

REVIEWS

The Impact of Antibiotic Use on the Human Gut Microbiome: A Review

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General Interest in the gut microbiome has been steadily increasing over the last decade as diagnostic tools for exploration have become readily available, which has increased information available and lead to novel innovation in the way some diseases are treated. Gut microbiota contributes to host health much more than previously thought, increasing the potential for healing bodies with therapies that use the body's natural defense mechanisms as an alternative to current practices that use antibiotics. This in turn reduces the risk of antibiotic resistance. Treatment initiatives that focus on drug therapy can lead to overuse of broad-spectrum antibiotics, which have been shown to have detrimental effects on the human body and can cause many gut issues. In addition, antibiotic resistance remains a critical public health challenge, necessitating the exploration of alternative therapeutic strategies to traditional antibiotics. This review examines the impact of antibiotic use on gut microbiota and consequences on host health and immunity both short- and long-term effects, with a focus on the potential for microbial dysbiosis. The connection between dysbiosis and antibiotic resistance is explored as well as the potential for recovery through alternative therapies, including microbiota-modulating and targeted antimicrobial therapies, by assessing their short- and long-term potential as antimicrobial alternatives.

Introduction

The human gastrointestinal tract is colonized by bacteria, archaea, and eukarya estimated to be about 10^{14} microorganisms in abundance.¹ These species collectively make up the gut microbiome and have coevolved with humans in a mutually beneficial relationship over the course of several millennia. 2 of the 3 major gut bacteria families were found to have a common ancestor that originated more than 15 million years ago.² These benefits include strengthening gut integrity, harvesting energy, protecting against pathogens, and regulating the hosts' immunity. The microbiota strengthens gut integrity through induction of tight junction proteins, stimulating proteins such as sprr2A, which support epithelial cell cohesion and enhance mucus production. The microbes present in the gut are able to harvest energy through breaking down proteins from the food consumed, regulating lipid metabolism, and fermenting dietary carbohydrates. There are several mechanisms that the microbiota use to protect against pathogens and regulate host immunity including modulating gut-associated lymphoid tissue development, which influences B- and T-cell populations; inducing

cytokine production; and maintaining immune homeostasis.³ As with all homeostasis mechanisms in the body, these benefits rely on the delicate interactions between the microbiota and the mucosal immune system for proper function.¹ One factor that can interfere with this homeostatic nature is antibiotics.⁴

Antibiotics are used to defend bodies against harmful pathogens, but they incite unintentional agitation in the commensal gut microbiome and have the potential to cause substantial long-lasting damage.⁴ This has been especially noted with the use of broad-spectrum antibiotics that do not have a specific target and work to effectively remove most microorganisms within a given area of the body, thus decreasing microbiome diversity and leading to compromised immunity. A fitting example of this is *Clostridioides difficile* infections, which are common and severe gastrointestinal illnesses that can occur due to compromised immunity. Broad-spectrum antibiotic use creates space for the overgrowth of these opportunistic bacteria by eradicating the beneficial bacteria of the gut, leading to symptoms that range from mild diarrhea to death depending on the immunity of the host.⁵ Once in the body, these medications break down and reach both the cecum and colon where they can exert those devastating effects. Short-term effects of broad-spectrum antibiotics include diarrheal disease and to the potential for antibiotic-resistant bacteria strains by promoting antibiotic-resistant genes, which will be discussed more in depth later in this review. Long-term effects are still being studied, though some studies have suggested a link to allergies and obesity. These conditions are due to the decrease in diversity of species from antibiotic therapy.⁶ Full recovery of the microbiota is possible after use of antibiotics, but is dependent on several factors including lifestyle, diet, duration of use, age, and type of antibiotic used.

The diversity of microbiomes is evaluated by the number and abundance of distinct types of organisms.⁷ There is an ideal balance of microbial species needed for a healthy microbiome. For example, a decrease in gut microbiota can lead to inflammatory bowel disease, while an increase in vaginal diversity can lead to urinary tract infections or bacterial vaginosis. While there are many body microbiomes that are of concern, the gut microbiome is the main focus of this review.^{6,8,9}

