Advanced International Journal for Research (AIJFR)
E-ISSN: 3048-7641 ● Website: www.aijfr.com ● Email: editor@aijfr.com

The Role of Chronic Microplastic Exposure in the Pathogenesis of Non-Communicable Diseases: A Narrative review

Sourav Singla[1], Kartikey Sringari[2], Mandeep Singh[3], Vaibhav Singhal[4], Jahnavi Sharma[5], Navya Malhotra[6]

[1,2,3,4,5,6] MBBS Student, Shaheed Hasan Khan Mewati Government Medical College Nalhar, Nuh

Abstract

Background: Presence of microplastic in the environment causes chronic microplastic exposure to human through ingestion, inhalation and dermal contact. This chronic microplastic exposure lead to various non-communicable disease. Despite widespread environment prevalence of microplastic, the health risks associated with microplastic exposure are still uncertain. Microplastics, defined as plastic particles smaller than 5 mm in diameter, have become ubiquitous environmental contaminants due to the exponential growth in plastic production and inadequate waste management. Human exposure to microplastics occurs through ingestion, inhalation, and dermal contact, resulting in their accumulation in various tissues and organs. Emerging evidence suggests that chronic microplastic exposure may contribute to the development of noncommunicable diseases (NCDs) through multiple biological mechanisms.

Objective: To review current evidence regarding the role of chronic microplastic exposure in the pathogenesis of major non-communicable diseases, including cardiovascular disease, diabetes mellitus, cancer, and chronic respiratory diseases.

Methodology: A narrative review of published literatures was conducted to examine the relationship between chronic microplastic exposure and non-communicable disease development. Evidence from epidemiological, experimental, in vitro, and animal studies was reviewed to evaluate the biological pathways linking microplastic exposure to disease pathogenesis. Key mechanisms including oxidative stress, chronic inflammation, immune dysregulation, gut microbiota alteration, and cellular dysfunction were analyzed.

Result: Available evidence indicates that chronic microplastic exposure may contribute to the development and progression of NCDs through multiple interconnected mechanisms. Microplastics have been associated with vascular inflammation and atherosclerotic plaque formation, increased oxidative stress, disruption of glucose homeostasis, alterations in gut microbiota, and activation of oncogenic signaling pathways. Experimental studies have demonstrated that microplastic uptake by immune cells can induce pro-inflammatory responses and cellular damage, potentially increasing susceptibility to cardiovascular disease, diabetes, cancer, and chronic lung disease.

Conclusion: Although direct causal relationships in humans remain incompletely established, current evidence suggests that chronic microplastic exposure represents a potential emerging environmental risk factor for non-communicable diseases. Further longitudinal and mechanistic studies are required to clarify long-term health consequences and inform public health policies aimed at reducing human exposure to microplastics.

Keywords: Microplastics, Non-communicable diseases, Chronic inflammation, Oxidative stress, Cardiovascular disease, Diabetes mellitus, Cancer.

1. Introduction

The rapid growth of plastic production over the last several decades, combined with inadequate waste management practices, has resulted in the widespread contamination of terrestrial, freshwater, and marine ecosystems by plastic debris. According to the United Nations Environment Program (UNEP), more than 430 million tonnes of plastic are produced globally each year, with approximately two-thirds consisting of short-lived products that quickly become waste and enter the environment.[1]

Growing evidence indicates that human exposure to microplastics is widespread and unavoidable. Individuals may be exposed through multiple routes, including ingestion, inhalation, and dermal contact. Inhalation represents another important route of exposure, particularly in urban and indoor environments where synthetic textile fibers, household dust, and industrial emissions contribute to airborne microplastic particles. Although dermal absorption appears to be less significant, microplastics present in personal care products and cosmetics may also contribute to overall exposure.[2]

