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Review Article

Lung aging and climate exposures: Molecular processes and consequences for enhancing pulmonary health

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Abstract

Worldwide, inhaled environmental exposures result in more than 12 million fatalities annually. Air pollution and tobacco use continue to be the major public health issues causing the rising incidence of respiratory illnesses, even in the face of regional initiatives to limit environmental exposures. This review describes how environmental exposures accelerate lung aging and cause lung illness. Silica and coal mining, tobacco, and fossil fuel usage induce lung inflammation and oxidative stress. DNA damage, epigenetic instability, mitochondrial malfunction, and cell cycle arrest in lung progenitor cells, which are crucial for lung expansion, can result from long-term oxidative stress. Consequently, vital healing processes are compromised, causing the lung parenchyma to prematurely degrade. By harming the lungs and the cells that repair wounds, inhaled environmental exposures hasten the aging process of the lungs. Taking steps to lower exposure to harmful antigens is important for improving lung health, and more research is needed to learn more about treatments that might slow down or stop lung aging before it starts.

Keywords: Lung inflammation, Oxidative stress, DNA damage, Mitochondrial dysfunction, Respiratory illnesses

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1. Introduction

Inhaled environmental chemicals and pathogens are constantly interacting with the human lung. The aging process of the lungs is influenced by both heredity and environmental factors. As a result, the respiratory tract undergoes structural remodelling, leading to a gradual decline in lung function.[1] Standard airflow from the trachea is via the bronchi, which divide into bronchioles and, finally, into groups of alveoli. Near a little interstitial gap, beneath the basement membrane and epithelial layer that encircle the alveoli, is the pulmonary capillary network.[2] In the interstitial space,[3] elastic and collagen fibres make up the extracellular matrix. These fibres keep the structure of the lung in good shape. Lung connective tissue weakens with age, leading to early collapse of the tiny airways and gradual dilation of the airspaces.[4] As a result, oxygenation and exercise capacity decline with age due to a reduction in the lung's surface area accessible for gas exchange.[5] These changes are even more clear in age-related lung diseases like COPD (characterized by increased mucus production and damage to the alveolar wall) and IPF (characterized by interstitial fibrosis). The lung changes shape as we age because of things we breathe in that hurt the lung epithelium and the tissue underneath it.[8] To avoid pathological remodelling and keep the lung's structure intact, effective repair mechanisms are essential. In order to aid in the healing process, specialized type II alveolar epithelial cells produce new type I epithelial cells upon damage. These cells then cover the majority of the alveolar surface.[9] Multipotent mesenchymal stem cells, which are abundant in subepithelial lung tissue and may develop into several cell types, including epithelial cells, macrophages, and reparative fibroblasts, also play a role in wound healing.[10,11] The extracellular matrix in the interstitial space

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of the lung is fixed by pulmonary fibroblasts so that the alveolar architecture can be restored and kept.[6] In spite of this, stopping lung progenitor cells and cellular repair processes changes the structure of the lungs in a way that isn't normal. This leads to early loss of pulmonary function.[7] Deterioration of cellular repair pathways is a hallmark of aging in the lung (**Figure 1**). As a result of oxidative stress, DNA damage, epigenetic instability, telomere attrition, mitochondrial injury, and protein homeostasis issues in important stem cells and structural cells, the lung's ability to recover itself is reduced by inhaled exposures.[12,13] A buildup of damage promotes cellular senescence and cell death in mesenchymal stem cells and stem cell loss in type II alveolar epithelial cells and lung fibroblasts.[13] Due to cell-specific senescence, which means that cells stop growing and work less well, lung damage can show up in a number of way.[14] For example, when the lungs are hurt, senescent fibroblasts make abnormal collagen in the extracellular matrix,[15,16] and senescent alveolar epithelial cells can't help the re-epithelialization process. Inhaled pollutants cause inflammatory oxidative stress that builds up over time, which in turn causes premature lung aging and cellular senescence in the lungs.[17,18] Environmental exposures can have an effect on lung health, and this review summarizes those risks and information. Environmental exposures hasten lung aging and the processes and biochemical routes by which this happens.

