Exposure to air pollutants and the gut microbiota: a potential link between exposure, obesity, and type 2 diabetes

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ABSTRACT
Work has shown that increased exposure to air pollutants independently contributes to obesity and type 2 diabetes risk, yet the exact mechanisms underlying these associations have not been fully characterized. The current review summarizes recent findings regarding the impact of inhaled and ingested air pollutants on the gut microbiota. Animal and human studies provide evidence that air pollutants, such as particulate matter, nitrogen oxides, and ozone, have the potential to alter the gut microbiota. Further, studies suggest that such exposure-induced alterations to the gut microbiota may contribute to increased risk for obesity and type 2 diabetes through inflammatory pathways. Future work is needed to fully understand the complex interactions between air pollution, the gut microbiome, and human health. Additionally, advanced sequencing methods for gut microbiome research present unique opportunities to study the underlying pathways that link increased air pollution exposure with obesity and type 2 diabetes risk.

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Introduction
Globally, approximately 39% of adults are overweight and 13% are obese.¹ These high rates of overweight and obesity have profound implications for global health as excess adiposity increases the risk of numerous diseases, including cardiovascular disease and type 2 diabetes.² By 2030 an estimated 552 million people worldwide will have diabetes,³ and an estimated 90-95% of these cases will be type 2 diabetes.⁴ Although low levels of physical activity and a Western diet are the primary risk factors for obesity and type 2 diabetes, genetics,⁵ low socioeconomic status, and geographic location⁶ also contribute to the etiology of these diseases. Furthermore, exposure to ambient and traffic-related air pollution has been shown to influence the development of obesity⁷,⁸ and type 2 diabetes.⁹–¹² For example, a recent study reported that substantial geographical variation in the global burden of diabetes was attributable to air pollution, with elevated concentrations of particulate matter <2.5 µm in diameter (PM<sub>2.5</sub>) contributing to approximately 3.2 million incident cases of diabetes.¹³

Based on current evidence, exposure to air pollutants may contribute to obesity and type 2 diabetes through inflammatory pathways and alterations to metabolism. For example, animal models indicate that exposure to traffic-related air pollution and ambient particulate matter (PM) result in inflammation in the lungs and adipose tissue.¹⁴–¹⁶ Animal models also illustrate an increase in white adipose tissue and inhibition of lipolysis due to increased exposure to polycyclic aromatic hydrocarbons (PAH),¹⁷ which are byproducts of traffic-related fuel combustion. Furthermore, certain components of PM such as transition metals and lipopolysaccharides may penetrate the systemic vasculature and activate inflammatory pathways via toll-like receptors.¹⁸ In addition to the above mentioned hypothesized mechanisms, a growing body of literature suggests that air pollutants may alter the composition and function of gut bacteria.¹⁹–²⁵

The gut microbiome plays a vital role in human health by facilitating an array of metabolic and immune processes through microbial-host exchanges of metabolites,²⁶,²⁷ breakdown of complex
dietary carbohydrates, and synthesis of essential vitamins. Within a healthy gut, the intestinal mucosal barrier limits the translocation of microbes and microbial products from the lumen into systemic circulation. Notably, obesity and type 2 diabetes are characterized by distinct gut microbial profiles, including decreased microbial diversity and alterations in the relative abundance of Firmicutes and Bacteroidetes, which may alter bacterial fermentation pathways that decrease the production of short-chain fatty acids (SCFAs) and increase inflammation. These alterations can also induce bacterial overgrowth, breakdown of the intestinal barrier, bacterial and metabolite translocation, and/or chronic endotoxemia. Therefore, exposure to air pollutants may contribute to increased risk for obesity and type 2 diabetes through alterations to gut microbial composition and function.

Although the relationships between air pollutants and the gut microbiota are a relatively new field of study, a growing body of work suggests that increased exposure to air pollutants may impact obesity and type 2 diabetes via the gut microbiota. In 2014, a literature review summarized the evidence linking exposure to air pollutants with the gut microbiota and inflammatory diseases. The current review provides an updated summary of studies published between February of 2000 and June of 2019 in order to synthesize recent evidence linking increased exposure to air pollutants with alterations to the gut microbiota in both human studies and animal models. The review further discusses the potential health implications of such exposure-induced alterations to the gut microbiota in regard to obesity and type 2 diabetes.