Similarly, Ragusa et al. demonstrated the presence of microplastics in human placental tissue, raising concerns regarding fetal exposure and potential developmental consequences. These findings have intensified scientific interest in understanding the potential impact of microplastic exposure on human health. NCDs are chronic diseases that are generally not transmitted from person to person and include cardiovascular diseases, cancer, chronic respiratory diseases, and diabetes mellitus. According to the World Health Organization (WHO), NCDs were responsible for at least 43 million deaths globally in 2021, accounting for approximately 75% of all nonpandemic-related deaths.[3]

The increasing prevalence of NCDs has traditionally been attributed to well-established risk factors such as tobacco use, unhealthy diet, physical inactivity, harmful alcohol consumption, obesity, and air pollution.[3,4]

Recent advances in analytical techniques have enabled researchers to identify microplastics in previously inaccessible human tissues, further strengthening concerns regarding their potential health effects.[5,6] The ability of microplastics to cross biological barriers and accumulate in various organs has raised concerns regarding their long-term biological consequences.[6,7] These contaminants may be released following ingestion or inhalation and subsequently interact with cellular and molecular pathways involved in disease pathogenesis.[8] Furthermore, microplastics may serve as vectors for a variety of toxic substances, including heavy metals, polycyclic aromatic hydrocarbons, bisphenol A, and phthalates, thereby increasing their potential to induce adverse
health effects.[8,9]

Numerous experimental studies have demonstrated that exposure to microplastics can trigger excessive production of reactive oxygen species, activate inflammatory signaling pathways, disrupt mitochondrial function, and impair cellular homeostasis.[10,11] Persistent activation of these mechanisms may contribute to chronic low-grade inflammation, which is recognized as a central feature in the development of cardiovascular diseases, metabolic disorders, cancer, and other chronic conditions.[11,12] In addition, microplastic exposure has been associated with alterations in gut microbiota composition and intestinal barrier function, potentially contributing to metabolic dysfunction and systemic inflammation.[13]

Consequently, even modest adverse health effects may translate into a substantial disease burden at the population level. As global plastic production is projected to continue increasing in the coming decades, concerns regarding cumulative exposure and its impact on chronic disease prevalence are likely to intensify. Therefore, investigating the potential contribution of microplastics to NCD pathogenesis represents an important research priority for environmental health, preventive medicine, and public health policy.[14]

Despite growing evidence regarding the presence of microplastics in human tissues and their potential biological effects, significant uncertainties remain concerning the extent of their impact on human health.[15,16] Most current evidence originates from experimental animal models and in vitro studies, while human epidemiological data remain limited.[15] Furthermore, variations in particle size, shape, polymer composition, concentration, and duration of exposure complicate the assessment of health risks and hinder the establishment of standardized exposure thresholds.[16] The lack of universally accepted methodologies for sampling, detection, and quantification of microplastics further contributes to inconsistencies among studies and challenges the interpretation of findings.[17]

Environmental exposures have historically played a crucial role in shaping disease patterns across populations.[18] Air pollution, heavy metals, endocrine-disrupting chemicals, and occupational toxins have all been implicated in the development of chronic diseases through mechanisms involving oxidative stress, inflammation, and cellular dysfunction.[18,19] Their persistence in the environment, widespread distribution, and ability to accumulate within biological systems distinguish them from many conventional pollutants and underscore the urgency of understanding their long-term consequences.[19]

Of particular concern is the potential interaction between microplastics and existing risk factors for non-communicable diseases.[20] Individuals with obesity, diabetes mellitus, cardiovascular disease, and chronic respiratory conditions often exhibit underlying systemic inflammation and metabolic dysregulation.[20,21]

The growing recognition of microplastics as a potential threat to human health has prompted international organizations and public health agencies to prioritize research in this field.[22] Due to their extremely small size, nanoplastics can more readily cross cellular membranes, penetrate biological barriers, and interact directly with intracellular structures.[23] Experimental studies suggest that nanoplastics may enter the bloodstream, cross the blood-brain barrier, and accumulate within vital organs, potentially resulting in enhanced toxicity compared with larger
particles.[24,25]