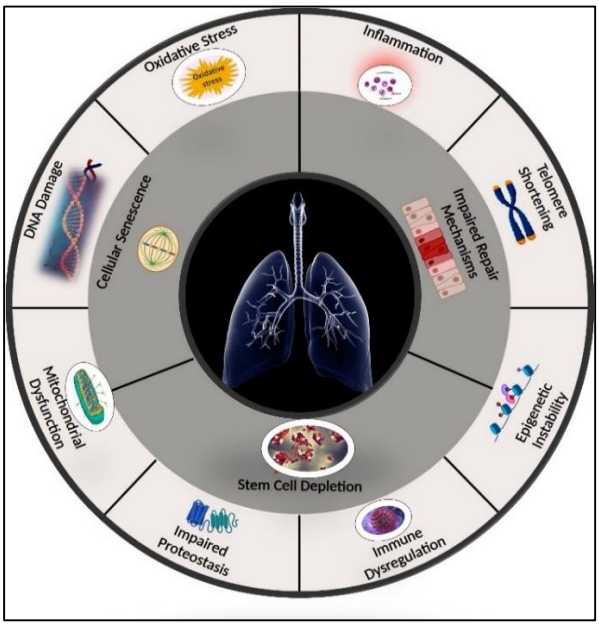

Figure 1: Summarizes the main ways environmental exposures age the lungs. After inhaling toxic chemicals, many lung cells experience oxidative stress, inflammation, DNA damage, mitochondrial dysfunction, epigenetic instability, immunological dysregulation, and poor proteostasis. Thus, pulmonary stem cells die, progenitor cells age, and repair pathways are damaged, causing early lung aging.

2. Materials and Methods

A literature search was conducted on various database sources (like science direct, PubMed) with the help of the combination of different keywords: Lung Aging and Climate Exposures. The search was customized by applying the appropriate filter so as to get the most relevant articles to meet the objective of this review. The various numbers of papers are present on the Pulmonary Health for the newly prevention approaches to determine their better Pulmonary Health activity.

Table 1: Inclusion and exclusion criteria for a cross-sectional multicenter study of patients with COPD in Latin America.[54]

Inclusion criteria	Exclusion criteria
Adults ≥40 years of age Diagnosis of COPD at least for 1 year	• Diagnosis of sleep apnea or any other chronic respiratory disease Any acute or chronic condition that would limit the ability of the patient to participate in the study
At least one spirometry in the last year with a postbronchodilator FEV1 /FVC < 0.70	Any acute or chronic condition that would limit the ability of the patient to participate in the study
Current or former smokers (> 10 pack-years)	Refusal to give informed consent
Stable disease (no recent exacerbation)	