Methods

A comprehensive examination of the literature was conducted to summarize findings related to exposure to air pollutants and the gut microbiota that were published between February of 2000 and June of 2019. Searches were performed between October 2018 and October 2019. PubMed and ProQuest were searched for articles that contained terms in the title and/or abstract that were relevant to the current review. The terms included: “gut microbiome” or “gut microbiota” AND (“ambient air pollution,” or “particulate matter,” or “traffic-related air pollution,” or “nanoparticles”). Bibliographies of relevant articles were also examined as well as papers previously known to the authors.

As shown in Supplemental Figure 1, PubMed and ProQuest yielded 40 references that were read and evaluated for their relevance to the current review. Of these 40 references, 18 were excluded since they did not examine air pollution and 6 were excluded as they did not include assessment of the gut microbiota. Ten references were excluded for being literature reviews; however, these reviews were used to identify two additional references that were relevant to the current review. This process resulted in eight references that we included in the current review (five animal studies and three human studies). Lastly, if a reference included both animal and human studies, each was reported separately in their respective sections.

Studies included in this review examined the gut microbiota using 16S rRNA amplicon sequencing or phenotypic indicators of bacterial function such as SCFA production. Studies also examined a wide range of air pollutants, including ambient nitrogen dioxide (NO₂), ozone (O₃) and PM of various size fractions, including PM <10 µm (PM₁₀), <5 µm (PM₅), <2.5 µm (PM₂.₅), and <0.1 µm in aerodynamic diameter (ultrafine/nanoparticles). Nitrogen oxides (NOx), sulfur dioxide (SO₂) and PAHs were also included. Many of these particles are released by vehicles via the burning of fossil fuels, as well as from other sources, like the degradation of tires and brakes.

Potential mechanisms by which air pollutants impact the gut microbiota

Epidemiological studies have provided preliminary evidence that exposure to airborne particles may alter the gut microbiota and gut health. For example, several studies have found associations between elevated levels of air pollution exposure and gastrointestinal diseases, including inflammatory bowel disease (IBD), irritable bowel syndrome, appendicitis, and gastroenteric disorders in infants. Cigarette smoke, which contains particulates that are present in ambient and
Figure 1. Hypothesized mechanisms linking exposure to air pollutants with the gut microbiota and increased risk for obesity and type 2 diabetes.

The figure shows hypothesized mechanisms linking air pollutants with the gut microbiome. Inhaled air-borne particles reach the gut via ingestion following mucociliary clearance of mucus membranes (or ingestion of contaminated food or water). Additionally, ozone-induced activation of the sympathetic nervous system may result in the release of hormones. Each of these pathways may result in gut microbiome facilitated biotransformations, changes in gut bacterial diversity, alterations in the relative abundance of gut bacterial taxa, tight junction disruption, and activation of immune cells and adrenergic neurons.
traffic-related air pollution, has also been shown to alter the composition of the gut microbiota in human and animal studies. Upon human exposure to air pollutants, there are several potential routes of interactions between air pollutants and the gut microbiome, which may alter gut bacterial composition as well as the luminal environment. For example, the gastrointestinal (GI) tract is exposed to air pollutants through inhalation and ingestion. Following inhalation, large particles are deposited in the upper airway, trachea, or larger bronchi while smaller particles, such as PM$_{2.5}$, can reach the bronchioles and alveolar spaces where they are sequestered by alveolar macrophages. Particles in the mucus layer, as well as those isolated by macrophages, are transported to the oropharynx via mucociliary clearance and are subsequently ingested, providing a pathway for airborne particles to reach the GI tract. In addition to inhalation, PM can be directly ingested through contaminated food and water. It has been estimated that humans ingest an average of $10^{12}$–$10^{14}$ particles a day on a Western diet. Thus, the GI tract is exposed to large amounts of PM, which has the potential to exert pro- and anti-bacterial effects on gut bacteria that may alter gut physiology, including immune responses, metabolism, and intestinal permeability (Figure 1). For example, studies suggest that components of air pollution, such as elemental carbon, black carbon, nitrates, sulfates, and toxic metals like lead and arsenic may alter host bacterial communities through antimicrobial properties and the formation of biofilms.