Certain population groups may be particularly vulnerable to the adverse consequences of microplastic exposure.[26] Infants and children may experience greater exposure relative to body weight because of

behavioral factors, dietary patterns and developmental susceptibility.[26] Pregnant women represent another population of concern, given evidence demonstrating the presence of microplastics within placental tissue and the possibility of fetal exposure during critical developmental periods.[27] Likewise, individuals with pre-existing chronic diseases, compromised immune systems, or occupational exposure to plastic particles may be at increased risk of adverse health outcomes.[27]

The potential health effects of microplastics extend beyond individual disease outcomes and may have broader socioeconomic implications. If chronic microplastic exposure contributes even modestly to the incidence or progression of NCDs, the cumulative public health and economic consequences could be considerable.[28] Understanding environmental contributors to disease therefore has important implications not only for healthcare systems but also for sustainable development and economic planning.[28]

The concept of One Health provides a useful framework for understanding the interconnected nature of microplastic pollution and human disease.[29] One Health recognizes that human health is closely linked to the health of animals and the environment.[29] Microplastics contaminate ecosystems globally, affecting marine organisms, wildlife, livestock, and ultimately human populations through interconnected food chains and environmental pathways.[30]

Recent scientific advances have also highlighted the importance of investigating combined environmental exposures rather than evaluating microplastics in isolation. Humans are simultaneously exposed to numerous pollutants, including air pollutants, heavy metals, pesticides, endocrine-disrupting chemicals, and plastic-associated compounds.[31] Interactions among these exposures may amplify biological responses and influence disease susceptibility through synergistic mechanisms involving inflammation, oxidative stress, and immune dysregulation. Therefore, future research should adopt a comprehensive exposome-based approach to better characterize the cumulative impact of environmental contaminants on non-communicable disease
risk.[32]

2. Methodology

This narrative review was conducted to evaluate current evidence regarding the role of chronic microplastic exposure in the pathogenesis of major non-communicable diseases (NCDs), including cardiovascular diseases, cancer, diabetes mellitus, and chronic respiratory diseases. The review also aimed to examine the biological mechanisms that may mediate the relationship between microplastic exposure and disease development.

A comprehensive literature search was performed using multiple electronic databases, including PubMed, Scopus, Web of Science, and Google Scholar. Relevant articles published between January 2010 and June 2025 were considered for inclusion in the review. Additional articles were identified through manual screening of reference lists from eligible studies and review articles. Data extracted from eligible studies included study design, population characteristics, exposure assessment methods, disease outcomes, major findings, and proposed biological mechanisms. Evidence from human epidemiological studies was prioritized where available; however, because of the limited number of human studies in this emerging

field, experimental animal studies and mechanistic in vitro investigations were also included to provide insight into potential pathogenic pathways.

The findings were synthesized narratively and organized into thematic sections addressing the association between microplastic exposure and major non-communicable diseases. Special attention was given to mechanistic pathways, including oxidative stress, chronic inflammation, immune dysregulation, endocrine disruption, gut microbiota alterations, and mitochondrial dysfunction, which have been proposed as key contributors to disease development and progression. The collected evidence was subsequently integrated to provide a comprehensive overview of the current understanding of microplastic-associated health risks and to identify gaps requiring further investigation.

1. This narrative review was conducted to evaluate the current evidence regarding the association between microplastic exposure and cardiovascular disease (CVD), with particular emphasis on the underlying biological mechanisms that may contribute to cardiovascular pathogenesis. The review aimed to synthesize findings from epidemiological studies, clinical investigations, experimental animal studies, and in vitro research examining the cardiovascular effects of microplastic exposure.

2. This narrative review was conducted to evaluate the current evidence regarding the association between microplastic exposure and cancer development, with particular emphasis on the molecular and cellular mechanisms involved in carcinogenesis. The review aimed to synthesize findings from human studies, experimental animal models, in vitro investigations, and review articles that examined the potential role of microplastics in cancer initiation, promotion, and progression. Particular attention was given to mechanisms including oxidative stress, DNA damage, activation of inflammatory signaling pathways such as nuclear factor-kappa B (NF-κB), and alterations in tumor suppressor genes including p53, which have been implicated in cancer pathogenesis.

