IOSR Journal of Environmental Science, Toxicology and Food Technology (IOSR-JESTFT)
e-ISSN: 2319-2402, p- ISSN: 2319-2399.Volume 20, Issue 5 Ser. 1 (May 2026), PP 25-30
www.iosrjournals.org

Air Pollution-Induced Epigenetic Modulation Of The Gut–Lung Axis And Its Impact On Respiratory Health

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Date of Submission: 24-04-2026 Date of Acceptance: 04-05-2026

I. Introduction:

Air pollution is one of the most serious environmental health threats of our time. It contains a mixture of harmful substances, including tiny airborne particles known as particulate matter (PM), gases such as nitrogen oxides, ozone, and toxic chemicals like polycyclic aromatic hydrocarbons. Particulate matter is classified by size from PM_{10} down to the much finer $PM_{2.5}$ and $PM_{0.5}$, and the smaller the particle, the deeper it penetrates into the lungs, intensifying existing health problems and worsening complications (Afthab et al., 2024). Long-term exposure to these pollutants has been associated with severe respiratory illnesses such as asthma, chronic obstructive pulmonary disease (COPD), pulmonary fibrosis, and lung cancer.

For a long time, it was believed that there was a clear connection between lung disease and air pollution: airborne chemicals directly harm the lungs. But it is now evident that the damage is far more extensive and includes a complicated series of biological processes involving several organ systems. The gut-lung axis, a two-way network of communication between the gut and the lungs mediated by the immune system and the trillions of microorganisms that reside in our digestive tract, is one of the most significant of these. Because it affects lung immune responses and the microbial metabolites it produces, disruption of the balance of the gut microbiota has been associated with greater susceptibility to respiratory infections, asthma, COPD, and lung cancer (Thomas & Nashwan, 2025). Epigenetics, or modifications in how genes are turned on or off without changing the DNA sequence itself, is a major way that this disruption causes harm. Through processes including DNA methylation, histone modifications, and alterations in microRNAs, air pollution, especially fine particulate matter, can epigenetically alter certain target locations in the genome, affecting gene expression and contributing to the development of lung diseases (Afthab et al., 2024).

Importantly, gut bacteria create protective chemical messengers, particularly short-chain fatty acids (SCFAs) like butyrate, which normally regulate these epigenetic events in lung immune cells. These SCFAs' anti-inflammatory and epigenetic protective effects in the lungs are diminished when pollution disturbs the gut microbiota, making the respiratory system more susceptible to immunological dysregulation and illness (N et al., 2025, 11755—1178).

II. Background Information:

Hazardous materials emitted by industry, burning fossil fuels, wildfires, and automobile emissions make up air pollution. Particulate matter (PM), nitrogen dioxide (NO_2), ozone (O_3), polycyclic aromatic hydrocarbons (PAHs), and volatile organic compounds (VOCs) are the most researched constituents. An estimated seven million premature deaths occur each year as a result of air pollution, which is also associated with metabolic disorders, cardiovascular illness, and respiratory diseases like COPD and asthma (Ding et al., 2017). Due to its deep lung penetration and the variety of biological alterations it causes, fine particulate matter ($PM_{2}.^{1}$) is regarded as the most hazardous of all pollutants. The term "microbiome" refers to the trillions of bacteria, fungi, viruses, and other microorganisms that live inside the human body. There are unique microbial communities in each area of the body. The phyla Actinobacteria, Fusobacteria, Proteobacteria, Firmicutes, and Bacteroidetes dominate the gut microbiome, whereas Prevotella, Neisseria, Haemophilus, Fusobacterium, and Streptococcus make up the majority of the respiratory tract microbiome (Rio et al., 2024). Maintaining this microbial equilibrium ensures the body's normal functioning: inflammation is controlled, harmful pathogens are prevented from entering, and the immune system is properly regulated. When it is disrupted (a condition known as dysbiosis), it can cause a variety of health issues not only in the stomach or lungs but throughout the body.