Ozone is another central component of ambient air pollution that is produced by the reaction of NOx, volatile organic compounds, and sunlight. Although numerous studies have demonstrated the deleterious effects of increased O$_3$ exposure on respiratory health, few have examined the impact of O$_3$ on the central nervous system and gut health. Prior work suggests that increased O$_3$ exposure may impact the hypothalamic-pituitary-adrenal (HPA) axis, resulting in increased corticosteroid levels and alterations to the gut microbiota. Further, O$_3$ exposure has been shown to increase plasma adrenocorticotropic hormone and corticosterone concentrations. PM and SO$_2$ exposure have also been shown to stimulate the autonomic nervous system, which may impact the gut microbiota through changes to GI tract motility and secretion. Lastly, reciprocal communication through the gut-brain axis may increase the production of norepinephrine at the gut lamina propria, thereby contributing to increased proliferation of gram-negative bacteria in the lumen.

### Exposure to air pollutants alters the composition of the gut microbiota in animal models

A number of rodent studies have investigated the effects of inhaled and ingested air pollutants on measures of gut microbial diversity, and a summary of these five studies can be found in Table 1. A higher diversity may be indicative of a healthier gut microbiota and these measures include alpha and beta diversity, which are measures of species richness and evenness within a sample and differences in microbial abundances between multiple samples, respectively. In one study, 12 low-density lipoprotein receptor knock out mice were fed a high-fat diet and exposed to ambient ultrafine particles for 10 weeks (3 d/week). As a result of this exposure, gut microbial alpha diversity decreased and beta diversity increased. Another study exposed 10 mice to PM$_{2.5}$ for 3 weeks using concentrated air from Chicago, and found increased alpha diversity in the small intestine, colon, and feces, as well as increased beta diversity throughout the GI tract. In another study, 10 mice were exposed to PM$_{2.5}$ concentrated air from Shanghai for 1 y. While PM$_{2.5}$ exposure did not alter gut microbial alpha diversity, there were decreases in measures of gut bacterial community richness (i.e., ACE and Chao-1) when compared to mice exposed to filtered air. These mixed findings may be due to regional differences in the chemical composition of PM$_{2.5}$ as well as differences in the duration of exposure.

In addition to measures of gut microbial diversity, PM exposure has also been shown to alter the relative abundance of gut bacterial taxa in murine models (Figure 2). In one study, the relative abundance of bacterial taxa was measured in mice exposed to PM$_{2.5}$ concentrated air from Shanghai for 1 y. Increased exposure resulted in an increase
of nine bacterial taxa, a decrease of 15 bacterial taxa, as well as impaired insulin and glucose tolerance. In another study, mice exposed to inhaled PM$_{2.5}$ for 3 weeks displayed a decrease in the relative abundance of Firmicutes and several bacterial families within the Firmicutes phyla, such as Staphylococcaceae. At the same time, exposed mice exhibited an increase in the relative abundance of Verrucomicrobia bacteria and TNF-alpha levels.

The table shows the five murine studies included in this review. The table describes the animals examined, experimental design, and key findings. These studies examined the associations between inhaled and/or ingested air pollutants and gut microbial diversity, the relative abundance of gut bacterial taxa, gut barrier integrity, intestinal inflammation, and/or insulin resistance.
abundance of bacteria belonging to the *Bacteroidetes* phyla, including bacteria in the *Rikenellaceae* family. In another study, seven interleukin-10 knock out mice (IL-10\(^{-/-}\)) and eight wild type (WT) mice were gavaged with ambient PM\(_{10}\) collected from Ontario, Canada for 35 d. In WT and IL-10\(^{-/-}\) mice, ingestion of PM\(_{10}\) increased in the relative abundance of the *Verrucomicrobia* phyla relative to the control mice. Both groups of mice exposed to PM\(_{10}\) also had alterations in cecal concentrations of SCFAs (e.g., increased isovalerate and isobutyrate, and decreased butyrate and valerate), which are important mediators of gut barrier integrity and predominantly produced by bacteria in the gut.