3. This narrative review was conducted to evaluate current evidence regarding the association between microplastic exposure and diabetes mellitus, with particular emphasis on the biological mechanisms through which microplastics may contribute to metabolic dysfunction. The review aimed to synthesize findings from epidemiological studies, experimental animal models, in vitro investigations, and review articles examining the effects of microplastics on gut microbiota composition, insulin resistance, glucose homeostasis, chronic inflammation, and metabolic health. Given the rapidly increasing global prevalence of diabetes and the growing concern regarding environmental contaminants, understanding the potential role of microplastics in metabolic disease development has become an important area of research.

4. This narrative review was conducted to evaluate the current evidence regarding the association between microplastic exposure and chronic lung disease, with particular emphasis on inhalation exposure, alveolar macrophage activation, pulmonary inflammation, and respiratory toxicity. The review aimed to synthesize findings from epidemiological studies, occupational health investigations, experimental animal studies, in vitro research, and review articles examining the potential respiratory health effects of microplastics and nanoplastics. Given the increasing concentration of airborne microplastics in both indoor and outdoor environments, understanding

their contribution to chronic respiratory disease has become an important area of environmental health research.

5. This narrative review was conducted to examine the potential biological mechanisms through which chronic microplastic exposure may contribute to the development and progression of non-communicable diseases (NCDs). Particular emphasis was placed on five major mechanistic pathways that have been repeatedly identified in experimental and emerging human studies: oxidative stress, chronic inflammation, endocrine disruption, immune dysregulation, and mitochondrial dysfunction. Understanding these mechanisms is essential because they represent common pathological processes underlying several major NCDs, including cardiovascular diseases, diabetes mellitus, cancer, and chronic respiratory diseases.

Study Selection and Data Extraction

Retrieved articles were initially screened based on titles and abstracts. Potentially relevant studies underwent full-text assessment to determine eligibility. Studies meeting the predefined inclusion criteria were selected for detailed review.

Data extracted from eligible studies included study design, exposure characteristics, biological models, investigated mechanisms, biomarkers assessed, and principal findings. Particular emphasis was placed on studies demonstrating molecular and cellular alterations following microplastic exposure and their relevance to chronic disease pathogenesis.

Data Synthesis

The findings were synthesized narratively and organized according to five principal biological mechanisms:

1. Oxidative Stress

Evidence examining excessive generation of reactive oxygen species (ROS), lipid peroxidation, DNA damage, protein oxidation, and impairment of antioxidant defense systems following microplastic exposure was reviewed.

2. Chronic Inflammation

Studies investigating activation of inflammatory pathways, cytokine production, inflammasome activation, and persistent low-grade inflammatory responses were evaluated.

3. Endocrine Disruption

Evidence regarding interference with hormonal signaling pathways, endocrine-disrupting chemicals associated with plastics, and their impact on metabolic and reproductive health was examined.

4. Immune Dysregulation

Studies assessing alterations in innate and adaptive immune responses, immunotoxicity, immune activation, and impaired immune homeostasis were reviewed.

5. Mitochondrial Dysfunction
Evidence concerning mitochondrial damage, impaired energy metabolism, altered ATP production, mitochondrial oxidative stress, and apoptosis-related pathways was analyzed.

The collected evidence was subsequently integrated to provide a comprehensive overview of the mechanistic pathways linking chronic microplastic exposure to the development of noncommunicable diseases and to identify important gaps for future research.

3. Discussion

The present narrative review synthesizes current evidence regarding the potential role of chronic microplastic exposure in the development of major non-communicable diseases (NCDs), including cardiovascular diseases, cancer, diabetes mellitus, and chronic respiratory diseases. The findings suggest that microplastics are emerging environmental contaminants with the capacity to influence multiple biological systems through mechanisms such as oxidative stress, chronic inflammation, endocrine disruption, immune dysregulation, and mitochondrial dysfunction. Although definitive causal relationships in humans have not yet been established, the growing body of experimental and observational evidence raises important concerns regarding the long-term health implications of continuous microplastic exposure.