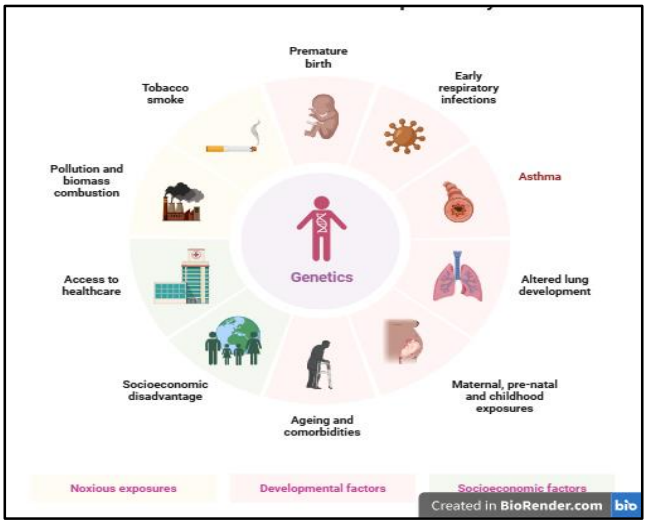

Figure 2: Risk factors for chronic obstructive pulmonary infections

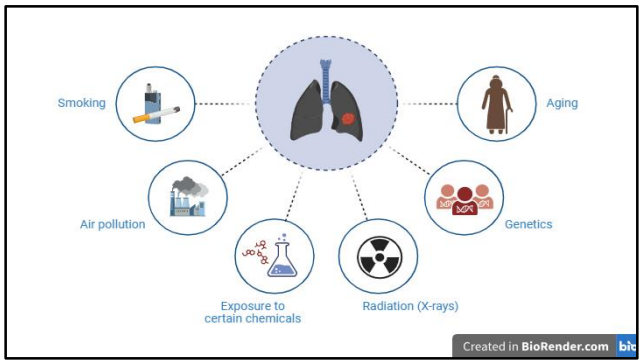

Figure 3: The lung's biological response to environmental factors

2.1. Tobacco

When you light up a cigarette, hundreds of harmful chemicals, such as carbon monoxide, hydrogen cyanide, and polycyclic aromatic hydrocarbons, are released into the air.[19] When exposed to air, many of the chemicals in cigarette smoke undergo chemical reactions that produce free radicals and block the action of antioxidants.[20] Because it burns toxic chemicals and makes harmful reactive oxygen species (ROS), tobacco smoke does a lot of damage to tissues that is similar to biological aging (**Table 2**).[21] Lung cells and circulating leukocytes have their DNA methylation levels changed by tobacco smoke at the molecular level.[22,23] Nicotine inhibits the activity of DNA methyltransferases, and the oxidative stress that smoking produces leads to DNA demethylation. To cytosine-phosphate-guanine (CpG) sites, these enzymes attach methyl groups.[24,25] This means that total DNA methylation is different between smokers and nonsmokers, as shown in earlier epidemiological studies.[26] Chronic exposure to second-hand smoke may increase the risk of smoking-related diseases via altering DNA methylation in gene-coding areas.[22] Scientists discovered that methylation of the Bcl-2 promoter was enhanced by cigarette smoke in a mouse model.[27] Cytoplasmic methylation causes a decrease in Bcl-2 expression, leading to cell death in the alveolar wall's early emphysema forms. Besides this, smoking lowers DNA methylation at the AHRR gene in tissues like blood and lungs, according to studies on people.[28-30] When you smoke, your AHRR methylation levels drop, which stops your body from making enzymes that clean up harmful substances like the hydrocarbons in cigarette smoke.[31] This suggests that AHRR demethylation brought on by smoking could be a cause of early lung illness in smokers.[32] Levels of DNA methylation are a strong indicator of biological aging, according to recent research.[33]

Table 2: An overview of how smoking causes alterations that hasten lung agin.[34-38]

S.No.	Scientist/Years	Changes associated with becoming older	Results	Physiologic effect associated with age-related change
1	Scientist Joehanes et al.[23] (2016)	Uncertainty in epigenomics	Cigarette smoke changes the amount of methylation at thousands of CpG sites in DNA, which controls the expression of more than 7,000 genes.	Modifications to DNA methylation caused by smoking impact the expression of genes that are associated with early emphysema.
2	Scientist Bradley et al.[3] (2023)	Proteostasis gone haywire	The endoplasmic reticulum of lung cells experiences decreased protein folding as a result of tobacco use.	Alveolar wall breakdown is triggered by the accumulation of defective proteins, which impede surfactant protein formation.
3	Scientist Walters et al.[48] (2008)	Termination of telomeres	Cell cycle arrest occurs in lung epithelial cells, basal progenitor cells, and fibroblasts as a result of tobacco smoking's effect on telomere length.	Alveoli and airway architecture and function are disrupted due to abnormal cell differentiation and defective epithelial remodeling.
4	Scientist Goldfarbmuren et al.[16] (2020)	A state of inflammation	An airway epithelial-mesenchymal transition is induced by persistent smoking-related inflammation.	Pulmonary fibrosis is caused by transformed mesenchymal cells, which create an unorganized extracellular matrix.
5	Scientist Bhat et al.[2] (2009)	Immune system imbalance	The phagocytosis of microorganisms by macrophages and the immunological responses of B and T cells are both dampened by tobacco smoking.	Respiratory tract infections, which can cause structural alterations in the lung, are caused by dysregulated immunity and can last for lengthy periods of time.