Developmental Considerations

Despite antibiotics being recognized as a major contributor to increased life expectancy in the 20th century, they have had an inadvertent negative impact on the human gut microbiome from overuse.¹⁰ The microbiome has a profound impact on the hosts' immunity as a whole. It begins with infants who get their microbiome from their mother, which plays an essential role in the development of gut immunity. Some researchers believe the gut microbiome begins developing before birth through the amniotic fluid,

placenta, and meconium but due to possible contamination of samples from normal flora and variation in interpretation, this is still highly debated in current literature.^{11,12} More testing to determine the state of prenatal colonization is needed before a definitive answer could be determined. The community of microorganisms gradually becomes more diverse with age, typically reaching stability at between 2 and 4 years of age. In infancy, facultative anaerobes make up the majority of colonized microbes and include *Staphylococcus*, *Streptococcus*, *Escherichia coli*, and *Enterobacteria*. Over time with exposure, the composition shifts to species such as *Bifidobacterium*, *Bacteroides*, and *Clostridioides*. At that 4-year mark, an abundance of Bacteroidetes and Firmicutes are seen.¹³ These first 4 years of life are critical for establishing a healthy microbial community, which is shaped by factors including, but not limited to, feeding methods, diet, environmental exposures, and birthing route. Use of antibiotics in infants has been seen to alter gut microbial diversity, which leads to lower immunity and inferred increased susceptibility to infection.¹⁴

Several studies reviewed by McDonnell et al¹⁵ that involved children aged 1 to 12 years who were exposed to antibiotics found that the children had statistically significant reductions in microbiome richness with long-term exposure to antibiotics, which was noted to be between 12 to 14 days of use. While the microbiome did not recover from this reduction for up to 2 years, most cases did see full recovery after treatment. The same group of studies found that there was a decrease in alpha diversity, the number of individual bacteria from each species present in a sample and disrupted taxonomic composition depending on the antibiotic used.¹⁵ There was an overall reduction in alpha diversity using the Shannon diversity index, with a mean reduction of 0.86 but total values ranging from a reduction of 0.13 to 1.59. The Shannon Diversity Index measures the species diversity in a community; this study in particular used the index to analyze the alpha diversity of the group.¹⁵

Similarly in adults, antibiotic use caused changes in microbial composition that can persist for several weeks or lack restoration altogether. This research determined that disruptions in microbiota were influenced by antibiotics that reduced diversity from weeks to sometimes years. Certain antibiotics, such as clindamycin, disrupt microbiota long-term, which makes sense as it is generally thought of as a broad-spectrum antibiotic. Other influences included whether the drug was bactericidal or bacteriostatic, with the former being associated with increased gram-positive bacteria and the latter gram-negative bacteria. Bactericidal drugs kill the bacteria by targeting cell wall synthesis, DNA replication, or protein synthesis and are associated with rapid depletion of the short-chain fatty acid (SCFA) producing bacteria. Bacteriostatic drugs inhibit the growth of bacteria by interfering with metabolic processes and, due to this partial survival, they lead to gradual shifts

in abundance and selective overgrowth. Clinical consequences of bactericidal drugs include increased risk of *C. difficile* and long-term resistance gene persistence, while bacteriostatic drug use can lead to systemic inflammation.¹⁶

Time-Dependent Dysbiosis

Dysregulation of the immune system can leave the host susceptible to infection. Microbiota are essential for the proper development and maturation of the immune system as they prevent colonization of pathogenic microbes, stimulate antimicrobial peptides through pattern recognition receptor- microbe-associated molecular pattern (PRR-MAMP) interactions, and produce metabolites that lower pH or disrupt pathogen membranes.³ Antibiotic-induced changes influence several aspects of microbiota functionality as well as bacterial behavior such as changes in metabolites, accumulation of xenobiotics, changes in bacterial signaling patterns, and increasing the antibiotic resistance gene (ARG) reservoir in the human gut.¹⁴ Three mouse studies found disturbances in metabolites including upregulation of cytokine gene expression, metabolite composition variation with clindamycin treatment, and decreased levels of SCFAs and amino acids, which are products of bacterial metabolism.¹⁷⁻¹⁹ The decrease in SCFAs suggest reduced bacterial fermentation due to antibiotic-induced microbial changes. The study by Zhao et al¹⁹ in particular highlighted the link between microbiota composition and host metabolism. The microbiota present in the gut affects xenobiotic half-life, the chemical structure of pharmaceutical compounds, and may influence the host's ability to metabolize these antibiotics, heavy metals, or environmental chemicals. Bacterial signaling patterns can be induced from aminoglycosides, β -lactams, vancomycin, and oxacillin and these signals can be responsible for biofilm formation. The formation of these biofilms then feeds back into antibiotic resistance.²⁰ ARGs have reported strong resistance to β -lactams, fluoroquinolones, tetracycline, macrolide, and sulfonamide, which are all very commonly prescribed classes of antibiotics. The study by Díaz-Palafox et al,²¹ however, used bacteria isolated from wastewater treatment facilities and therefore may have direct relevance limitations when discussing in the gut microbiome, as there are several outside factors that could have influenced the results of the study.