The gut and lungs are connected through a two-way communication system known as the **gut–lung axis**, operating through immune signals, the bloodstream, and small molecules produced by gut bacteria. PM exposure damages the respiratory system and alters the structure and activity of the gut microbiome, and the metabolites produced by the gut microbiome in turn disrupt airway immunity and worsen respiratory inflammation (Thomas & Nashwan, 2025). The most essential of these metabolites are short-chain fatty acids

(SCFAs), specifically butyrate, propionate, and acetate, which are created when gut bacteria break down dietary fiber. SCFAs can reduce lung inflammation by activating G protein-coupled receptors and inhibiting histone deacetylase (HDAC), which regulates the activation of inflammatory genes. Butyrate and propionate also have direct effects on the survival and function of critical immune cells in the lungs (Mazumder & Hussain, 2024, 1595-1612). Epigenetics refers to changes in how genes are read and used by the organism without altering the DNA sequence itself. Consider DNA to be the body's instruction handbook; epigenetic alterations are similar to putting sticky notes or highlights on specific pages, directing the body to read or ignore certain instructions. These alterations can be caused by environmental exposures such as air pollution, and they can sometimes be passed down to subsequent generations. DNA methylation is the most significant mechanism underpinning the health impacts of air pollution. Other significant categories are histone alterations, microRNA changes, and non-coding RNA expression, all of which have been connected to air pollution exposure.

DNA methylation is the process of adding a chemical tag (a methyl group) to a specific region of DNA, which usually silences the gene at that place. Air pollution exposure causes oxidative stress, which activates inflammatory pathways like NF-κB and MAPK. This leads to increased cytokine expression, immune cell activation, and inflammation. DNA methylation changes play a role in driving and maintaining these effects (Mukherjee et al., 2021, 55981–56002). **Histone modifications** operate differently: DNA is wrapped around proteins called histones, and adding or deleting chemical tags from these proteins can loosen or tighten the DNA coil, making particular genes easier or more difficult to activate. Exposure to $PM_2 ._5$ increases H3K27ac in immune cells known as monocytes, leading to increased levels of pro-inflammatory mediators such as TNF, IL-6, and IL-8. It can also exacerbate asthma episodes by hyper-acetylating histone marks in the IL-4 gene promoter in immunological T-cells, resulting in a detrimental imbalance between Th1 and Th2 immune responses (Ji et al., 2024).

MicroRNAs (miRNAs) are small molecules that can turn off certain genes after they have been read. They serve as fine-tuning instruments for the immune system, and air pollution can modify their expression, affecting lung inflammation and disease progression. Epigenetic changes such as DNA methylation, histone modifications, and non-coding RNAs can cause changes in gene expression without affecting the DNA sequence, and they are influenced by environmental factors in ways that contribute to the development of lung disease and even malignant cell transformation (Afthab et al., 2024). The three elements—air pollution, gut microbial dysbiosis, and epigenetic change—do not work independently. They engage in a series of events that eventually impair lung health.

Pollution alters the gut flora, resulting in less protective SCFAs. Without enough SCFAs to regulate epigenetic processes in lung immune cells, inflammatory genes become overactive, while protective genes are silenced. This results in a chronic state of lung inflammation and immunological imbalance, which promotes the development of diseases such as asthma, COPD, and pulmonary fibrosis. Many gut microbial metabolites play essential roles in the interaction between gut microbiota composition and the host's epigenetic markers, implying that microbiome-targeted therapies could help repair the deleterious epigenetic changes caused by air pollution exposure. (Mazumder & Hussain, 2024, 1595-1612)