Strength of Current Evidence

The current evidence linking microplastic exposure to NCDs is expanding rapidly but remains heterogeneous in quality and scope. Experimental studies have consistently demonstrated that microplastics can induce oxidative stress, inflammatory responses, cellular toxicity, and metabolic disturbances in various animal models and cell cultures. These findings provide strong biological plausibility for a relationship between microplastic exposure and chronic disease development. Numerous studies have reported increased reactive oxygen species production, DNA damage, inflammatory cytokine release, and disruption of cellular homeostasis following exposure to microplastics and nanoplastics.
Recent human studies have further strengthened concerns regarding potential health effects. The detection of microplastics in human blood, placental tissue, breast milk, lung tissue, liver tissue, and atherosclerotic plaques indicates that these particles can enter the human body, circulate systemically, and accumulate within organs. Particularly noteworthy is the recent evidence demonstrating the presence of microplastics within carotid artery plaques and their association with increased cardiovascular events. Such findings provide direct evidence of human tissue exposure and suggest possible clinical relevance.
However, despite these advances, the overall strength of evidence remains moderate rather than conclusive. Most available studies focus on mechanistic pathways rather than direct disease outcomes. Furthermore, considerable variation exists in exposure assessment methods, particle characteristics, experimental models, and outcome measures, making direct comparisons difficult. Therefore, while the biological plausibility is strong, robust epidemiological evidence establishing causality remains limited.

Human Evidence versus Animal Evidence

A critical observation from this review is the imbalance between animal and human evidence. The majority of current knowledge regarding microplastic toxicity originates from experimental animal studies and in

vitro investigations. Animal models have demonstrated associations between microplastic exposure and endothelial dysfunction, insulin resistance, pulmonary inflammation, immune dysregulation, reproductive toxicity, and carcinogenic processes. These studies have been instrumental in identifying potential mechanisms linking microplastics to chronic disease development.

Nevertheless, the translation of findings from animal models to human populations requires caution. Experimental studies often utilize exposure concentrations that may exceed those encountered under real-world conditions. Differences in physiology, metabolism, exposure duration, and particle characteristics may influence the applicability of these findings to humans. Consequently, animal studies should primarily be viewed as evidence supporting biological plausibility rather than definitive proof of human health effects.

Human evidence remains comparatively limited but is steadily increasing. Epidemiological studies investigating direct associations between microplastic exposure and disease outcomes are still scarce. Most human studies have focused on detecting microplastics in biological tissues rather than evaluating long-term health consequences. As a result, substantial uncertainty remains regarding exposure thresholds, dose-response relationships, cumulative exposure effects, and susceptibility among different populations. Future large-scale prospective cohort studies will be essential to determine whether chronic microplastic exposure contributes meaningfully to the burden of NCDs.

4. Public Health Implications

The potential public health implications of microplastic exposure are considerable. Unlike many environmental contaminants that affect specific occupational groups or geographic regions, microplastics have become ubiquitous pollutants present in air, water, soil, food products, and consumer goods worldwide. Consequently, exposure occurs across all age groups and socioeconomic settings, making microplastic pollution a truly global environmental health issue. Even if the health effects of individual exposures are relatively small, the widespread nature of exposure means that the cumulative impact at the population level could be substantial. Given that NCDs already account for approximately three-quarters of global deaths, the identification of additional environmental risk factors is of considerable importance. If chronic microplastic exposure contributes to cardiovascular disease, diabetes, cancer, or chronic respiratory diseases, reducing exposure may represent an important preventive public health strategy.

Particularly vulnerable populations may include pregnant women, infants, children, older adults, and individuals with pre-existing chronic diseases. The detection of microplastics in placental tissue and breast milk raises concerns regarding early-life exposure during critical developmental periods. Such exposure may have lifelong consequences for health and disease susceptibility, highlighting the need for precautionary approaches until more definitive evidence becomes available.

