ISSN: 2457-0400

Impact Factor: 7.675

WORLD JOURNAL OF ADVANCE HEALTHCARE RESEARCH

www.wjahr.com

Volume: 10, Issue: 2

Page N. 231-237

Year: 2026

Review Article

Coden USA: WJAMA3

INTERKINGDOM CROSSTALK BETWEEN BACTERIAL COMMUNITIES AND VIRAL PATHOGENS: MICROBIOME-DRIVEN MODULATION OF VIRAL ENTRY, REPLICATION, AND IMMUNE ESCAPE

Shahad Kh.Al-Qaisi[1*], Reham Saad Kadhim[2], Ruqaya Bashar Ismail[3]

[1]Diyala University, College of Science, Biotechnology Department, Diyala, Iraq.
[2]Diyala University, College of Medicine, Department of Anatomy.
[3]Ibn Sina University For Medical And Pharmaceutical Sciences, College of Medicine, Department of Anatomy.

Article Received: 26 December 2025 **Article Revised:** 15 January 2026 **Article Published:** 1 February 2026

***Corresponding Author: Shahad Kh. Al-Qaisi**

Diyala University, College of Science, Biotechnology Department, Diyala, Iraq.

DOI: https://doi.org/10.5281/zenodo.18516135

How to cite this Article: Shahad Kh.Al-Qaisi[1*], Reham Saad Kadhim[2], Ruqaya Bashar Ismail[3] (2026). Interkingdom Crosstalk Between Bacterial Communities And Viral Pathogens: Microbiome-Driven Modulation Of Viral Entry, Replication, And Immune Escape. World Journal of Advance Healthcare Research, 10(2), 231–237.

ABSTRACT

Human microbiome has emerged to be a determinant of host vulnerability to viral infections in addition to its traditional functions of promoting physiological homeostasis. An increasing body of evidence indicates that there is a complex interkingdom crosstalk that occurs between bacterial groups and viral pathogens, which proves that microbiome controls the entry of viruses, virus replication, and immune evasion. The bacterial populations also influence the dynamic of viral infections, by direct interaction with the viral particles, expression control of host receptors and intracellular environments control that either stimulate or suppress viral replication. Besides these direct effects, the microbiome forms the core of the development of innate and adaptive immune responses. Depending on the production of microbiome-generated metabolites and signaling pathways by the microbiome, the differentiation of immune cells, cytokine generation, and antiviral defense mechanisms are controlled and therefore influence viral clearance and persistence. Microbiome has been observed to dictate the severity of the disease and clinical outcome because dysbiosis, an imbalance of microbes and reduced functional diversity has been associated with imperfect immune regulation and hosts viral infection. This review provides a summary of microbiome processes in detail, which control viral pathogenesis, and, in particular, bacterial community-virus interactions and immune control. We discuss the evidence which is beginning to emerge on the relationship between microbiome composition and viral immune escape systems and speculate on the clinical potential of microbiome-based interventions. It has provided a new perspective on the treatment of viral diseases as well as enabled new insights into the management of bacterial communities and interkingdom crosstalk between bacterial communities and viral pathogens which makes the microbiome a promising antiviral therapeutic platform in the future.

KEYWORDS: Besides These Direct Effects, The Microbiome Forms The Core Of The Development Of Innate And Adaptive Immune Responses.

INTRODUCTION

The human body is a dynamic and sophisticated microbial community, in which physiological homeostasis and host immunity formation cannot take place without the help of microbes. As the group of bacteria, they occur on many areas of the body, including the gut, respiratory system, skin, and the urogenital. High-throughput sequencing and metagenomic technologies have transformed microbiome studies over the past decade demonstrating that it is a major contributor to host health and disease predisposition.[1,2]

Traditionally, the viral pathogenesis study has largely been conducted as an independent study with an oriented interest in the viral cell interactions and the host immune responses. However, there is new evidence today, which identifies the microbiome as a significant mediator that can significantly control viral infections. It is also

becoming actively known that bacterial communities are also involved in contributing towards the entry, replication, persistence, and transmission of the virus. This interkingdom cross talk between bacteria and viruses is a complex biological network, in which metabolites of microbes, surface molecules and immune-modulating signals influence the behavior of the virus and its disease development.[3,4]

