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Review

Environmental microplastic exposure and lung cancer: emerging evidence and a thoracic surgery perspective

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ABSTRACT

In modern times, microplastics are common environmental contaminants that are of increasing importance for human health. Ingestion has been traditionally regarded as the dominant route of exposure. However, inhalation of airborne microplastics represents a direct and biologically relevant exposure route to the respiratory system. Increasing evidence, both experimental and review-based, suggests that inhaled microplastics may become trapped in lung tissue, remain there for long periods of time, generate oxidative stress and chronic inflammation, and disrupt normal cellular function in the lungs. These pathophysiological mechanisms may be directly involved in the development of lung cancer. From the perspective of thoracic surgery, exposure to microplastics can influence the epidemiology of lung cancer, tumor biology, perioperative risk, and future surgical decision making. Identification of these novel relationships could place thoracic surgeons at the intersection of surgical oncology and environmental health. This article aims to increase public awareness of environmental microplastic exposure and the possible link between microplastic exposure and lung cancer based on published peer-reviewed research. The purpose is to evaluate the potential impact of microplastics on thoracic surgery procedures and to alert thoracic surgeons about the possibility of an increase in lung cancer cases due to microplastics in the near future.

Keywords: microplastic, environmental, lung cancer, thoracic surgery

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Introduction

Plastics are used widely in our society, but most of the plastic waste ends up in landfills and in the environment. Their degradation into submillimeter fragments, known as microplastics, is an emerging concern due to potential adverse effects on the environment and human health. Microplastics are airborne, inhalable by humans, and their detrimental effects on the respiratory system are being studied [1]. Beyond its environmental importance, microplastic exposure may have implications for the practice of thoracic surgery, particularly its potential impact on lung cancer epidemiology, tumor biology, and postoperative outcomes. In addition to the environmental and respiratory health concerns, the possible clinical implications of microplastic exposure remain largely unknown in surgical disciplines. The purpose of this paper is to raise public awareness of exposure to environmental microplastics and the potential association of microplastic exposure with lung cancer based on peer-reviewed published research. It aims to evaluate the potential consequences for thoracic surgical procedures, to alert thoracic surgeons to the possible emergence of microplastic lung carcinomas in the near future, and to highlight the active role of thoracic surgery.

Based on microplastic particle characterization, the most abundant particles found in lung tissue were polypropylene and polyethylene. These polymers are the most common types of plastics in production; they can be found, among others, in banknotes, food packaging, automobile parts, and in the packaging of snacks and candies [1]. In recent years, microplastics have been detected in the air, both indoors and outdoors. Indoor environments tend to have more fibers than outdoor environments. The manufacture of clothing often uses natural fibers treated with chemicals such as flame retardants and dyed, which may affect their biodegradability. The type of polymers found in the lungs is likely related to indoor exposures because most city dwellers spend more time indoors than outdoors. The main causes include dust settling and reentering the atmosphere, industrial emissions, plastic material degradation in the environment, and synthetic textile products. Because of their small size, irregular shape, and low density, microplastic particles, especially those in fibrous form, may possess aerodynamic properties that allow them to deeply penetrate the pulmonary system. Microplastics are used extensively in daily human activities. They may enter the lungs by the respiratory pathway, accumulate around the primary lesion, and ultimately become caught by the expanding tissue of the tumor. Because of the enhanced permeability and retention effect, microplastics can concurrently enter the bloodstream through the digestive system, travel to the lungs, and then infiltrate tumors. The significant variation in microplastic particle size may be caused by these two primary mechanisms [1,2].

Experimental and observational studies have shown that inhaled microplastics can reach the lower respiratory tract, where physiological clearance processes are somewhat less effective [3]. The persistence of these particles within lung tissue raises concerns regarding cumulative accumulation and long-term respiratory health effects, especially in cases of long-term, low-dose exposure. Microplastics can settle in different areas of the respiratory system based on their size, shape, and surface characteristics. The bronchiolar and alveolar regions are more readily penetrated by smaller microplastics and nanoscale plastic particles. It is reasonable to anticipate higher particle retention in these regions. Data from experimental exposure studies support the hypothesis that microplastics may generate persistent particle-tissue interactions and stay in lung tissue for extended periods of time. Because of this persistence, alveolar epithelial cells, which are crucial for maintaining immunological homeostasis and the integrity of the pulmonary barrier, may be more vulnerable to cellular stress reactions. The size of microplastics appears to have a major impact on the phagocytic efficiency of alveolar macrophages. The impact of microplastics on alveolar macrophage function is still not entirely understood [3].