5. Research Gaps

Despite growing scientific interest, numerous knowledge gaps remain. First, there is currently no universally accepted method for measuring human microplastic exposure. Variations in sampling techniques, laboratory methodologies, and particle characterization contribute to inconsistencies across studies. Standardized protocols are urgently needed to improve comparability and reliability.

Second, the majority of available studies are cross-sectional or experimental in nature. Longitudinal epidemiological studies assessing chronic exposure and long-term health outcomes remain scarce. Prospective cohort studies involving diverse populations are necessary to establish temporal relationships and causality.

Third, little is known regarding dose-response relationships. It remains unclear what levels of exposure may be considered safe or harmful. Similarly, the effects of different particle sizes, shapes, polymer types, and associated chemical contaminants require further investigation.
Fourth, most studies evaluate microplastics in isolation, whereas humans are exposed simultaneously to multiple environmental pollutants. Future research should adopt an exposomebased approach that considers interactions between microplastics, air pollution, heavy metals, endocrine-disrupting chemicals, and other environmental stressors.

Finally, limited information exists regarding susceptibility factors such as age, sex, genetic predisposition, nutritional status, and pre-existing disease. Identifying vulnerable populations will be essential for targeted prevention and risk assessment strategies.

6. Policy Implications

The findings of this review have important implications for environmental and public health policy. Although current evidence does not yet justify definitive conclusions regarding causality, the widespread presence of microplastics and their demonstrated biological effects support the application of the precautionary principle. Preventive action may be warranted even while scientific uncertainties remain.
Governments and regulatory agencies should prioritize strategies aimed at reducing plastic pollution at its source. These measures may include strengthening waste management systems, promoting recycling initiatives, limiting unnecessary single-use plastics, and encouraging the development of sustainable alternatives. Improved environmental monitoring and surveillance programs are also needed to assess population exposure levels and identify high-risk regions.

Public health agencies should support interdisciplinary research examining the health effects of microplastics and establish standardized methodologies for exposure assessment. International collaboration will be particularly important given the transboundary nature of plastic pollution and the global scale of exposure.

Furthermore, healthcare professionals and policymakers should recognize microplastic pollution as a potential environmental determinant of chronic disease. Integrating environmental health considerations into NCD prevention strategies may contribute to more comprehensive approaches for reducing the global burden of chronic diseases.

Conclusion of Discussion

In summary, current evidence suggests that chronic microplastic exposure may contribute to the pathogenesis of non-communicable diseases through multiple interconnected biological mechanisms, including oxidative stress, inflammation, endocrine disruption, immune dysregulation, and mitochondrial dysfunction. While experimental evidence is substantial, human epidemiological evidence remains limited, preventing definitive causal conclusions. Nevertheless, the ubiquity of exposure, emerging

evidence of human tissue accumulation, and potential implications for public health justify continued investigation. Future research should focus on standardized exposure assessment, longitudinal human studies, and mechanistic validation to clarify the role of microplastics as an emerging environmental risk factor for non-communicable diseases.

7. Conclusion

Microplastic pollution has emerged as a rapidly growing environmental and public health concern due to its ubiquitous presence in air, water, food, and various consumer products. The evidence reviewed in this study suggests that chronic microplastic exposure may play a role in the development and progression of major non-communicable diseases (NCDs), including cardiovascular diseases, diabetes mellitus, cancer, and chronic respiratory diseases. Although direct causal relationships in humans have not yet been conclusively established, accumulating experimental and observational evidence indicates that microplastics can interact with multiple biological systems and induce adverse cellular and molecular changes.

Several interconnected mechanisms have been identified that may link microplastic exposure to NCD pathogenesis. These include oxidative stress, chronic inflammation, endocrine disruption, immune dysregulation, mitochondrial dysfunction, alterations in gut microbiota, and cellular toxicity. Such mechanisms are recognized contributors to the development of chronic diseases and provide biological plausibility for the observed associations. Furthermore, recent detection of microplastics in human blood, placental tissue, lung tissue, liver tissue, breast milk, and atherosclerotic plaques highlights the potential for systemic distribution and long-term accumulation within the human body.