It has been demonstrated in a number of studies that commensal and pathogenic bacteria can interact directly with the viral particles either facilitating or inhibiting viral attachment and entry into host cells. In addition, the microbiome can indirectly regulate viral replication through the regulation of the host cellular environment, including nutrient availability, receptor expression and intracellular signaling pathways. Besides the effects, bacterial communities play a central role in shaping the innate and adaptive immune response, and consequently, in influencing the viral immune avoiding mechanisms and chronic infection establishment. Knowledge of microbiome-based control of viral diseases is especially applicable to the emergent and re-emerging viral diseases. The composition and the functional state of the host microbiome have been found to affect respiratory viruses, enteric viruses, and persistent viral pathogens. Dysbiosis that is either brought by antibiotics, diet, or underlying disease is thus possible to predispose individuals to higher viral vulnerability or worse clinical outcomes.[5,6]

This review will seek to give an overall review of interkingdom crosstalk between bacterial communities and viral pathogens. We pay attention to the mechanisms of the microbiomes to regulate viral entry, replication, and immune escape and comment on the clinical implications of such interactions. Through the incorporation of the existing experimental and clinical data, the present review provides an understanding of the microbiome as a primary factor in determining viral pathogenesis and suggests that the microbiome is a promising antiviral therapy target.[7]

Bacterial Communities and the Host Microbiome

The most functionally important and the most common part of human microbiome is represented by bacterial communities that constitute a highly complex ecosystem which is interwoven with host physiology and immune regulation. These communities settle on various body locations, among them: the gastrointestinal tract, respiratory system, skin and urogenital tract whereby they form niche-specific relationships with the host tissues (Figure 1). It is in the gut microbiome, especially, that the greatest bacteria density and diversity are found, and the epicenter of host-microbe and microbe-microbe interactions are happening.[8,9]

The genome of bacterial communities, commonly known as the microbiome gene pool, is far more extensive than the coding ability of the human genome. This increased genetic repertoire allows bacterial populations to execute metabolic and regulatory processes which cannot be accomplished by the host alone. The bacterial communities have been demonstrated to affect the production of bioactive metabolites such as short-chain fatty acids by fermentation of dietary substances and effects of bacterial communities on the ratios between epithelial barriers, energy homeostasis, and immune cell differentiation. These abilities do not restrict their effects into local settings but instead they have systemic effects where they influence host vulnerability to infectious diseases including viral infections.[10,11]

The communities of bacteria play a significant role in the training and control of the adaptive and innate immunity. Commensal bacteria suppress immune cell development, regulate inflammatory responses pathways, and non-pathogenic antigen immune tolerance. This immunomodulatory capacity causes the microbiome to be a critical component of an antiviral immune response. Microbial quality changes can lead to a disturbance of immune homeostasis, with the state of too much or no antiviral defences.[12]

It is important to note that the microbiome is a dynamic organism and it is very sensitive to any environmental changes, e.g. antibiotic exposure, dieting change, infection and lifestyle changes. Such perturbations can cause the dysbiosis, i.e. reduced variety of microbes and functional disequilibrium. The dysbiosis has been associated with increased vulnerability to viral pathogens since it is capable of penetrating mucosal surfaces, is able to alter the expression of host receptors and is able to alter intracellular signaling pathways that govern viral entry and replication.[13,14]

Bacterial communities do not passively reside in the host environment, but actively contribute to the formation of the host environment in which viral pathogens are operating. Their metabolic activities combined with their spatial arrangement and immune-modulating properties lead to the formation of the conditions that can either restrict or help in the viral infection.[15]

The composition and dynamics of bacterial communities in the host microbiome is thus imperative to the clarification of the processes by which interkingdom crosstalk exists between bacteria and viruses. This theoretical framework forms the foundations of examining the effect of microbiome-based mechanisms on viral entry, replication, and immune escape as covered in further sections of this review.[16]