Another study examined PM smaller than 0.1 µm in diameter, which is the smallest fraction of PM and may have differential effects on physiology when compared to larger particles. This study orally exposed 12 mice to ultrafine particles 3 d a week for 10 weeks and found that the relative abundance of *Verrucomicrobia* was significantly higher in mice exposed to ultrafine particles than the control group. Additionally, mice exposed to ultrafine particles had a lower relative abundance of *Actinobacteria*, *Cyanobacteria*, and *Firmicutes* when compared to the control group. While numerous studies have examined the associations between exposure to PM\(_{10}\) or ultrafine PM and the gut microbiota in rodents, only one study has specifically focused on exposures from traffic sources. This study exposed 11 mice to benzo[a]pyrene (BaP) via oral gavage for 28 d. While BaP exposure did not impact alpha diversity, it did alter the relative abundance of 15 bacterial families, including an increase in the relative abundance of *Paraprevotella*, *Alcaligenaceae*, and *Bacteroides* as well as a decrease in the relative abundance of *Lactobacillus* and *Oscillospira* compared to baseline. BaP is a PAH that is commonly produced in automobile exhaust fumes and the burning of biomasses and is representative of an exposure that is common in traffic-related air pollution. Thus, results from this study suggest that exposure to components of traffic-related air pollution, in addition to PM, may alter the gut microbiota in mice.

### Summary of animal studies

Studies in rodents demonstrate that increased exposure to air pollutants, through inhalation or ingestion, have the potential to alter the gut microbiota, intestinal inflammation, and risk for type 2 diabetes and obesity. These animal studies demonstrate important exposure-induced alterations to the diversity and relative abundance of gut bacterial taxa. Despite the strengths of these experimental studies, they are limited by the use of 16S rRNA sequencing, which prevents examination of exposure-induced effects on gut bacterial species or bacterial function. Moving forward, the use of stool shotgun sequencing and fecal metabolomic profiling would allow for detailed

### Alterations to the gut microbiota may mediate the associations between air pollution, Obesity, and type 2 diabetes in animal models

In addition to gut microbial profiles, exposure to air pollutants was associated with several markers of inflammation as well as glucose intolerance in animal models. Several studies found that PM exposure was associated with increased pro-inflammatory cytokine expression in the murine intestine. All of these studies observed increased expression of tumor necrosis factor-alpha (TNF-α) in mice exposed to PM. The gut microbiota may also contribute to the regulation of circulating levels of TNF-α, and importantly TNF-α has been found to be associated with obesity and markers of insulin resistance and type 2 diabetes. These findings suggest a potentially novel gut microbial mediated link between air pollution and obesity and type 2 diabetes. Additionally, histological evidence of inflammation of the intestine was found in mice exposed to ultrafine particles and BaP. One study found that alterations to bacterial richness (Chao-1 estimator) mediated the observed associations between PM exposure and glucose intolerance. Results from these studies suggest that exposure to air pollutants may induce changes in the gut microbiota and increase systemic markers of inflammation that may contribute to the risk for type 2 diabetes.
characterization of the impact of air pollutants on gut bacterial composition (down to the species level) as well as elucidate the impact of air pollution exposure on gut microbial function. To date, only one study has included a mediation analysis and found that the gut microbiota mediated the observed associations between exposure to PM$_{2.5}$ and glucose intolerance. Including these analyses in future studies will help to determine if the gut microbiota mediates the associations between air pollution exposure and obesity or type 2 diabetes risk.

The use of murine models has allowed for experimental studies that are not feasible in humans. While these investigations are a useful tool for predicting the possible effects of environmental exposures on the human gut microbiota, there are limitations to using animal studies to infer the impact of air pollutants on the human gut. For example, only 15% of the genera of microbes in the distal gut of mice have been identified in the human gut. Moreover, there are differences in the anatomy of the human and murine GI tract as humans lack a forestomach and mice lack an appendix and have different distributions of goblet and Paneth cells. These differences in the composition of the gut microbiota and anatomy of the GI tract have the potential to impact microbe, host, and environment interactions. Given this, it is important to examine the associations between increased exposure to air pollutants and the gut microbiome in humans.

### Exposure to air pollutants is associated with the composition of the gut microbiota in humans

Three human studies have examined the associations between exposure to air pollutants and the gut microbiota, which are summarized in Table 2. Two of these were epidemiological studies that examined the associations between exposure to traffic-related air pollutants and PM$_{2.5}$ with the gut microbiota, respectively. The other was an experimental study that examined the effects of nanoparticles on the gut microbiota in a human model colon. As described below, these studies suggest that exposure to PM and nanoparticles may impact the composition of the gut microbiota as well as the intestinal immune response.

