation of "indigestible" prebiotics to short-chain fatty acids (SCFAs), which in turn modulate the release of gut hormones controlling insulin release and appetite. We show here that SCFAs trigger secretion of the incretin hormone glucagon-like peptide (GLP)-1 from mixed colonic cultures in vitro. These results highlight SCFAs and their receptors as potential targets for the treatment of diabetes. Initial study and recent study.[72,73]

Conclusion

Diabetes impairs blood sugar regulation and weakens the immune system, allowing bacteria to multiply rapidly. This population increase facilitates the emergence of resistance mutations. It was revealed that people with diabetes mellitus have a higher risk of developing infections caused by antibiotic-resistant bacteria.

Acknowledgments

None.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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