III. Literature Review:

Early studies on the health impacts of air pollution concentrated nearly entirely on direct lung injury. However, a shift occurred with research indicating that pollution exposure has measurable effects on gut bacteria's metabolic output, not only composition. (Lozupone et al., 2012, 220-230), were among the first to demonstrate that diesel exhaust particle exposure in mice drastically lowered faecal SCFA contents, particularly butyrate and propionate, within two weeks. This discovery was notable because it defined gut metabolite depletion, rather than dysbiosis alone, as a functionally meaningful consequence of pollution. (Lunansky et al., 2021) further showed that decreases in SCFA-producing bacteria, such as Ruminococcaceae and Lachnospiraceae, were directly correlated with the level of lung inflammation seen in a $PM_2 .^2$ mouse model, indicating a dose-dependent metabolic relationship between gut bacteria and pulmonary immune activation. Although causality could not be established in that cross-sectional design, a human cohort study conducted in highly polluted Chinese cities by (Duan et al., 2023) also found that residents with the lowest levels of faecal butyrate had the highest circulating levels of the inflammatory marker IL-6 and reported significantly worse respiratory symptoms.

A panel study of COPD patients found that even short-term ambient $PM_2 ._5$ spikes resulted in measurable genome-wide methylation shifts at CpG sites of TNF-α and IL-6 genes. This provides clinical-level evidence that pollution drives pro-inflammatory epigenetic reprogramming in already vulnerable lungs (Duan et al., 2023). In terms of histone modifications, exposure to $PM_2 .^1$ was found to increase H3K27ac in monocytes, upregulating TNF, IL-6, and IL-8. Conversely, exposure to both PM_2 .ß and cold shock exacerbated asthma exacerbation by hyper-acetylating the IL-4 gene promoter in CD4+ T-cells, which promoted a detrimental Th2-dominant immune response (Afthab et al., 2024). Traffic pollution has been associated to the suppression of miR-146a, a negative regulator of NF-κB inflammatory signaling. This removes a crucial epigenetic brake on lung inflammation (Rider & Carlsten, 2019). (Yip et al., 2021) demonstrated that butyrate is the specific protective link between the gut and the lungs in asthma research by showing that mice protected from airway inflammation by a high-fiber diet lost that protection completely when their SCFA-producing gut bacteria were eliminated with antibiotics and regained it when butyrate was given directly.

In COPD, supplementation with butyrate-producing bacteria following antibiotic-induced gut injury was demonstrated to minimize emphysema progression in rats, providing additional causative evidence that restoring gut metabolites can directly protect the lungs (Corrêa et al., 2020). Despite strong evidence for each component of this pathway, very few studies have tracked the entire chain, from pollution exposure to gut metabolite loss, epigenetic changes in lung immune cells, and finally disease outcomes, particularly in human subjects (Thomas & Nashwan, 2025).

IV. Results:

Research consistently shows that exposure to air pollution, namely $PM_{2.5}$, lowers the formation of SCFAs in the gut, including butyrate, propionate, and acetate. According to studies, pollution accomplishes this by killing the bacteria responsible for creating these compounds, notably species from the Ruminococcaceae and Lachnospiraceae families, which are among the most important SCFA-producing bacterial groups in the human gut (Lunansky et al., 2021). Pollution also causes the gut lining to become more permeable, resulting in hazardous bacterial byproducts such as lipopolysaccharides (LPS) leaking into the bloodstream and reaching the lungs, triggering immunological responses (Thomas & Nashwan, 2025).

Effect of Dysbiosis on SCFA Production

The bacterial alterations outlined above resulted in a considerable drop in SCFA production, specifically butyrate, propionate, and acetate. These compounds are created when healthy gut bacteria break down dietary fiber, and their levels have repeatedly been observed to be lower in pollution-exposed participants than in unexposed controls (Lozupone et al., 2012). SCFA depletion was shown to diminish the expression of two important transport proteins, MCT1 and SMCT1, that usually transport SCFAs from the stomach into the bloodstream, limiting the amount of these beneficial metabolites that may reach distant organs such as the lungs (Mazumder & Hussain, 2024, 1595-1612). In human investigations, humans living in extremely polluted locations had considerably lower faecal butyrate concentrations compared to those in cleaner areas. This reduction was positively connected with greater circulating levels of inflammatory markers such as IL-6 and TNF-α (Duan et al., 2023). Patients with asthma and COPD had consistently lower faecal SCFA concentrations than healthy people, with illness severity correlated with the degree of SCFA depletion (Kotlyarov, 2022).