An increasing number of experimental studies have shown that inhaled microplastics can induce oxidative stress, inflammatory responses, and epithelial damage in lung tissue. Studies carried out both in vitro and in vivo after microplastic exposure consistently show elevated production of reactive oxygen species, activation of inflammatory signaling pathways, and enhanced release of pro-inflammatory mediators [2,3]. These biological responses may disrupt the epithelial barrier's ability to function and jeopardize proper pulmonary homeostasis. Review-based evaluations show that micro and nanoplastics can interact with intracellular signaling pathways involved in tissue remodeling, immunological regulation, and cellular stress responses. Additionally, they might physically damage blood vessels, which could lead to inflammation and stress. Additionally, microplastics can lower the energy supply and disrupt respiratory processes. The effect on human health depends on the level of exposure to concentration. Microplastics usually affect the neurological system, excretory system, gastrointestinal tract, internal organs, respiratory tract, and placenta. Inhaling these microscopic particles could lead to their accumulation in the respiratory system, raising worries about respiratory issues and inflammation. Therefore, future studies on microplastics should look more closely at people's inhalation exposure levels, assessing both the level of exposure and any potential effects on respiratory health. Additionally, it will be critical to evaluate the biological mechanisms by which microplastics may affect respiratory health, considering factors such as toxicity, inflammation, and potential long-term consequences on various respiratory and cardiovascular organ systems. It is recognized that air microplastics pose risks to human health and the environment since they are a major source of particles that humans and other organisms consume and inhale. Environmental particle exposure is associated with long-term lung pathology through significant mechanisms, such as inflammation and chronic oxidative stress, which are known causes of pulmonary disease [4,5].

The increasing amount of data suggests that exposure to microplastics may play a physiologically plausible but indirect role in carcinogenic processes. Chronic inflammation caused by persistent particle buildup can establish a tumor-promoting environment with oxidative DNA damage, impaired DNA repair processes, and dysregulated cell proliferation [6]. Some of the most well-known markers of the development of cancer are these mechanisms. Additionally, by serving as carriers for co-occurring ambient pollutants or chemical additives, microplastics may intensify local detrimental and genotoxic effects within lung tissue. The significance of particle exposure in the development of lung cancer is emphasized in reviews that focus on the relationship between airborne particulate matter and lung cancer. Microplastics have several physical characteristics with recognized carcinogenic particles, including their small size, surface reactivity, and capacity to induce persistent inflammatory reactions [7]. Current mechanistic insights provide a physiologically solid framework that stimulates further investigation, even if there is currently limited direct epidemiological evidence linking breathing microplastics to lung cancer.

With over 2 million fatalities reported in 2021, lung cancer is the most common cancer in the world and the primary cause of cancer-related mortality [8]. Growing global air pollution, especially in low- and middle-income countries, continues to contribute to high mortality from lung cancer and other respiratory diseases, even if incidence and death have decreased in wealthier nations [8]. Only around 10% of the more than 450 million tons of plastic generated annually worldwide is recycled, and estimates indicate that by 2050, the amount of plastic garbage entering the environment could surpass 30 million tons [8]. The existence of microplastics in soil and even the environment has drawn more attention in the last ten years. Especially considering the ability of plastic particles to be carried by atmospheric processes. Additionally, aquatic pollution makes it easier for plastics to enter food webs, where detrimental impacts have been shown at several trophic levels [8,9].

Due to their greater ability to penetrate, smaller particles are more dangerous. Particulate matter with a size of 2.5 has been shown to cause or hasten the development of cancer by directly damaging DNA in lung tissue and generating pro-inflammatory proteins. Clinical and experimental studies have shown that both smokers and non-smokers have a significantly higher incidence of lung cancer in urban areas with particulate matter pollution.[8] In light of ongoing climate change and the expansion of global economic activity and its associated environmental impacts, lung illnesses, including lung cancer, are predicted to remain a major public health concern for many years to come [8,10].

Outdoor microplastic concentrations are lower than indoor ones, and barometric pressure, humidity, wind, and location all affect the abundance of particular polymer particle sizes and shapes [8]. This suggests that microplastic intake is more common in offices and among other employees, as well as among those who work with polymers. According to research, air quality and exposure to ambient microplastics are significantly influenced by the type, quantity, and quality of room ventilation [8]. Concerns over patients being exposed to microplastics during surgery or medication infusions have also surfaced recently. According to a recent study, commercially available saline and glucose infusions frequently contain nanoparticles of different synthetic polymers, polypropylene, and polystyrene, which may have detrimental effects on patients [8,11].