The severity of dysbiosis that occurs is directly related to how long a patient has been using the antibiotic therapy.²² Short-term use, approximated to be 5 to 7 days, was found to result in significant disturbances in the normal flora of the gut up to 2 years after treatment.¹⁴ A study published in 2022 sought to understand this further and found that short-term antibiotic use, a 5-day course, decreased the microbiome bacterial load and richness, as well as agitated the community structure.⁴

With prolonged antibiotic use, potential long-term effects include obesity, immune system changes, and insulin resistance.²² As of 2021, evidence suggests that dysbiosis is linked to metabolic diseases including obesity. Reduced diversity has shown correlation with increased intestinal permeability, metabolic endotoxemia (endotoxins entering the bloodstream), and systemic inflammation, which are all factors that can promote obesity. Some animal studies have found compelling evidence to support that early-life antibiotic exposure leads to changes in SCFA production. This relates to obesity SCFAs that are found in higher abundance when compared with individuals with no known underlying condition, but this link is understudied, which limits what conclusions can be drawn. While animal studies strongly support a link between antibiotic use and obesity via disruption of the gut microbiome, human data are limited.²³ There is a need for large-scale randomized clinical trials to definitively explain this relationship.

Changes in the immune system that directly relate to the gastrointestinal system can include reduced gastric motility, improper development and function of intestinal cells, and reduced thickness of the colons mucus layer, which leaves it more vulnerable to opportunistic pathogens.¹⁴ A 2015 study by Candon et al²⁴ found that interleukin 17 (IL-17), a major cytokine of the host immune system, was reduced with broad-spectrum antibiotics in those with diabetes. The same study found a connection between antibiotic-induced changes in the microbial colonization and type 1 diabetes.²⁴

Mechanisms of Resistance and Microbial Adaptation

The gut is a well-known reservoir for a vast number of ARGs and a suitable place for the horizontal exchange of those genes spreading adaptive traits. In an analysis published in 2019, Willman et al²⁵ looked at changes in the gut resistome, the collection of ARGs found within the gut microbiome, when treated with the oral antibiotics ciprofloxacin and cotrimoxazole. The ARG classes present in this study were aminoglycosides, β -lactamases, fluoroquinolones, glycopeptides, macrolide-lincosamide-streptogramin, nitroimidazoles, phenicols, sulfonamides, tetracyclines, and trimethoprim. The abundance of ARGs sulfonamide and trimethoprim rose significantly with cotrimoxazole when compared with that of ciprofloxacin. The selection pressure was varied among the ARG classes; for example, there was positive pressure for CTX-M (enzymes that grant resistance to a wide range of β -lactam antibiotics) with ciprofloxacin but negative for cotrimoxazole. This could mean that while antimicrobial therapy is necessary, it is also a driver for antibiotic resistance. Cofactors such as bilirubin, creatinine, and proton pump inhibitors all shaped the abundance of ARGs and were suspected of being the drivers for the great diversity seen. This could be attributed to the specificity of a patient's microbiome as well as resistome and was confirmed by multivariate analysis.²⁵

A 2015 study²⁶ showed Swedish exchange students who traveled to the Indian peninsula or Central Africa had elevated ARGs in their gut microbiomes for routinely used antibiotics including sulfonamide, trimethoprim, and β -lactams. Of the total 178 ARGs detected, 23 were found among all samples and an additional 35 were found in more than 90% of the sequencing libraries. However, many of the core resistome high abundance genes, including vancomycin and tetracycline resistance genes, remained stable and had little to no change when interacting with antibiotics. The core resistome refers to the ARGs that stay consistently present and stable across multiple samples. This study may contribute to the efforts being made for alternative treatment by providing insight into the geographical spread of antibiotic resistance.²⁶