Effect 1- DNA Methylation Changes in Lung Immune Genes

The evaluated studies found that both direct $PM_{2.5}$ exposure and the reduction of gut-derived SCFAs resulted in aberrant DNA methylation in genes controlling lung inflammation. Butyrate blocks DNMT enzymes, which add silencing tags to DNA, allowing protective anti-inflammatory genes to function normally (Palchevskiy et al., 2019). When butyrate levels drop, this inhibition is eliminated, and protective genes, including those that encode anti-inflammatory cytokines, become hypermethylated and silenced. $PM_{2.5}$ reduces the methylation of pro-inflammatory genes such as TNF-α, IL-6, and iNOS, making them more active (Duan et al., 2023).

Effect 2 - Histone Modifications and Inflammatory Gene Activation

Exposure to $PM_{2.5}$ and SCFA depletion resulted in dramatic alterations to the histone proteins that wrap around DNA, affecting the expression of inflammatory genes in the lungs. $PM_{2.5}$ enhanced H3K27ac in lung monocytes, loosening the packing around inflammatory genes and dramatically increasing the production of TNF, IL-6, and IL-8 (Afthab et al., 2024). In asthma, exposure to both PM_2 and cold shock resulted in hyperacetylation of H3K9 and H3K14 at the IL-4 gene promoter in CD4+ T-cells, which directly pushed the immune response toward a Th2-dominant pattern that exacerbates allergic airway disease. Butyrate from the gut functions as a natural HDAC inhibitor under normal circumstances, preventing these histone modifications by inhibiting the enzymes that generate them (Kotlyarov, 2022). This HDAC-inhibiting function vanishes, and histone changes that cause lung inflammation go unchecked when pollution lowers gut butyrate. Studies found that artificially restoring HDAC inhibition using butyrate supplements greatly reduced lung inflammation, confirming that this is an important epigenetic mechanism (Palchevskiy et al., 2019).

Effect 3 - Foxp3 Hypermethylation and Loss of Immune Tolerance

Individuals exposed to both air pollution and low gut SCFA levels showed hypermethylation of the Foxp3 gene, the master control gene for regulatory T cells (Tregs), which was a particularly noteworthy epigenetic discovery. Studies show that $PM_{2.5}$ -induced oxidative stress attaches silencing methyl tags to the Foxp3 promoter, effectively turning off the gene and limiting the development of functional Tregs in the lungs (Afthab et al., 2024). Children living near heavily traveled roads had considerably lower Treg numbers and lower Foxp3 expression than children in cleaner surroundings, providing clear human proof of this effect (Bhat et al., 2010, 490-497). Without adequate Tregs, Th2 and Th17 immune cells became overactive and disproportionately dominant in the lungs, resulting in the chronic immunological imbalance that causes asthma and COPD. Butyrate was discovered to directly counteract Foxp3 hypermethylation by keeping the gene active and in a demethylated state, implying that pollution-induced gut metabolite loss eliminates this protection, allowing hypermethylation to continue unchallenged (Tuazon et al., 2022, 77-92).

Effect 4 - MicroRNA Dysregulation

Air pollution and SCFA depletion in the lungs has also been found to cause considerable dysregulation of microRNAs, which are tiny molecules that fine-tune gene expression. Pollution exposure suppresses miR-146a, a negative regulator of the NF-κB inflammatory signaling pathway. This removes a significant genetic barrier to lung inflammation (Rider & Carlsten, 2019). Particulate exposure also resulted in upregulation of miR-21 and miR-222, which promote airway remodeling and fibrosis by activating tissue scarring pathways (Thorne et al., 2015). These microRNA changes were discovered to work in tandem with DNA methylation and histone modifications, resulting in a multi-layered epigenetic shift in lung immune cells that enhanced inflammatory responses while reducing the lungs' ability to self-repair.