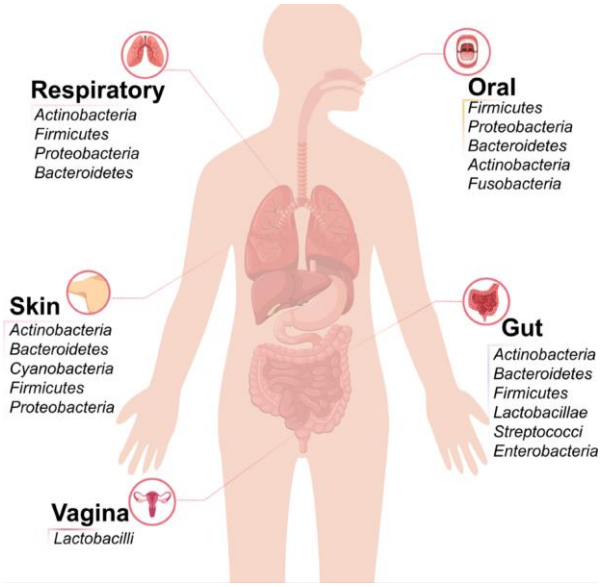

Fig 1: Overview of bacterial communities within the human microbiome and their roles in host physiological and immune regulation.

Microbiome–Virus Interactions: Modulation of Viral Entry and Replication

There is increasing evidence that bacterial communities in the host microbiome have a determinative role in determining dynamics of viral infection directly and indirectly affecting viral entry and replication. These are some of the important interactions of interkingdom crosstalk, where bacterial components, bacterial metabolites, and host responses mediated by the microbiome all contribute to measuring viral infectivity and tissue tropism.[17,18]

A highly direct way that bacteria can alter viral entry is by direct interaction of bacterial surface molecules and viral particles. Viral proteins may be bound by certain bacterial lipopolysaccharides, peptidoglycans and capsular polysaccharides and this is a stabilizing force on viral particles that increases their ability to attach to host cell receptors. It has been well observed in a number of enteric viruses whereby binding to bacteria enhances viral persistence in the extracellular space and facilitates efficient entry of the virus into host cells. On the other hand, there are bacteria that are able to trap viruses or disrupt receptor binding and thus hamper viral access to target cells.[19]

In addition to direct interactions, viral entry can be affected by bacterial communities by altering the expression of host receptors. Signals produced by the microbial flora are capable of controlling the expression of viral entry receptors and co-receptors on epithelial and immune cells and modulate host vulnerability to infection. Alterations in microbial composition, especially in the case of dysbiotic conditions, could, therefore, enhance accessibility of viral entry points that then promote the process of infection and spread of viruses.[20]

The microbiome has an enormous influence on the viral replication, as well, as it modulates the intracellular environment of the host cells. Short-chain fatty acids, secondary bile acids and indole derivatives are the bacterial metabolites that control cellular metabolic pathways, redox balance and signaling cascades which are exploited by viruses to replicate. They can either promote or inhibit viral replication, by providing a friendly metabolic environment, or by triggering antiviral signaling pathways, such as interferon-mediated responses.[21]

Moreover, the viral replication is also indirectly controlled by the bacterial communities as they influence the host immune activation. Balanced microbiome aids in the maintenance of basal immune surveillance and quick antiviral responses and dysbiosis may provoke a defect on innate immune signaling and permit viruses to replicate more effectively in the early phases of infection. The described immune modulation indicates that the microbiome acts as a gatekeeper by which the exposure to a virus leads to an effective infection or successful containment.[22,23]

Altogether, the interactions of microbiomes and viruses are a complicated system of physical, molecular, and immunological interactions that regulate the process of viral accessibility and multiplication. Knowledge of these processes is critical to the explanation of the role bacterial communities play in viral pathogenesis and preconditions the study of microbiome-mediated immune modulation and virus immune escape in the next section.[24]

Microbiome-Mediated Modulation of Host Immune Responses and Viral Immune Escape

Host immune system is the interface between the microbial communities and the invading viral pathogens, so it is a major mediator of interkingdom crosstalk. The microbiome communities of bacteria are central to the formation of innate and adaptive immune responses, which therefore affect viral clearance, viral persistence, and immune escape mechanisms. Instead of being protective, the microbiome can have either protective or permissive roles with regard to viral infections, depending on its makeup and its wellbeing.[25,26]

The Commensal bacteria play a role in basal immune priming through the regulation of pattern recognition receptor signaling, cytokines production, and immune cell maturation. The microbiome, through sustained low-level stimulation, keeps the immune in a state of readiness, which allows the microorganisms to respond with rapid anti-viral reactions to viral invasion. Such an immune disposition is required to sense interferon and prematurely limit viral replication. Conversely, these signaling pathways can be hampered by dysbiosis leading to the development of delayed or inadequate antiviral immunity which permits the viruses to invade.