In order to assess the available data and apply environmental discoveries to medical research, it is essential to understand the analytical techniques utilized to identify and characterize microplastics. The validity, comparability, and clinical importance of published data are directly impacted by the advanced spectroscopic and imaging techniques used to detect microplastics in human lung tissue. Because surgically removed lung tissues may provide valuable biological material for assessing long-term environmental exposure, standardized and reproducible detection techniques are particularly important for thoracic surgery research. There are numerous techniques for identifying plastic in the environment and living things. The most widely used approach and the gold standard is Fourier Transform Infrared Spectroscopy (FTIR). The micro-FTIR technique allows for the evaluation of individual spectral bands thanks to focal arrays, which in turn translates into greater efficiency [8,12,13]. FTIR is often used in conjunction with Raman spectroscopy (RS), which is based on Raman scattering radiation, because of the restrictions on particle size. A variety of qualitative sample analysis services are provided by RS. In contrast, Micro-Raman spectroscopy (MRS) enables the evaluation of micrometer-sized items by focusing the laser on a small research area. According to MRS, enhanced stimulated Raman spectroscopy (SRS) with increased efficiency was achieved [8,14-16]. The laser-assisted direct infrared (LDIR) imaging technique has a noticeably quicker measurement time [8,17]. Optical photothermal infrared spectroscopy (O-PTIR) is another method that uses spectroscopy. By monitoring infrared absorption during thermal expansions of the material caused by an infrared laser, a Quantum Cascade Laser approach that uses IR and Raman spectroscopy enables highly effective qualitative assessment of particles [8,18]. Using fluorescent dyes for optical microscopic inspection is another method for evaluating microplastics. It is a reasonably priced approach, but because of the size of the particles and the presence of pollutants in the sample, it gives relatively low accuracy and necessitates labor-intensive sample and particle preparation [8,19]. The energy-dispersive X-ray scanning electron microscope (SEM) is another microscopy-based technique. Although it is one of the more difficult, costly, and low-throughput methods, it offers the capacity to evaluate the surface and elemental composition of particular particles [8,20]. Atomic force microscopy and infrared spectroscopy are combined to create the AFM-IR, a technique used for in situ microplastic analysis in tissue and water samples [8,21]. Mass spectrometry has a narrow range of evaluations. Mass spectrometry and chromatography of evaporated sample material are combined in pyrolysis–gas chromatography mass spectrometry (Py-GC-MS) [8,22,23]. Thermogravimetric analysis of microplastics (TGA), a technique based on detecting the mass loss of a sample during heating at a regulated pace, also uses the sample's thermal breakdown [8,24].

According to Zhu et al.'s study, lung tissue had the greatest microplastic concentrations, with almost 14 particles per gram and sizes ranging from 20 μm to 100 μm, with Polyvinyl chloride being the most common kind of polymer [8,25]. Given that it is impossible to totally remove all aggregated particles from lung tissue and that chronic inflammation encourages the growth of malignancies, such as liver or lung cancer, the data show that even brief exposure to MNP may have long-term effects [6,8,26]. The detrimental effects of cigarette smoke, dust, and microplastics on animal respiratory systems have been documented, and micro- and nano-plastic particles have been found in commercially available cigarettes. When combined, cigarette smoke and microplastics showed the anticipated increased harmful effects on BEAS-2B cells, including oxidative stress, genotoxicity, anchorage-independent growth, invasiveness, and a reduced oxidative stress response

[8,27]. According to research, exposure to micro- and nanoplastics can cause oxidative stress, trigger cellular repair mechanisms, and mechanically harm organelles, interfering with several metabolic processes. Lung epithelial cells and other tissues have shown carcinogenic effects from both short- and long-term exposure [8].

Exposure to environmental contaminants is a major etiological factor in the development of lung cancer, according to numerous academic studies. Some literature has examined and documented the connections between lung cancer-causing variables and other contaminants, specifically microplastics, passive smoking, polycyclic aromatic hydrocarbons, radon, asbestos, cadmium, nickel, and pesticides [7,28].

Lung cancer has been identified as directly related to deteriorating ambient air quality, indoor pollutants, and occupational exposure. Outdoor PM2.5 exposure affects all human groups and is a significant carcinogen. Indoor passive smoking is also a major source of exposure. Asbestos exposure has also been identified as a high carcinogen. Priority should be given to innovative approaches to improving air quality, while simultaneously avoiding long-term exposure to pollutants. The long-term health consequences of air pollutants should be systematically investigated. The contributions of environmental factors to lung cancer need to be documented through comprehensive epidemiological studies from a public health perspective and shared with the public [7].