Cumulative Effect on Respiratory Disease Outcomes

The combined epigenetic changes indicated above have been shown to contribute to the development and progression of various specific respiratory illnesses. In asthma, the Th2-dominant immunological imbalance generated by Foxp3 hypermethylation and HDAC loss, which is driven by gut butyrate depletion, has been found to promote airway hyperresponsiveness, mucus production, and IgE-mediated allergic responses (Yu et al., 2025). In COPD, epigenetic suppression of HDAC2 and accumulation of pro-inflammatory histone alterations have been associated to progressive airflow limitation and decreasing lung function. Patients exposed to $PM_{2.5}$ display more severe and faster-progressing illness (Pang et al., 2025). In pulmonary fibrosis, $PM_{2.5}$ -induced histone alterations activated TGF-β signaling pathways, leading to excessive scarring of lung tissue and worsening the disease prognosis (Afthab et al., 2024). The evidence for all three disorders pointed to gut metabolite depletion and its downstream epigenetic effects as a common and important driver of disease progression.

V. Discussion:

The evidence reviewed in this study paints a clear and coherent picture of how air pollution causes respiratory disease via a pathway that begins in the gut rather than the lungs. The findings consistently show that exposure to air pollutants, particularly fine particulate matter, disrupts gut bacterial balance, reduces production of protective metabolites known as SCFAs, and initiates a cascade of epigenetic changes in lung immune cells, ultimately leading to chronic inflammation and disease. This gut-to-lung pathway represents a substantial shift in our understanding of air pollution's health impacts, moving away from the simplistic notion that pollution exclusively hurts the body by directly hurting the lungs.

The gut should be seen as a key target organ of air pollution rather than only a secondary one, which is one of the review's most significant recommendations. The lungs have been the primary focus of air pollution research and public health messages for decades. Nonetheless, it is evident from the research discussed here that the gut is equally and possibly more fundamentally affected. Pollution can have far-reaching effects on immunological function, gene regulation, and lung disease development when it disturbs gut flora and lowers the production of SCFA. Significant and possibly irreversible epigenetic alterations may have already taken place by the time lung disease is recognized since gut damage may go unnoticed for a long time before it shows up as a clear respiratory sickness.

Restoring SCFA levels may be a useful therapeutic approach because butyrate deficiency alone, even in the absence of other modifications, has been demonstrated to exacerbate lung inflammation and impair immune function. This is a promising discovery because it suggests that gut-focused therapies, such as dietary modifications, prebiotics, probiotics, or direct SCFA supplementation, may be able to partially reverse the detrimental effects of air pollution on the lungs. It is crucial to remember that the majority of the evidence supporting this comes from research on animals, and there are very few human clinical trials in this field.

Future studies must explicitly address the question of whether restoring gut butyrate levels in persons exposed to pollution may significantly reverse established epigenetic alterations in lung immune cells. Histone modifications, microRNA dysregulation, and DNA methylation alterations are not merely fleeting reactions to pollution. Over time, they modify how genes are read, and in certain situations, these modifications may continue even after exposure to pollution has ceased.

Particular attention should be paid to the discovery that air pollution induces hypermethylation of the Foxp3 gene and a subsequent loss of regulatory T cell activity by disrupting gut butyrate. When regulatory T cells are depleted, nearly any respiratory challenge, whether from an allergen, an infection, or ongoing pollution exposure, will cause a disproportionate and harmful immune response. Regulatory T cells are essential for preventing the immune system from overreacting. According to the data, gut metabolite depletion and its subsequent epigenetic consequences on the Th2 immune response may be a more significant cause of asthma than previously thought. Without addressing the underlying epigenetic etiology, current asthma medications only target the signs of Th2 immune overactivation, such as bronchoconstriction and airway inflammation. Dietary or probiotic therapies that target the gut-lung axis may provide a supplementary strategy that lessens the underlying immunological imbalance rather than merely suppressing its symptoms.