3. Air Pollution

3.1. Ceramic industry

A 30-year mortality and respiratory morbidity study of refractory ceramic fibre workers did not find an increase in SMR for lung cancer. Similarly, Meijers and colleagues have found that silicosis in the ceramics sector is associated with an increased risk of lung cancer. That this technique does not damage DNA is supported by this. There was no statistically significant correlation between cumulative dose response and lung cancer as determined by the researchers in their study of silica exposure. Additionally, they did not see an uptick in fatalities among the ceramic workers as a whole or due to any particular reason. Silica exposure is likely only indirectly linked to lung cancer in most people; however, the dosage may differ between those who are silicosis and those who are not.

3.2. Coal gasification and coke production

A significantly increased risk of lung cancer was seen among workers in the coal/coke and associated product sectors (RR = 1.55, 95% CI = 1.01-2.37). Gasifying coal was shown to raise the risk of lung cancer, according to researchers (93, 94). Many different chemicals pose a threat to workers in the coal gasification industry. These include polycyclic aromatic hydrocarbons (PAHs), silica, arsenic, cadmium, lead, nickel, hydrocarbons, sulfuric acid, sulphur dioxide, aldehydes, and many more. To protect themselves against PAH aerosols, workers on British coke furnace roofs were mandated to wear helmet respirators, namely the Airstream helmet.

3.3. Building sector

According to NIOSH, nearly 70 different compounds may expose U.S. construction workers. Although several epidemiological studies failed to discover the relationship, a large number of them reported that construction workers had a substantially higher death rate for lung cancer. Among the construction trades that have reported an elevated mortality rate for lung cancer are roofers, water proofers, allied workers, insulation workers, carpenters, painters, operating engineers, and bricklayers. The risk of lung cancer increased with increasing exposure time for construction workers, according to one study. Due to lower levels of exposure, supervisors, engineers, and higher-ranking officials in the construction industry have not experienced this danger**.**

3.4. Mason

A number of lung carcinogens, librettos and crystalline silica, are potentially present in the work environment of bricklayers. For this reason, bricklayers and other craft workers are more likely to get lung cancer and other ailments linked to inorganic dust. There was a statistically significant increase in the SMRs and PMRs for lung cancer among these employees (158 SMRs; 130–190 PMRs; 110,112). Researchers in Italy found that bricklayers had a higher chance of developing lung cancer compared to the general population (147 cases vs. 81 controls; odds ratio 1.57; 95% CI: 1.12-2.21). An increased risk of squamous cell carcinoma (OR 2.0333; 95% CI: 1.32-3.13, 56 exposed cases) and small cell carcinoma (OR 2.29; 95% CI: 1.29-4.07, 21 exposed cases) was associated with bricklayers, despite a lower frequency of adenocarcinoma compared to the general population.

3.5. Visual-artist

There is growing worry about the potential health implications of exposures to paint, which affect a huge number of workers. Although they are still used elsewhere, several nations have decreased or removed the usage of some harmful compounds in paint, such as benzene, phthalates, chromium, and lead oxides. Research has demonstrated an increased risk of cancer, specifically lung cancer, among artists (124, 125). Artists had a 1.3 (95% CI: 0.9-2.2) higher risk of developing lung cancer, according to research by Raman Kumar et al. When it comes to exposures, the odds ratios were 1.695% CI: 1.0–2.3, 1.3 (95% CI: 0.9–1.7), and 1.1 (95% CI: 0.8–1.6) for metal coatings. This means that jobs that involve paint, especially those that use wood varnishes and stains, raise the risk of lung cancer.