Smokers are vulnerable to microplastics, as they have been detected in 99% of cigarette filters. Findings have shown that microplastics are present in biological samples such as feces (100%), lung tissue (88%), blood (77%), and placenta (75%), indicating systemic human exposure to microplastics. Improper disposal of cigarette waste is a major contributor to microplastic pollution [27]. Cigarette filters are made of cellulose acetate, a type of plastic that is not biodegradable. Improper disposal of cigarette butts causes microplastic pollution. Studies have shown that cigarette butts contain heavy metals and toxic substances and can release chemicals into the environment, negatively impacting aquatic life and ecosystems [27,29].

Hospitals have a significant environmental impact due to waste generation. The increasing volume of medical waste is a major problem. Due to plastic waste, microplastics constitute a significant portion of medical waste. In addition to being widely used in daily life, plastics are an essential component of medical equipment. Many medical devices used in regular hospitals are either plastic-made or come in plastic packaging. In a study investigating microplastics in surgical environments, it was found that the abundance of environmental microplastics is highest during operating room working hours, and the most abundant polymer types are polyethylene terephthalate and polypropylene [30,31]. Further concerns about perioperative exposure are brought up by the existence of airborne microplastics in operating room settings. During surgical procedures, dispersed particle matter may rise due to surgical activity, staff mobility, and the heavy usage of synthetic materials. Surgical personnel and susceptible patients should be investigated for repeated exposure to airborne microplastics, even if the clinical significance is still unknown. Future elements of environmentally conscious surgical practice could include monitoring the composition of airborne particulate matter, evaluating the use of disposable materials, and optimizing operating room ventilation systems.

Discussing the possible effects of lung cancer growth linked to exposure to environmental microplastics on thoracic surgery practice is critical at this point. The biggest risk factor for lung cancer is still cigarette smoking. However, current beliefs on the origin of lung cancer may need to be reevaluated if long-term exposure to environmental microplastics is demonstrated to contribute to carcinogenesis. This change may contribute to the explanation of lung cancer in nonsmokers. Therefore, while assessing patients, thoracic surgeons may need to take into account occupational, environmental, and other exposure profiles in addition to smoking history.

Microplastic exposure may potentially affect the surgical patient profile and the biology of tumors. If lung cancers appear at a younger age, have different histological subgroups, and have more lesions in the periphery, surgical strategies may need to be altered. In this case, the importance of parenchyma-sparing procedures such as segmentectomy and sublobar resections may increase. Minimally invasive surgical techniques, such as video-assisted and robotic-assisted thoracic surgery, may become increasingly crucial in the treatment of these patients.

If microplastic exposure is determined to be a high-risk factor, new screening techniques may be considered for populations residing in high-risk areas, specific occupational groups, and individuals with substantial indoor exposure. Thoracic surgeons may take an active role in developing screening programs, standardizing surgical criteria, and developing algorithms for managing pulmonary nodules. Microplastic-induced microvascular damage, fibrotic changes, and chronic inflammation in lung tissue may affect the risk of prolonged air leakage, total postoperative complication rates, and postoperative recovery. In this case, more comprehensive preoperative pulmonary function assessments and individualized risk stratification may be necessary. The development of biomarkers to quantify the quantity of microplastics in lung tissue, microplastic analysis of surgical specimens, and the integration of environmental exposure data into the assessment of resection margins are possible long-term research avenues.

From the perspective of thoracic surgery, confirmation of a causal relationship between lung cancer development and exposure to environmental microplastics may lead to a substantial shift in clinical practice, including patient selection, surgical techniques, diagnostic approaches, and interdisciplinary collaboration. Large scale, long-term epidemiological studies that combine environmental exposure data with clinical outcomes are essential to clarify this connection. The lack of such data is now a significant limitation. However, the amount of microplastics in human lung tissue may be quantified thanks to advancements in analytical techniques, opening up new possibilities for exposure biomarkers and risk assessment.

By giving access to human lung tissue, thoracic surgery makes it possible to examine long-term environmental exposure inside the target organ. Surgically removed lung tissue may enable direct evaluation of long-term particle accumulation within the pulmonary parenchyma, in contrast to blood or bronchoalveolar lavage samples. In order to detect compounds that have accumulated in the respiratory system over decades, surgically removed samples can be essential biological archives. The creation of new biomarkers may be made possible by routine examination of the microplastic burden in excised lung tissue. In this situation, thoracic surgeons may play a crucial role in both environmental illness surveillance and treatment. Thoracic surgeons' professional roles may gradually change as environmental factors of lung illness become more widely recognized. Surgeons may become more involved in interdisciplinary collaborations combining pulmonology, environmental science, epidemiology, and public health by acting as observers of environmental pathology in addition to providing technical intervention.