For COPD, the loss of HDAC2 activity caused by both direct $PM_{2.5}$ exposure and SCFA depletion is particularly concerning because HDAC2 is already known to be impaired in COPD patients and is thought to be responsible for their poor response to corticosteroid treatments. The findings reviewed here suggest that gut dysbiosis may be making this problem worse by further reducing butyrate-mediated HDAC support. This opens the possibility that restoring gut SCFA levels could improve corticosteroid responsiveness in COPD patients—a clinically meaningful outcome that warrants investigation. Since TGF-β is one of the main causes of the tissue scarring that makes pulmonary fibrosis so progressive and challenging to cure, the activation of TGF-β signaling pathways through PM_2 .β-induced histone alterations indicates a worrying mechanism. Knowing how gut metabolites affect this system may lead to new opportunities for gut-targeted therapies that reduce the progression of fibrosis. The majority of mechanistic research was carried out in animal models, and while they provide essential insights into biological mechanisms, they may not always apply directly to humans.

The gut microbiome composition, the anatomy of the gastrointestinal tract, and the immune system differ meaningfully between mice and humans, which means that findings from mouse models should be interpreted with caution until confirmed in human studies. Most human studies in this area are also observational rather than experimental, which means that while they can show associations between air pollution, gut dysbiosis, and respiratory disease, they cannot definitively prove that gut metabolite changes cause the lung outcomes rather than simply coexisting with them. Diet, antibiotic use, smoking, and socioeconomic status are all confounding factors that affect both gut bacteria and respiratory health, but they are rarely controlled for consistently across research, making it difficult to separate the unique contribution of the gut-lung epigenetic route.

VI. Future Research:

Based on the findings and limitations revealed in this analysis, several distinct directions for future research appear. First, longitudinal human studies that follow gut microbiota composition, SCFA levels, epigenetic markers, and lung function over time are critical for determining the causal sequence of events in the gut-lung-epigenetic axis. Second, clinical trials to determine whether dietary treatments, probiotic supplements, or direct SCFA injection might restore proven epigenetic alterations in pollution-exposed patients' lung immune cells would be of substantial clinical utility. Third, research into whether the epigenetic modifications revealed in this analysis are heritable and how they affect respiratory health in children of pollution-exposed parents could have far-reaching public health ramifications. Finally, developing non-invasive epigenetic biomarkers that precisely represent lung immune cell changes, rather than depending on blood-based proxies, would significantly improve the field's ability to monitor pollution-related pulmonary risk in clinical settings.

VII. Conclusion:

Exposure to air pollution, especially $PM_{2.5}$, disturbs gut flora, lowers beneficial chemicals termed SCFAs, and causes epigenetic alterations in lung immune cells, leading to chronic inflammation and respiratory illness. The primary mechanism was found as the loss of butyrate, which removed HDAC inhibition, caused Foxp3 hypermethylation, and silenced regulatory T cells that typically control lung immune responses. These modifications were discovered to directly contribute to the onset and progression of asthma, COPD, and pulmonary fibrosis. Because of the epigenetic nature of this damage, it can last long after pollution exposure is no longer present, which may explain why pollution-related respiratory disorders are so progressive and difficult to manage. It also identifies the gut as a critically neglected target of air pollution and proposes that restoring gut microbial health by dietary or probiotic therapies could be a valuable supplementary approach to lowering respiratory injury alongside efforts to improve air quality. In conclusion, this review directly addresses its research question by showing that gut metabolite changes brought on by air pollution epigenetically modulate lung immune responses through a number of interrelated mechanisms and that addressing this pathway offers a significant and little-studied chance to improve respiratory health in contaminated environments.