3.6. Rubber industry

Researchers at the International Agency for Research on Cancer have shown that rubber factory workers are at increased risk of developing cancer. Tires and other rubber products rely heavily on the carcinogenic chemical's benzene and 1,3-butadiene. Tires and rubber products are made using a variety of chemical compounds, some of which have carcinogenic effects on people. A new meta-analysis of observational data published up until April 2016 on occupational exposures in the rubber manufacturing industry found a practically statistically significant higher risk of cancer (SRR = 1.08; 95% CI: 0.99-1.17).

3.7. Sandblasting

Workers in the foundry, masonry, sandblasting, and pottery industries are exposed to crystalline silica. Exposure to silica has recently been linked to lung cancer, in addition to its well-known association with silicosis. Inhaled crystalline silica from occupational sources is now officially a known human carcinogen, according to the International Agency for Research on Cancer. Found that there were 60% higher instances of lung cancer in the silica-exposed group compared to the general population in the US (95% CI: 1.31-1.93). Because of the increased incidence of illnesses connected to silica exposure (133), there were more fatalities in this group from all causes (SMR = 1.23, 95% CI: 1.16-1.31). In a case report 134, Güngen et al. described the development of lung cancer in a 35-year-old female worker who had been engaged in denim sandblasting for 18 months. The inflammatory mediator LTB4 and its receptor BLT1 play

a crucial role in promoting the growth of lung tumors, which are induced by silica, according to scientists.

3.8. Drivers and vehicles

Working for a long time in the trucking industry, where diesel exhaust (DE) is a frequent pollutant, raises the risk of lung cancer. According to the IARC, DE is very probable to cause cancer in humans. The epidemiologic data comes from studies on lung cancer in laborers who operate trucks, buses, shipyards, and railroads. The excess risk throughout a person's lifetime that found was 136 times higher than the Occupational Safety and Health Administration's (OSHA) authorized 1 per 1,000 hazards. A similar study by Garshick et al. found that out of 31,135 male workers in the unionized U.S. trucking sector, there was an elevated incidence of lung cancer. Numerous critical reviews and epidemiologic meta-analyses support the notion that DE exposure raises the risk of lung cancer.

3.9. Traffic police

Officers whose job it is to guide or drive traffic in areas with more people tend to put in long days. Carcinogenic airborne contaminants, including benzene, PAHs, and persistent organic pollutants like dioxin, can be released into the air during these operations. The exposure to benzene among the officers was three to five times higher than the control group. Pollutants in the air, of which particulate matter (PM) is a key component, can lead to edema, inflammation, cell death, mutations in the respiratory tract, and oxidative DNA damage. Most law enforcement professionals are not expected to be exposed to these chemicals frequently or to a large level, especially with the right training and safety measures in place.

3.10. Contact with certain substances

Industrial and manufacturing chemicals, pesticides, and aflatoxin may elicit particular triggers in our homes and workplaces, according to strong evidence. Farmers and public health personnel are most exposed to these pesticides during handling, dilution, and application. Skin and respiratory pathways are the major routes of exposure during application. Some studies relate occupational pesticide use to lung cancer. The occupational hazard ratio was greater for those who were exposed to three pesticides. compounds the most during their lifetime, according to Bonner et al. This increased lung cancer risk Alavanja et al. examined the connection between 43 pesticides and 654 lung cancer cases in the Agricultural Health Study (AHS), which comprised 57,3100 pesticide applicators from Iowa and North Carolina, after 10 years of extended follow-up. The scientists believe chlorimuron ethyl and parathion may be linked to lung cancer in the AHS for the first time and should be researched further. The preceding study and others support the idea that pesticides cause lung cancer. Chlorophenols (CPs), dioxins, and related phenoxyacetic acids (PAs) can cause lung cancer. Numerous occupational lung cancer investigations have found a wide range. A meta-analysis of five cohort studies and six reports examined CPsR plant workers' lung cancer causes. Collins et al. examined two CPs exposure studies. IARC Multicentred Analysis includes 36 papers from earlier reports. The meta-analysis included 27,865 CPsR producers. The final analysis has five articles and six reports. The random model's lung cancer SMR was 1.18 [95% CI: 1.03–1.35, P=0.014]. Many studies reported no statistically significant association between pesticide exposure, strength, or total years exposed and lung cancer mortality in smokers or nonsmokers.