Microbiome-generated metabolites are the major regulators of immunity.[27]

The derivatives of the bile acids, the short-chain fatty acids, and tryptophan metabolites affect the differentiation and activity of immune cell subsets, the regulatory T cells, macrophages, and dendritic cells. Most of these effects increase antiviral defenses, whereas others increase immune tolerance and anti-inflammatory conditions that viruses can use to escape the immune system. Unremitting viruses, especially, can enjoy microbiome-mediated immunosuppression, which inhibits viral clearance.[28]

Microbiome-mediated immune modulation has developed a method of immune escape by viral pathogens. Manipulated microbial signaling can inhibit antigen presentation, down-regulate cytotoxic T cell actions or bias cytokine responses towards non-protective responses. These immune spaces prefer viral persistence and chronic infection. Notably, such effects are frequently indirect, which points to the microbiome as a mediator defining the interactions of hosts and virus without direct microbial-viral, direct interactions.[29]

The complexity of immunomodulation by bacterial communities highlights their significance in the determination of outcomes of the viral diseases. **Table 1** is a summary of the main microbiome-mediated immune mechanisms that can alter viral immune escape.[30]

Table 1: Microbiome-mediated mechanisms influencing host immunity and viral immune escape.[15,16,21,22]

Microbiome-related mechanism	Immune effect	Impact on viral infection
Regulation of pattern recognition receptors	Modulation of innate immune signaling and interferon responses	Alters early antiviral defense and viral replication efficiency
Production of short-chain fatty acids	Influences T cell differentiation and cytokine balance	Can enhance immune tolerance and support viral persistence
Modulation of antigen presentation	Affects activation of adaptive immune responses	Facilitates viral immune evasion
Induction of anti-inflammatory immune states	Suppression of excessive immune activation	Promotes chronic infection and immune escape
Dysbiosis-associated immune impairment	Reduced immune surveillance and delayed responses	Increased susceptibility to viral infection

This section highlights how bacterial communities indirectly govern viral immune escape by reshaping host immune landscapes. Understanding these interactions is critical for identifying microbiome-based strategies to restore effective antiviral immunity and limit viral persistence.[30]

Clinical Implications and Therapeutic Perspectives

The growing importance of interactions between the microbiome and viruses has a tremendous clinical implication in the prevention, diagnosis, and treatment of viral infections. As the presence of bacterial communities controls the viral entry, replication, and immune evasion, the modulation of the microbiomes can be considered an effective method in the strengthening of the effect of antivirals as well as reducing the intensity of the disease.[31]

The paradigm shift is not about the virus, but the host microbiome as a therapeutic target to take into account.

Amongst the most explored clinical applications, there is likely to be the recovery of microbial balance and antiviral immunity through the use of probiotics, prebiotic, and synabiotic. It has been demonstrated in other studies that some of the probiotics cultures can strengthen the mucosal defenses, enhance the effect of interferon, and reduce the viral load in respiratory and gastrointestinal infections. Such interventions would potentially lower the exposure of viruses to host cells and improve the appropriate immune responses as they enable the increase in the favorable bacteria populations.[32]

Antibiotic use is another clinically relevant condition which is associated with viral susceptibility. Widespread antibiotics can alter typical bacterial populations leading to dysbiosis and immunological regulations. This has been associated with severity of viral infections and reduced efficacy of vaccinations. Therefore, treatments with antibiotics and precautions to retain the health of microbiomes are imperative factors to take into account when treating viral diseases.[33]

Microbiome-derived biomarkers are also undergoing interest in their possible use to predict the risk of viral diseases and their advancement. Certain microbial patterns have been attributed to different immune responses and clinical outcome after being infected with a virus. These biomarkers can help to risk stratify and tailor treatment to the individual, especially to immunocompromised persons.[34]

Moreover, there are new therapeutic modalities that are to be directed against microbial metabolites and signal pathways that regulate viral proliferation and immune evasion. Regulating microbiome-produced metabolites or recapitulating positive microbial functions can be used to supplement traditional antiviral treatment. Despite the fact that these methods are still rather experimental, they

are a promising direction in antiviral interventions in the future.[35]