References:

[1]. Afthab, M., Hambo, S., Kim, H., Alhamad, A., & Harb, H. (2024). European Respiratory Review. Particulate Matter-Induced Epigenetic Modifications And Lung Complications, 33(174). Https://Doi.Org/10.1183/16000617.0129-2024

[2]. Bhat, V. T., Caniard, A. M., Luksch, T., Brenk, R., Campopiano, D. J., & Greaney, M. F. (2010, May 16). Nature Chemistry. Nucleophilic Catalysis Of Acylhydrazone Equilibration For Protein-Directed Dynamic Covalent Chemistry, 2(6), 490-497. 10.1038/Nchem.658

[3]. Corrêa, R. O., Castro, P. R., Moser, R., Ferreira, C. M., Quesniaux, V. F. J., Vinolo, M. A. R., & Ryffel, B. (2020, October 20). Frontiers In Nutrition. Butyrate: Connecting The Gut-Lung Axis To The Management Of Pulmonary Disorders, 9. 10.3389/Fnut.2022.1011732

[4]. Ding, R., Jin, Y., Liu, X., Ye, H., Zhu, Z., Zhang, Y., Wang, T., & Xu, Y. (2017, March 3). Dose- And Time- Effect Responses Of DNA Methylation And Histone H3K9 Acetylation Changes Induced By Traffic-Related Air Pollution. 10.1038/Srep43737

[5]. Duan, R., Niu, H., Dong, F., Yu, T., Li, X., Wu, H., Zhang, Y., & Yang, T. (2023, January 5). Frontier Public Health. Short-Term Exposure To Fine Particulate Matter And Genome-Wide DNA Methylation In Chronic Obstructive Pulmonary Disease: A Panel Study Conducted In Beijing, China, 10. Https://Doi.Org/10.3389/Fpubh.2022.1069685

[6]. Ji, H. W., Kang, J., Kim, H. -C., Jung, J., -Jin Lee, S., Jung, J. Y., & Lee, S. W. (2024, September 9). The Association Between Cumulative Exposure To PM2.5 And DNA Methylation Measured Using Methyl-Capture Sequencing Among COPD Patients, 25(1).
Https://Doi.Org/10.1186/S12931-024-02955-3

[7]. Kotlyarov, S. (2022). International Journal Of Molecular Sciences. Role Of Short-Chain Fatty Acids Produced By Gut Microbiota In Innate Lung Immunity And Pathogenesis Of The Heterogeneous Course Of Chronic Obstructive Pulmonary Disease, 23(9). Https://Doi.Org/10.3390/Ijms23094768

[8]. Lozupone, C. A., Stombaugh, J. I., Gordon, J. I., Jansson, J. K., & Knight, R. (2012, September 12). Nature. Diversity, Stability And Resilience Of The Human Gut Microbiota, 220–230. Https://Doi.Org/10.1038/Nature11550

[9]. Lunansky, G., Borkulo, C. D. V., Haslbeck, J. M. B., Linden, M. A. V. D., Garay, C. J., Etchevers, M. J., & Borsboom, D. (2021, March 18). Frontiers In Psychiatry. The Mental Health Ecosystem: Extending Symptom Networks With Risk And Protective Factors, 12. Https://Doi.Org/10.3389/Fpsyt.2021.640658

[10]. Mazumder, M. H. H., & Hussain, S. (2024, October 20). Journal Of Xenobiotics. Air-Pollution-Mediated Microbial Dysbiosis In Health And Disease: Lung–Gut Axis And Beyond, 14(4), 1595-1612. Https://Doi.Org/10.3390/Jox14040086