Despite growing concern, important knowledge gaps remain regarding exposure assessment, doseresponse relationships, long-term health outcomes, and the effects of combined environmental exposures. Most available evidence originates from experimental animal and in vitro studies, emphasizing the need for well-designed epidemiological studies and long-term prospective research in human populations. Standardized methodologies for measuring microplastic exposure and evaluating health outcomes are also essential to improve comparability and strengthen future evidence.

Given the widespread nature of microplastic contamination and the substantial global burden of NCDs, reducing environmental plastic pollution may represent an important preventive public health strategy. Addressing this emerging challenge will require multidisciplinary collaboration among researchers, healthcare professionals, policymakers, environmental agencies, and industry stakeholders. Therefore, an integrated One Health approach, recognizing the interdependence of human, animal, and environmental health, is crucial for understanding and mitigating the health consequences of microplastic exposure.

In conclusion, current evidence suggests that chronic microplastic exposure may represent an emerging environmental risk factor for non-communicable diseases. While further research is needed to establish definitive causal relationships, the existing findings underscore the urgent need for enhanced surveillance, preventive interventions, sustainable plastic management policies, and continued scientific investigation to protect population health in an increasingly plastic-dependent world.

8. Appendix

This narrative review examines the potential association between chronic microplastic exposure and major non-communicable diseases, including cardiovascular diseases, diabetes mellitus, cancer, and chronic respiratory diseases. The principal mechanisms discussed include oxidative stress, chronic inflammation, endocrine disruption, immune dysregulation, mitochondrial dysfunction, and alterations in gut microbiota.

9. Abbreviations

CVD, cardiovascular disease; DNA, deoxyribonucleic acid; NCDs, non-communicable diseases; NF-κB, nuclear factor kappa B; ROS, reactive oxygen species.

10. Conflict of Interest

The authors declare that they have no conflicts of interest related to the preparation and publication of this manuscript.

11. Acknowledgement

The authors would like to acknowledge Shaheed Hasan Khan Mewati Government Medical College, Nalhar, Nuh, Haryana, India, for providing the academic environment in which their interest in medical research has developed. The authors also acknowledge the contributions of researchers whose published work informed this narrative review. The authors are grateful for the collaborative learning and research experiences gained through participation in college-level and inter-college research activities.

12. Author Biographies

Sourav Singla is an undergraduate Bachelor of Medicine and Bachelor of Surgery (MBBS) student at Shaheed Hasan Khan Mewati Government Medical College, Nalhar, Nuh, Haryana, India. His academic interests include biomedical research, environmental health, and the pathogenesis of non-communicable diseases.

Kartikey Sringari is an undergraduate MBBS student at Shaheed Hasan Khan Mewati Government Medical College, Nalhar, Nuh, Haryana, India. His academic interests include medical research and collaborative undergraduate research initiatives.

Mandeep Singh is an undergraduate MBBS student at Shaheed Hasan Khan Mewati Government Medical College, Nalhar, Nuh, Haryana, India. He is interested in medical research and interdisciplinary approaches to understanding disease mechanisms.

Vaibhav Singhal is an undergraduate MBBS student at Shaheed Hasan Khan Mewati Government Medical College, Nalhar, Nuh, Haryana, India. His academic interests include biomedical research and participation in collaborative research projects.

Jahnavi Sharma is an undergraduate MBBS student at Shaheed Hasan Khan Mewati Government Medical College, Nalhar, Nuh, Haryana, India. He is interested in medical research and academic collaboration.

Navya Malhotra is an undergraduate MBBS student at Shaheed Hasan Khan Mewati Government Medical College, Nalhar, Nuh, Haryana, India. Their academic interests include biomedical research and participation in college-level and inter-college research projects.

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