Together, the microbiome modulation implemented as a part of the clinical practice provides new opportunities to use antiviral defenses and achieve better patient outcomes. Nonetheless, these findings still need to be translated into standardized therapies with help of more mechanistic investigations and properly planned clinical trials.[36]

Conclusion and Future Directions

Interkingdom crosstalk Interkingdom interaction of bacterio communities and viral pathogens is a crucial and hitherto underrecognized aspect of viral pathogenesis. This review pinpoints the complicated roles of the microbiome in controlling viral entry, viral replication and immune evasion through direct microbial-viral interactions and indirect through host immune regulation. Bacterial communities are not spectators of viruses as they establish their grip upon the host, but are active determinants of host susceptibility, disease and fate.[37]

There is increasing evidence that the composition of microbiomes, functional capacity and metabolic activity jointly provide the environment within the host that the viruses are able to work in. Dysbiosis may interfere with immune homeostasis and promote viral persistence and a healthy microbiome helps with successful antiviral immunity. These lessons as well question the classic virus-centered approach to infection and highlight the necessity to pay attention to the ecosystems of host microorganisms when studying virology.

Although this has already advanced a great deal, there are numerous gaps in knowledge. The exact molecular pathways of interaction of microbiomes and viruses are not completely understood, and the role of certain taxa of bacteria or functional pathways in comparison to each other is a topic of further research. Moreover, the microbiome composition among individuals can vary, which makes it difficult to take the results of the experiment and apply them to a universal clinical practice.

The next studies in the field should be done towards combining multi-omics techniques such as metagenomics, metabolomics, and immunoprofiling to have a better understanding of interkingdom interactions. Longitudinal and interventional research will be necessary to determine the cause-effect relationships and single out microbiome-based treatment objectives. Finally, the regulatory potential of the microbiome could result in new antiviral approaches to supplement the current ones and enhance the health outcomes of the global population.

REFERENCES

1. Venkatesan P. GOLD report: 2022 update. Lancet Respir Med, 2022; 10(2): e20. doi:10.1016/S2213-2600(21)00561-0
2. Chung K, Adcock I. Multifaceted mechanisms in COPD: inflammation, immunity, and tissue repair and destruction. Eur Respir J., 2008; 31(6): 1334–56. doi:10.1183/ 09031936.00018908
3. Barbu C, Iordache M, Man M. Inflammation in COPD: pathogenesis, local and systemic effects. Rom J Morphol Embryol, 2011; 52(1): 21–7.
4. Barnes P, Burney P, Silverman EK, Celi BR, Vestbo J, Wedzicha JA, et al. Chronic obstructive pulmonary disease. Nat Rev Dis Primers, 2015; 1: 15076. doi: 10.1038/ nrdp.2015.76
5. Fabbri L, Luppi F, Beghé B, Rabe K. Complex chronic comorbidities of COPD. Eur Respir J., 2008; 31(1): 204–12. doi: 10.1183/09031936.00114307
6. Saetta M, Turato G, Maestrelli P, Mapp CE, Fabbri LM. Cellular and structural bases of chronic obstructive pulmonary disease. Am J Respir Crit Care Med, 2001; 163(6): 1304 9. doi:10.1164/ajrccm.163.6.2009116
7. Adeloye D, Chua S, Lee C, Basquill C, Papana A, Theodoratou E, et al. Global and regional estimates of COPD prevalence: Systematic review and meta–analysis. J Global Health, 2015; 5(2). doi: 10.7189/jogh.05.020415
8. Natalini JG, Singh S, Segal LN. The dynamic lung microbiome in health and disease. Nat Rev Microbiol, 2022; 1–14. doi: 10.1038/s41579-022-00821-x
9. Li L, Mac Aogáin M, Xu T, Jaggi TK, Chan LL, Qu J, et al. Neisseria species as pathobionts in bronchiectasis. Cell Host Microbe, 2022; 30(9): 1311–27.e8. doi: 10.1016/ j.chom.2022.08.005
10. Singh S, Segal LN. A lung pathobiont story: Thinking outside the koch's postulate box. Cell Host Microbe, 2022; 30(9): 1196–8. doi: 10.1016/j.chom.2022.08.012
11. O'Donnell AE. Bronchiectasis in patients with COPD: a distinct COPD phenotype? Chest, 2011; 140(5): 1107–8. doi: 10.1378/chest.11-1484
12. Martínez-García MÁ, Soler-Cataluña JJ, Sanz YD, Serra PC, Lerma MA, Vicente JB, et al. Factors associated with bronchiectasis in patients with COPD. Chest, 2011; 140(5): 1130–7. doi: 10.1378/chest.10-1758
13. Patel IS, Vlahos I, Wilkinson TM, Lloyd-Owen SJ, Donaldson GC, Wilks M, et al. Bronchiectasis, exacerbation indices, and inflammation in chronic obstructive pulmonary disease. Am J Respir Crit Care Med, 2004; 170(4): 400–7. doi: 10.1164/rccm.200305 648OC
14. Agusti A, Calverley P, Celli B, Coxson HO, Edwards LD, Lomas DA, et al. Characterisation of COPD heterogeneity in the ECLIPSE cohort. Respir Res, 2010; 11(1): 1–14. doi: 10.1186/1465-9921-11-122