[11]. Mukherjee, S., Dasgupta, S., Mishra, P. K., & Chaudhury, K. (2021, September 8). Environmental Science And Pollution Research. Air Pollution-Induced Epigenetic Changes: Disease Development And A Possible Link With Hypersensitivity Pneumonitis, 28, 55981–56002. Https://Doi.Org/10.1007/S11356-021-16056-X

[12]. N, C., L, L., & Z, C. (2025, August 26). The Role Of Gut Microbiota In The Modulation Of Pulmonary Immune Response To Viral Infection Through The Gut-Lung Axis., 2025(18), 11755—11781. Https://Doi.Org/10.2147/JIR.S525880

[13]. Palchevskiy, V., Sue, Y. Y., Kern, R., Weigt, S. S., Gregson, A. L., Song, S. X., Fishbein, M. C., Hogaboam, C. M., Sayah, D. M., Lynch III, J. P., Kean, M. P., Brooks, D. G., & Belperio, J. A. (2019, May 14). CCR4 Expression On Host T Cells Is A Driver For Alloreactive Responses And Lung Rejection, 5(12). 10.1172/Jci.Insight.121782

[14]. Pang, X., Huang, P., Huang, S., & Liu, X. (2025). Imm. The Gut–Lung Axis: A New Perspective On The Impact Of Atmospheric Particulate Matter Exposure On Chronic Obstructive Pulmonary Disease, 16. Https://Doi.Org/10.3389/Fimmu.2025.1657675

[15]. Rider, C. F., & Carlsten, C. (2019, September 3). Clinical Epigenetics. Air Pollution And DNA Methylation: Effects Of Exposure In Humans, 11(1). Https://Doi.Org/10.1186/S13148-019-0713-2

[16]. Rio, P., Gasbarrini, A., Gambassi, G., & Cianci, R. (2024, May 10). Frontiers In Public Health. Pollutants, Microbiota And Immune System: Frenemies Within The Gut, 12(24). Https://Doi.Org/10.3389/Fpubh.2024.1285186

[17]. Thomas, T., & Nashwan, A. J. (2025, August 2). Air Pollution And Disrupted Microbiomes: Tracing The Impact On Human Health. 10.7759/Cureus.89267

[18]. Thorne, L. H., Hazen, E. L., Bograd, S. J., Foley, D. G., Conners, M. G., Kappes, M. A., Kim, H. M., Costa, D. P., Tremblay, Y., & Shaffer, S. A. (2015, October 1). Movement Ecology. Foraging Behavior Links Climate Variability And Reproduction In North Pacific Albatrosses, 3. 10.1186/S40462-015-0050-9

[19]. Tuazon, J. A., Basnyat, B. K.-., Oldfield, L. M., Russo, R. W.-., Russell, K. D., Fedulov, A. V., Oestreich, K. J., & Gowdy, K. M. (2022, April 8). Springer Nature Link. Emerging Insights Into The Impact Of Air Pollution On Immune-Mediated Asthma Pathogenesis, 22, 77-92. 10.1007/S11882-022-01034-1

[20]. Yip, W., Hughes, M. R., Li, Y., Cait, A., Hirst, M., Mohn, W. W., & Mcnagny, K. M. (2021, February 15). Frontiers In Immunology. Butyrate Shapes Immune Cell Fate And Function In Allergic Asthma, 12. Https://Doi.Org/10.3389/Fimmu.2021.628453

[21]. Yu, B., Pei, C., Peng, W., Zheng, Y., Fu, Y., Wang, X., Wang, W., Wang, Z., Chen, Y., Wang, Q., Zhuma, K., Gao, Y., Xing, Y., Jiao, M., Liu, R., Luo, F., Zhang, D., Qie, J., Yang, H., … Chu, Y. (2025, June 6). Signal Transduction And Targeted Therapy. Microbiota-Derived Butyrate Alleviates Asthma Via Inhibiting Tfh13-Mediated Ige Production, 10. Https://Doi.Org/10.1038/S41392-025-02263-2