To our knowledge, associations between traffic-related air pollution exposure and the gut microbiota have only been examined in one study that was conducted in 43 overweight and obese adolescents (17–19 y of age) from Southern California. This study estimated individual exposure to traffic-related air pollution using residential addresses. Results from this study found that traffic-related air pollution exposure was positively correlated with fasting glucose.

| Table 2. Summary of studies that examined air pollutants and the gut microbiota in humans. |
|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
| Author | Study Population/Model Colon | Study Design | Key Findings |
|--------|-------------------------------|--------------|--------------|
| Alderete et al. | Adolescents and young adults from Southern California (n = 43) | Correlations between traffic-related air pollution exposure and gut microbial composition | ↑ Traffic-related air pollution exposure was correlated with ↓ Bacteroidaceae, ↑ Coriobacteriaceae, and ↑ fasting glucose levels |
| Liu et al. | Adults from 14 randomly selected districts in southern China (n = 5,828) | Associations between PM$_{2.5}$ and PM$_{1}$ exposure with gut dysbiosis and glucose metabolism | ↑ PM$_{2.5}$ and PM$_{1}$ exposure was associated with ↑ impaired fasting glucose and ↓ alpha diversity; Association between particulate matter exposure and impaired fasting glucose was partially mediated by gut microbiota diversity |
| Taylor et al. | Model of proximal colon developed from a human donor | Gut microbial characterization after zinc oxide (ZnO), titanium dioxide (TiO$_2$), and cerium dioxide (CeO$_2$) exposure | ↑ Nanoparticles resulted in ↑ hydrophobicity and ↓ pH; ↑ CeO$_2$ resulted in ↓ butyric acid production |

The table shows the three human studies included in this review. The table describes the study population or model colon along with the study design and key findings. These studies examined associations between exposure to air pollutants or nanoparticles with gut microbial diversity, the relative abundance of gut bacterial taxa, gut barrier integrity, and/or measures of glucose metabolism.
levels and gut microbial taxa that have been linked with obesity and insulin resistance, including Bacteroidaceae and Coriobacteriaceae.\(^{93,94}\) Additionally, the gut microbial taxa that were correlated with traffic-related air pollution exposure accounted for 24% and 29% of the correlation between exposure to air pollutants and fasting glucose levels.\(^{24}\) Similar results were found in a recent study that examined 5,828 adults from south China.\(^{25}\) This study used a spatiotemporal land-use regression model to estimate exposure to ambient air pollutants and found that exposure to PM\(_{2.5}\) and PM\(_{1}\) was associated with a decreased gut microbial alpha diversity. PM exposure was also negatively associated with the gut microbial taxa Firmicutes, Proteobacteria, and Verrucomicrobia and was associated positively and negatively with several taxa within the Bacteroidetes phyla. Additionally, impaired fasting glucose was found to be positively associated with PM\(_{2.5}\) and PM\(_{1}\) exposure and further analyses revealed that this association was partially mediated by gut microbial diversity.\(^{25}\) These findings suggest that air pollution exposure may increase susceptibility to obesity and type 2 diabetes risk through alterations in the composition of the gut microbiota.

Similar to larger PM, nanoparticles can be inhaled, ingested, and impact gut health and the gut microbiota. Nanoparticles are smaller than 0.1 µm in size, allowing for a greater surface area to volume ratio of the particle that contributes to increased reactivity.\(^{92}\) Only one study analyzed the impact of nanoparticles on the gut microbiota in humans.\(^{91}\) This study examined zinc oxide (ZnO), cerium dioxide (CeO\(_{2}\)), and titanium dioxide (TiO\(_{2}\)) nanoparticles, which are manmade and are present in air pollution.\(^{95-97}\) Additionally, TiO\(_{2}\) has been used as a photocatalyst to clean pollutants from the air via oxidation of organic compounds.\(^{98}\) In this study, each nanoparticle was introduced separately to a model colon from human donors at levels that are similar to human exposures. The model colon was comprised of a reactor, wherein a dialysis tube was encapsulated by a glass tube, which was maintained at conditions similar to the proximal colon (e.g., pH, temp). This reactor was inoculated with a microbial community obtained from human feces. These exposures altered SCFA production and resulted in a microbial community that was different from the non-exposed group.\(^{91}\) These results offer preliminary evidence that ingested nanoparticles that are found in air pollutants have the potential to modulate the human gut microbiota.