15. Khamees, H. H., Mohammed, A. A., Hussein, S. A. M., Ahmed, M. A., & Raoof, A. S. M. (2024). In-Silico Study OF Destabilizing Alzheimer's Aβ42 Protofibrils with Curcumin. *International Journal of Medical Science and Dental Health*, *10*(05): 76-84.
16. Hou, K., Wu, Z. X., Chen, X. Y., Wang, J. Q., Zhang, D., Xiao, C., ... & Chen, Z. S. (2022). Microbiota in health and diseases. *Signal transduction and targeted therapy*, *7*(1): 135.17. Tiew PY, Jaggi TK, Chan LL, Chotirmall SH. The airway microbiome in COPD, bronchiectasis and bronchiectasis-COPD overlap. Clin Respir J., 2021; 15(2): 123–33. doi: 10.1111/crj.13294
17. Ding K, Chen J, Zhan W, Zhang S, Chen Y, Long S, et al. Microbiome links cigarette smoke-induced chronic obstructive pulmonary disease and dietary fiber via the gut-lung axis: A narrative review. COPD: J Chronic Obstructive Pulm Dis, 2022; 19(1): 10– 7. doi: 10.1080/15412555.2021.2019208
18. Dy R, Sethi S. The lung microbiome and exacerbations of COPD. Curr Opin pulm Med, 2016; 22(3): 196–202. doi: 10.1097/MCP.0000000000000268
19. Dickson RP, Erb-Downward JR, Martinez FJ, Huffnagle GB. The microbiome and the respiratory tract. Annu Rev Physiol, 2016; 78: 481. doi:10.1146/annurev-physiol- 021115-105238
20. Ditz B, Christenson S, Rossen J, Brightling C, Kerstjens HA, van den Berge M, et al. Sputum microbiome profiling in COPD: beyond singular pathogen detection. Thorax, 2020; 75(4): 338–44. doi: 10.1136/thoraxjnl-2019-214168.
21. Sharba, M. M., Mohammed, A. A., & Mohammed, S. F. (2022). Isolation and Characterization of tannase from isolated Bacillus subtilis.
22. Engel M, Endesfelder D, Schloter-Hai B, Kublik S, Granitsiotis MS, Boschetto P, et al. Influence of lung CT changes in chronic obstructive pulmonary disease (COPD) on the human lung microbiome. PloS One, 2017; 12(7): e0180859. doi: 10.1371/ journal.pone.0180859
23. Wang Z, Yang Y, Yan Z, Liu H, Chen B, Liang Z, et al. Multi-omic meta-analysis identifies functional signatures of airway microbiome in chronic obstructive pulmonary disease. ISME J., 2020; 14(11): 2748–65. doi: 10.1038/s41396-020-0727-y
24. Yatera K, Noguchi S, Mukae H. The microbiome in the lower respiratory tract. Respir Invest, 2018; 56(6): 432–9. doi: 10.1016/j.resinv.2018.08.003
25. Richardson H, Dicker AJ, Barclay H, Chalmers JD. The microbiome in bronchiectasis. Eur Respir Rev, 2019; 28(153). doi: 10.1183/16000617.0048-2019
26. Hussein, A. F., Mohammed, A. A., Hussein, S. A. M., & Malek, G. K. (2025). Serum Potassium, Phosphate, AND Calcium Levels AND Their Correlation with EGFR in Patients with Chronic Kidney Diseas. *International Journal of Medical Science and Dental Health*, *11*(11): 150-154.
27. Huang YJ, Erb-Downward JR, Dickson RP, Curtis JL, Huffnagle GB, Han MK. Understanding the role of the microbiome in chronic obstructive pulmonary disease: principles, challenges, and future directions. Trans Res, 2017; 179: 71–83. doi: 10.1016/ j.trsl.2016.06.007