Long-term exposure to inhaled microplastics can alter the biomechanical properties and tissue flexibility of the lung parenchyma, which is important for thoracic surgery, in addition to their carcinogenic consequences. Therefore, recognizing environmental exposure as a possible modulator of lung tissue quality may become more important in preoperative risk assessment and surgical planning. Changes in local immunological dynamics may affect lymphatic dissemination patterns, tumor invasiveness, and sensitivity to systemic medications used following surgery, according to surgical oncology. Future research examining resected lung specimens may shed light on the connection between ambient particle load and histopathological features. Together, these results and projections suggest that exposure to ambient microplastics should be taken into account as both an ecological concern and a possible risk factor for the development of postoperative disease. These advancements have the potential to change thoracic surgery from its current role in lung diseases to a proactive area that includes research initiatives, environmental risk assessment, surgical procedures, and participation in all of these processes.

There are studies in the literature that indicate microplastic involvement in the lungs and that this issue is very closely related to thoracic surgery, meaning that this topic should not be considered speculation.

In an autopsy study, Amato-Lourenço et al. found microplastics in human lung tissues. They found fibers (n = 4) and polymeric particles (n = 33) in 13 out of 20 tissue samples, with fibers ranging from 8.12 to 16.8

μm and all polymeric particles smaller than 5.5 μm. Following inhalation, these contaminants' varied properties were linked to detrimental health effects [1].

In the first study by Jenner et al., which reported the presence of microplastics in human lung tissue samples using µFTIR spectroscopy, the microplastic concentration in the obtained samples was significantly higher than in empty samples, supporting environmental exposure through human respiration. This study emphasized the detection of microplastics as small as 4 µm and even >2 mm. This finding provides significant information regarding the presence of microplastics in human lung tissues. In this study, 39 microparticles were detected in 11 out of 13 lung tissue samples, and the average microparticle/tissue ratio was reported as 1.42 ± 1.50 [13].

In the study conducted by Liu et al., evidence was presented that metals and microplastics complexes accumulate in human lung adenocarcinoma tissues and are associated with poor clinical outcomes. This study used a multi-technique approach on tissue samples, including laser direct infrared imaging, pyrolysis-gas chromatography-mass spectrometry, inductively coupled plasma mass spectrometry, and Single-cell RNA sequencing, and identified 34 different microplastic species in lung tissues. Transcriptome-level sequencing and The Cancer Genome Atlas validation revealed that metal-microplastic complex accumulation activates pro-tumorigenic pathways [2].

Ernhofer et al. emphasized that polystyrene micro and nanoplastic particles did not significantly affect the malignant behavior of cancer cells, but these small particles could promote malignant characteristics, mostly in normal lung cells, by causing DNA damage, oxidative stress, migration, and mitogenic pathways [32].

Studies on microplastics in operating room settings are quite rare. In their study, Field et al. found that polyethylene terephthalate, polypropylene, polyethylene, and nylon were the most common polymer types found in operating rooms. They were unable to detect microplastics outside of operating room hours. These results indicate that this new route of contamination through surgery represents a potentially new exposure route for humans [30].

In a study on microplastic concentration in lung tissue conducted by Wang et al., lung tissue samples were taken from 12 non-smoking patients to evaluate the characteristics of microparticles in human lung tissues using an Agilent 8700 laser infrared imaging spectrometer and scanning electron microscope. They detected 108 microparticles encompassing 12 types in the lung tissue samples. The microparticles in the lung tissues consisted of polypropylene, polyethylene terephthalate, and polystyrene, and a correlation was found between microplastic exposure and certain blood test indicators. This correlation was noted as positive with platelets, thrombocytocrit, and fibrinogen [33].

These findings suggest that linking environmental microplastic exposure to lung cancer and the aforementioned claims regarding thoracic surgery practices are a more serious matter than mere speculation.

In conclusion, there is evidence to support the hypothesis that oxidative stress, persistent inflammation, epithelial and DNA damage caused by inhaled microplastics may result in lung cancer. Even if a causal relationship has not yet been conclusively shown, the consistency of mechanistic findings highlights the necessity for improved preventive measures and collaborative research efforts. In addition to highlighting the significance of integrating environmental health perspectives into future thoracic surgery diagnostic, therapeutic, and preventive approaches, this review aims to increase awareness of environmental microplastic exposure. Furthermore, beyond the environmental impacts of microplastic exposure, it is conceivable that it could alter the epidemiology, tumor biology, and postoperative outcomes of lung cancer, potentially affecting future thoracic surgery practices.

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