Alternative and Adjunctive Therapies

Alternative therapies to antibiotics are gaining prominence in combating antimicrobial resistance and working to preserve gut health including prebiotics and probiotics, fecal microbiota transplants (FMTs), phage therapy, and monoclonal antibody treatments. Certain options, such as prebiotics, can be used in conjunction with antibacterial therapies to restore the disrupted microbiota and reduce risk of infections. Enhanced mucosal barrier function and reduced inflammation are 2 of the short-term effects that make this alternative appealing.²⁷ Probiotics are another option that is used in conjunction with current therapies that mitigate the risk of superinfections such as those seen with *C difficile*.²⁸

FMTs have also been used to regain microbial balance in the gut. This method is primarily used for treatment of *C difficile* infections and reinfections with resolution rates up to 90%. In addition to high recovery rates, the FMT procedure is considered a relatively cheap option with high efficacy, making it a strong alternative option.²⁹ The data on other gastrointestinal disorders, including Crohn disease, irritable bowel syndrome show low quality of evidence and show much lower clinical remission rates with FMTs.²⁹⁻³⁵ While short-term benefits have been studied and seen repeatedly in research results, potential long-term effects of altering a patient's microbiota are unknown and need to be studied to gauge the true risk of this procedure.³⁶ Currently, there is a National Institutes of Health-funded FMT national patient registry designed to assess these long-term effects with the study design expanding 10 years. The study is set to complete in 2027, but early results have shown strong efficacy and a good safety profile.³⁷

Alternatives such as phage therapy and monoclonal antibodies are being investigated for their potential in bacterial resistance. The major advantages to these therapies are they can be highly target specific, which mitigates the adverse effects on the host microbiota and only focuses on the pathogens present. With the steady rise in antimicrobial-resistant pathogens, there is

an urgent need for these types of therapies. Bacteriophages only replicate in target bacteria and work without disrupting the normal microbiome. This makes them a particularly desirable candidate for precision medicine. Something to note with this method, however, is that there is a lack of clinical standard when it comes to dosage volume and the phage itself can be unstable. While phage therapy is a promising avenue for treatment to reduce antibiotic resistance, more research is needed to determine a standard protocol for dose administration as well as studying how to make the phage more stable.²⁸ Monoclonal antibodies (mAbs) are produced in a laboratory and mechanistically behave like human antibodies. They have been used in both viral and bacterial infections in humans. These mAbs can prevent biofilm formation, provide complement-mediated killing, and can directly neutralize bacterial toxins, which are distinctly different mechanisms than those of antibiotics. One of the major benefits that come with mAbs is that there is reduced selective pressure for resistance, which is a growing public health concern. There are currently 3 antibodies approved to treat bacterial infections.³⁸ Despite mAbs being a great prospect that have substantial specificity possibilities when it comes to treatment, they have a high cost of production and could benefit from further research to find optimal targets in the body when it comes to bacterial infections.

Conclusions

Antibiotics have played a vital role in combating diseases and extending human life expectancy, although their impact on the gut microbiome is concerning. These medications, especially broad-spectrum antibiotics, not only target harmful pathogens, but also destroy beneficial native bacteria that are essential for maintaining gut and overall health. The evidence presented in this review highlights the extent of microbiota dysbiosis that comes with antibiotic therapy. Both short- and long-term use can lead to significant disruptions in microbial diversity. This disruption is especially critical during early life, where the establishment of a diverse and stable microbiome is vital for immune development. Adults are not immune to these effects either, with studies demonstrating that antibiotic courses can induce persistent shifts in microbial composition and function for months or even years after treatment.

Despite full recovery of microbiota being possible in most cases, the risk of antibiotic resistance and persistent dysbiosis emphasizes the urgent need for more targeted antimicrobial strategies. Alternative therapies such as probiotics, FMTs, phage therapy, and monoclonal antibodies seem promising, but further research is essential to refine these approaches and to fully understand long-term efficacy and safety. These alternatives highlight a growing shift toward precision medicine, which would work to preserve the gut microbiome in the patient during treatment. This would be a great point for future research opportunities, to include alternative treatment methods that would help preserve the intestinal microbiome from the consequences of

dysbiosis during treatments. More research is needed to explore the specific targets of such alternative treatments as well as the financial feasibility of incorporating them into medical usage. As it stands, the 2 most specific options, phage therapy and mAb therapy, have an excessive cost that makes them undesirable for use. Ultimately, understanding the intricate relationship between antibiotics and the gut microbiome is essential for developing safer and more effective clinical practices.

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