3.11. Exposure to radiation

The Urban Air Toxics Strategy lists arsenic compounds as one of 33 hazardous air pollutants that pose the highest public health risk in urban environments. Potential occupational carcinogens include inorganic arsenic compounds. OSHA has developed extensive recommendations to avoid drug exposure in healthcare settings.

3.12. Beryllium (157)

The DOE's Chronic Beryllium Sickness Prevention Program safeguards workers from beryllium exposure and sickness. EPA and Clean Air Act National Emission Standards treat beryllium compounds as hazardous air-pollutants.

3.12. Chrome (158)

The U.S. EPA limits total chromium in drinking water to 0.1 mg/L. The FDA regulates bottled water chromium levels to 0.1 mg/L.

3.13. Nickel (159)

The U.S. EPA requires controls for mobile-source air toxics such as nickel compounds. National Emission Standards for Hazardous Air Pollutants classify nickel and its compounds as air pollutants. According to the Urban Air Toxics Strategy, nickel compounds are one of 33 hazardous air pollutants that pose the biggest public health risk in urban environments.

3.14. Genetics

In 2006, Pauline L. Lee et al.[25] looked into whether variations in genes related to innate immunity make people more likely to get infectious and non-infectious lung diseases like tuberculosis and sarcoidosis, and may also be a risk factor for inhalation anthrax. 45 non-synonymous polymorphisms in ten genes—p47phox (NCF1), p67phox (NCF2), p40phox (NCF4), p22phox (CYBA), gp91phox (CYBB), DUOX1, DUOX2, TLR2, TLR9, and alpha 1-antitrypsin (AAT)—did not make people more likely to get lung disease in a group of 95 people with and without lung disease.[39]

3.15. Aging

Cellular damage accumulates with aging, reducing tissue function and increasing sickness and death. Older people's lungs show genomic instability, telomere attrition, epigenetic alterations, proteostasis loss, mitochondrial dysfunction, cellular senescence, stem cell exhaustion, intercellular communication changes, and ECM dysregulation. The eleven markers of aging are connected to older lung diseases. As people age, lung environment and mechanical function dramatically impact breathing and infection risk. Reducing lung elasticity with age is the main lung function decrease. A feedback loop stiffens lungs by changing ECM protein expression. Alveolar duct dilatation and air space expansion due to collagen fiber network modifications reduce alveolar surface tension and lung compliance. Additionally, respiratory muscle strength and function decline, limiting ventilation and chest wall compliance. Lower expiratory and inspiratory strengths impact respiration. Older people's rib cages stiffen, reducing thoracic cage expansion as they breathe. Aging lung mucociliary clearance function diminishes, causing pneumonia. Due to age-related lung structural changes, healthy seniors are unlikely to develop clinically significant abnormalities, but those who are compromised or have a lower respiratory tract infection may. Single-cell molecular profiling identified over 40 lung cell types. Several hazardous chemicals in lung air may change cellular composition and function over time. The main problem with aging lungs is poor tissue repair and remodeling by epithelial, fibroblast, immunological, and progenitor cells, which change with age. Alveolar epithelial type-2 cells (AEC2) decrease regeneration potential with age. AEC2 loss lowers progenitor cell AEC1 numbers. Alveolar changes stimulate fibroblast growth and airway remodeling. As humans age, senescent lung cells increase. Senescent-associated secretory phenotype (SASP) changes the environment around these cells, affecting nearby cells. Fibroblast senescence has been connected to pulmonary remodeling and age-related lung disorders such pulmonary fibrosis. It's unclear how senescence affects the aging lung. Aging increases senescent cell growth and geriatric infection risk. Cellular aging and lung bacterial ligand expression increase pneumococcal pneumonia susceptibility.[41]