28. Bowerman KL, Rehman SF, Vaughan A, Lachner N, Budden KF, Kim RY, et al. Disease-associated gut microbiome and metabolome changes in patients with chronic obstructive pulmonary disease. Nat Commun, 2020; 11(1): 1–15. doi: 10.1038/s41467-020-197010
29. Russo C, Colaianni V, Ielo G, Valle MS, Spicuzza L, Malaguarnera L. Impact of lung microbiota on COPD. Biomedicines, 2022; 10(6): 1337. doi:10.3390/ biomedicines10061337
30. Wang Z, Singh R, Miller BE, Tal-Singer R, Van Horn S, Tomsho L, et al. Sputum microbiome temporal variability and dysbiosis in chronic obstructive pulmonary disease exacerbations: an analysis of the COPDMAP study. Thorax., 2018; 73(4): 331–8. doi: 10.1136/thoraxjnl-2017-210741
31. Wang YJ, Wu SS, Chu J, Kong XY. Lung microbiome mediates the progression from chronic obstructive pulmonary disease to lung cancer through inflammation. Yi Chuan= Hereditas., 2021; 43(1): 30–9. doi: 10.16288/j.yczz.20-315
32. Malek, G. K., Hussein, S. A. M., & Mohammed, A. A. (2025). Effect of Puberty and Gender on Metabolic Hormones Level and Lipid Profile in Patients with Growth Hormone Deficiency. *International Journal of Medical Science and Dental Health*, *11*(11): 63-72.
33. Dicker AJ, Huang JT, Lonergan M, Keir HR, Fong CJ, Tan B, et al. The sputum microbiome, airway inflammation, and mortality in chronic obstructive pulmonary disease. J Allergy Clin Immunol (2021) 147(1):158–67. doi: 10.1016/j.jaci.2020.02.040
34. Diao W, Shen N, Du Y, Erb-Downward JR, Sun X, Guo C, et al. Symptom-related sputum microbiota in stable chronic obstructive pulmonary disease. Int J chronic obstructive pulm dis., 2018; 13: 2289. doi: 10.2147/COPD.S167618
35. Rogers GB, Zain NMM, Bruce KD, Burr LD, Chen AC, Rivett DW, et al. A novel microbiota stratification system predicts future exacerbations in bronchiectasis. Ann Am Thorac Soc, 2014; 11(4): 496–503. doi: 10.1513/AnnalsATS.201310-335OC
36. Lee SH, Lee Y, Park JS, Cho Y-J, Yoon HI, Lee C-T, et al. Characterization of microbiota in bronchiectasis patients with different disease severities. J Clin Med., 2018; 7(11): 429. doi: 10.3390/jcm7110429
37. mucilaginosa is an anti-inflammatory bacterium in the respiratory tract of patients wi chronic lung disease. Eur Respir J., 2022; 59(5). doi: 10.1183/13993003.01293-2021
38. Segal LN, Clemente JC, Tsay J-CJ, Koralov SB, Keller BC, Wu BG, et al. Enrichment of the lung microbiome with oral taxa is associated with lung inflammation of a Th17 phenotype. Nat Microbiol, 2016; 1(5): 1–11. doi: 10.1038 nmicrobiol.2016.31

39. Wu BG, Sulaiman I, Tsay J-CJ, Perez L, Franca B, Li Y, et al. Episodic aspiration with oral commensals induces a MyD88-dependent, pulmonary T-helper cell type 1 esponse that mitigates susceptibility to streptococcus pneumoniae. Am J Respir Crit Care Med, 2021; 203(9): 1099–111. doi: 10.1164/rccm.202005-1596OC