**Summary of human studies**

To date, only two human studies have examined the associations between exposure to air pollutants and the gut microbiota.\(^{24,25}\) Each of these studies suggest that air pollutants may increase the risk for obesity and type 2 diabetes via alterations to the gut microbiota. These studies considered potentially important confounders by adjusting for factors known to be associated with exposure to air pollutants, the gut microbiota, and risk for obesity and type 2 diabetes (e.g., diet, body fat percent). However, it is important to note that residual confounding may have still been present and could partially account for some of the observed associations in these studies. These investigations estimated human exposure to air pollutants based on residential addresses and may have also been limited by exposure misclassification, yet exposure misclassification should be random across participants (non-differential) and likely would bias their reported estimates toward the null.\(^{99}\) Lastly, one study found that the ingestion of nanoparticles alters gut bacterial composition and SCFA production in a human model colon.\(^{91}\) While these studies support epidemiological findings, further research is needed to determine the specific impact of air pollutants on the human gut microbiome, including the composition and functional potential of gut bacteria. For this reason, human studies should include personal exposure monitoring, detailed measures of possible confounders (e.g., diet, socioeconomic status), repeated fecal sampling, metabolomic profiling, and detailed shotgun sequencing samples to fully characterize the gut microbiome.

**Air pollutants and the gut microbiota: implications for obesity and type 2 diabetes**

As summarized in this review, early evidence from animal\(^{19-23,73}\) and human studies\(^{24,25,91}\) suggest that exposure to air pollutants may decrease gut bacterial
diversity and alter the relative abundance of gut bacterial taxa (e.g., Firmicutes), both of which have been linked with obesity and type 2 diabetes. While not true in all cohorts, most studies have shown that the composition of the gut microbiota is altered during obesity and type 2 diabetes where there is decreased microbial diversity, increased Firmicutes, and decreased Bacteroidetes. Further, mouse studies have shown that obesity and metabolic dysfunction are independently transmissible through fecal transplants.

In addition to altering the composition of the gut microbiota, increased exposure to air pollutants may also modify gut bacterial function, including the production of gut bacterial-derived metabolites involved in biological processes related to obesity and type 2 diabetes. Indeed, studies have shown that specific gut bacteria are involved in SCFA, lipid, amino acid, bile acid, and tryptophan metabolism, which have been linked with gut barrier integrity, satiety, body weight, adipose tissue inflammation, and type 2 diabetes. Bacteria in the gut also play an important role in xenobiotic metabolism of environmental toxicants. For example, the human colon microbiota has been shown to biotransform PAH to estrogenic metabolites, which has the potential to modulate pathways related to insulin resistance and obesity.

**Conclusions**

Animal and human studies provide evidence that exposure to air pollutants, either through inhalation or ingestion, contributes to alterations to the gut microbiota. This is supported by numerous experimental studies that have observed important shifts in the composition of the gut microbiota (i.e., diversity and relative abundance), decreased gut barrier integrity, and increased inflammation in the murine GI tract. Epidemiological studies have also found that exposure to air pollutants is associated with gut microbial diversity and the relative abundance of bacterial taxa. Additionally, the gut microbiome is known to influence numerous physiological processes related to the development of disease and specific gut microbial profiles have been observed in patients with obesity and type 2 diabetes. Together, results from these studies suggest that the associations between exposure to air pollutants and obesity and type 2 diabetes may be partially mediated by exposure-induced changes to the gut microbiota. A better understanding of these effects has the potential to reduce the global burden of disease through public health policies aimed at improving air quality. Additionally, interventions targeting the human gut microbiota (i.e., pre- and probiotic supplementation) have been shown to alter gut bacterial phyla that have also been linked with exposure to air pollutants (e.g., Firmicutes, Proteobacteria, Actinobacteria).

Despite recent advances in our understanding of the effects of air pollution exposure on the gut microbiota using animal models, few studies have examined the impact of inhaled pollutants on the gut microbiota in humans. Additionally, there are currently no longitudinal studies that have examined the chronic and dynamic impacts of air pollution on the gut microbiome, and their subsequent implications for obesity and type 2 diabetes risk over the life course. In order to understand the mechanisms linking air pollution exposure with alterations in gut bacteria, future studies should perform a detailed characterization of the gut microbiome using whole genome sequencing. Additionally, pairing genomic sequencing with fecal metabolomic profiling may allow for a more in-depth understanding of the impact of air pollutants on the composition and function of the gut microbiome. In summary, there is emerging evidence that links air pollution exposure with alterations in the gut microbiota that may have broad implications for human health. However, future studies are needed to fully characterize the effects of air pollution on the composition and function of gut bacteria.

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