4. Methods to Enhance Air Quality and Respiratory Hygiene

The onset of age-related respiratory disorders is accelerated by environmental exposures, which also hasten the aging of the lungs. When you breathe in antigens, they cause inflammation and oxidative stress, which damage and scar the lung parenchyma. This leaves the lung's structure permanently changed. In order to improve lung health and avoid aging-related lung illnesses, it is necessary to analyze hazardous environmental exposures and take measures to limit them. Exposure to ambient air pollution can be decreased in a number of ways. Reducing the amount of indoor air pollutants is possible with filters, whether they are central or portable. Indoor air contaminants might include organic dust and substances released while cooking. Clinical trials found that HEPA in-duct filtration systems improved microvascular health, a sign of lung health, and reduced particle numbers in the indoor air. Air conditioning and heating, ventilation, and air filtration systems help passengers in cars and buses reduce their exposure to particulate matter during travel. To further reduce exposure to air pollutants, it is recommended to refrain from strenuous outdoor activities during periods of high concentrations. Networks that track air pollution, like the Air Quality Index maintained by the EPA, calculate concentrations of dangerous air pollutants and use mathematical models to predict future levels of air pollutants. Reducing strenuous outdoor exercise and other outdoor activities might help lower intake of hazardous particles when air pollution levels are high. It is possible to lessen harmful exposures by limiting time spent in microenvironments with high pollution levels. If you want to keep your lungs in top shape, you should, for instance, stay away from busy roadways. Similarly, in order to protect the lungs of smokers and those around them, it is crucial that they stop smoking and eliminate all sources of secondhand smoke. Helping people quit smoking and reducing second-hand smoke pollution are two of many proven strategies. Minimizing dust exposure is the most effective technique for preventing occupational exposures to substances including asbestos, silica, and coal dust. Individual preventative measures, such as using a mask and wiping dust off skin and clothes, may assist in lessening hazardous exposures; however, engineering controls, such as dust extraction systems, are the primary means of lowering exposures. Miners who have x-rays showing they have lung disease are legally allowed to work with better dust protection. This means that secondary prevention methods, such as checking high-risk individuals for lung function loss and early x-ray signs of disease, can help keep serious diseases from happening. Immunizations against influenza, pneumococcal, and COVID-19 are examples of primary preventative methods that can lower the risk of respiratory infections and improve lung health in people who are at a high risk because of the things they breathe in. To prevent serious respiratory infections, it is suggested that those over the age of 65, especially those with chronic medical issues, keep up with their vaccines. There is some evidence from previous studies that dietary and pharmaceutical therapies can lessen sensitivity to inhalation exposures. Taking nonsteroidal anti-inflammatory drugs (NSAIDs) slowed down the loss of lung function after being exposed to particulate matter (PM), and a small study found that taking NSAIDs slowed down the loss of lung function after being exposed to ozone. Taking extra B vitamins also lowered changes in DNA methylation levels caused by PM2.5 in a small study of adults. This shows that B vitamins may be able to fight the oxidative stress that PM causes. A research study on young individuals exposed to ozone found that dietary antioxidants, such as vitamin C, also

protected lung function. Transplanting stem cells made from adipose tissue reduced inflammation in mice caused by PM2.5. This shows that stem cell treatments could be a useful way to reduce pollution in the future. Pharmacological, biological, and behavioural therapies to reduce or reverse the effects of ambient inhalation exposure need more study, although early results are encouraging.[40]

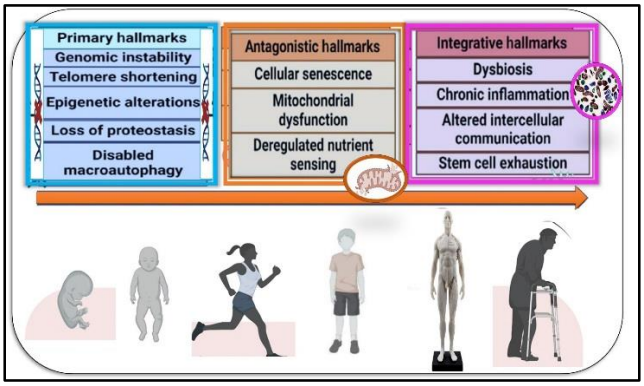

Figure 4: We divide the twelve aging markers into three groups. Genome and organelle damage are the main markers. They include genomic instability, telomere shortening, epigenetic changes, proteostasis loss, and macroautophagy impairment. Senescence, mitochondrial malfunction, and unregulated nutrition sensing are the opposing characteristics of cell injury. Finally, integrative characteristics occur when the organ cannot adjust for primary and antagonistic impairments. These include dysbiosis, persistent inflammation, disrupted intercellular communication, and stem cell depletion.

4.1. Future-perspective

We can implement effective, feasible, and cost-effective environmental and occupational interventions to reduce the occurrence of lung cancer. Research and understanding point to a specific environmental and occupational risk factor for lung cancer, but many developing nations have failed to take action. Many developing nations, including India and China, continue to widely utilize asbestos. There are regulations on the use of tanning beds in some nations.[42-45] The government of many nations does not have a strategy in place to avoid occupational lung cancer, and there are no guidelines on occupational dangers put forth by their occupational health institutes. The issue lacks priority.[46] Has to be a studied on the effects of air pollution on lung cancer, specifically how much it is and ow it changes with the seasons.[47] There is a lack of data on the differences in pollution exposure among nations and cities.[48] Particularly in the developing world, there is a need for robust systems and approaches to limit exposure to chemicals used in ignorance. Additional study is required to fully understand the genetic predisposition and environmental risk factors for lung cancer; however, several studies have shown these aspects.[49,50] Environmental pollution is associated with an elevated incidence of lung cancer.[51,52] Prioritizing policies that lower pollution levels are essential. We should monitor and evaluate data on carcinogenic risk factors for the chemical and commercial sectors. Products should undergo testing before being brought to the market. Further investigation is necessary to understand the risk of lung cancer in non-smokers exposed to environmental pollutants.[53]

4.2. Strengths and limitations

The scoping review was reported using PRISMA guidelines. The team and research librarian collaborated on developing search phrases. Scoping reviews gather data from diverse study designs and techniques, ensuring rigor and transparency.

A scoping review of both qualitative and quantitative data on the challenges, strategies, and ways to measure physical activity in COPD patients will help researchers better understand their behaviour and offer additional information for clinical practice. Furthermore, this scoping review has significant limitations. We limited our search to Chinese or English literature, missing crucial data from related works in other languages. This review did not conduct a quality evaluation of RCTs. The sample size was insufficient to reflect the entire population. Further study will focus on monitoring new material and examining literature in different languages to better understand the physical activity behavior of COPD patients**.**

5. Conclusions

Trying different lung cancer prevention methods may lower mortality and health care costs. To reduce lung cancer risk and its medical, personal, economic, and societal effects, physical, chemical, and biological exposures to recognized carcinogens should be drastically reduced. This should include workplace environmental initiatives. To deal with the many environmental carcinogens that people can't do much about, we need a multi-sectoral, collaborative effort that includes everyone. This is needed on a national, regional, and even global level to protect public health. The rising number of cases of lung cancer in those who do not smoke highlights the critical need for these therapies. We need to learn more about the environmental and occupational factors that lead to lung cancer so that we can come up with a plan for primary prevention and include exposures at work and in the environment in the global cancer agenda. This can only be achieved by first identifying the current state of affairs and the obstacles that stand in the way.

6. Source of Funding

None.

7. Conflict of Interest

None.

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