Review

The Impact of Ambient Environmental and Occupational Pollution on Respiratory Diseases

Chinatsu Nishida and Kazuhiro Yatera *

Department of Respiratory Medicine, University of Occupational and Environmental Health,
Fukuoka 807-8555, Japan; c-nishi@med.uoeh-u.ac.jp
* Correspondence: yatera@med.uoeh-u.ac.jp; Tel.: +81-93-691-7453

Abstract: Ambient pollutants and occupational pollutants may cause and exacerbate various lung and respiratory diseases. This review describes lung and respiratory diseases in relation to ambient pollutants, particularly particulate matter ($PM_{2.5}$), and occupational air pollutants, excluding communicable diseases and indoor pollutants, including tobacco smoke exposure. $PM_{2.5}$ produced by combustion is an important ambient pollutant. $PM_{2.5}$ can cause asthma attacks and exacerbations of chronic obstructive pulmonary disease in the short term. Further, it not only carries a risk of lung cancer and death, but also hinders the development of lung function in children in the long term. It has recently been suggested that air pollution, such as $PM_{2.5}$, is a risk factor for severe coronavirus disease (COVID-19). Asbestos, which causes asbestosis, lung cancer, and malignant mesothelioma, and crystalline silica, which cause silicosis, are well-known traditional occupational pollutants leading to pneumoconiosis. While work-related asthma (WRA) is the most common occupational lung disease in recent years, many different agents cause WRA, including natural and synthetic chemicals and irritant gases. Primary preventive interventions that increase awareness of pollutants and reduce the development and exacerbation of diseases caused by air pollutants are paramount to addressing ambient and occupational pollution.

Keywords: ambient pollution; occupational pollution; respiratory disease; lung disease

1. Introduction

Air and occupational pollution are well known to negatively affect human health and cause chronic inflammation [1], leading to the development and exacerbation of respiratory diseases. Various human health issues have recently been raised due to urbanization, industrialization, population growth, climate change, and aging of the working population [2,3]. According to the World Health Organization, air pollution accounts for >6.5 million deaths worldwide per annum, equating to 11.6% of all global annual deaths [2]. Air pollution, particularly particulate matter$_{2.5}$ ($PM_{2.5}$) (≤ 2.5 μm in aerodynamic diameter), is a leading cause of several adverse health effects, including pulmonary diseases, and leads to 3.3 million premature deaths per annum worldwide [3]. Occupational air pollution is another crucial public health issue. Exposure to respirable dust, fumes, vapors, gases, volatile organic compounds, chemicals, and metals is hazardous and may cause occupational lung diseases [4]. Occupational lung diseases include diverse diseases such as asthma, chronic obstructive disease (COPD), pneumoconiosis, hypersensitivity pneumonitis (HP), and malignant diseases (lung cancer and malignant mesothelioma), with work-related asthma (WRA) being the most common [5]. Air pollution and occupational air pollution can be avoided, and primary prevention is critical for their control and prevention.

This review describes lung and respiratory diseases in relation to ambient pollutants, particularly $PM_{2.5}$, and occupational air pollutants, excluding communicable diseases and indoor pollutants, including tobacco smoke exposure. The aim of this review was to understand the current knowledge of environmental and occupational pollution and

Int. J. Environ. Res. Public Health **2022**, *19*, 2788

2 of 12

their adverse health effects based on recent information, as well as to highlight their effects on respiratory medicine and occupational health and increase awareness of active preventive interventions.

2. Methods

A search of the peer-reviewed English literature was conducted using MEDLINE (PubMed). The search was limited to reviews, systematic reviews, meta-analyses, randomized controlled trials, clinical trial, and books and documents published between 1 January 2017, and 1 December 2021, using the following search terms: (environmental) AND (occupation) AND (pollution) AND (lung OR respiratory OR pulmonary). A total of 275 extracted studies, 20 studies were screened by title and abstract, then 13 studies were extracted by full text, and eventually 8 studies were included in this review.

3. Ambient Pollution and Occupational Pollution

3.1. Ambient Pollution

Environmental pollution, including air pollution (ambient indoor and outdoor pollution), water pollution, and soil pollution, is well known to cause various health effects in humans. Air pollution can damage almost any organ system and cause acute or chronic lung and heart diseases [1,6]. Air pollutants are defined as any ambient substances that may be harmful to humans, animals, plants, or materials [6]. Such pollutants emerge from various sources, including industry, transportation, soil, volcanoes, and the ocean, each of which has different characteristics depending on its components, such as biological materials and aeroallergens [1,6]. In addition, air pollution is a complex mixture of different gaseous and particulate components, the composition of which varies widely by region and season [7]. However, it is difficult to unequivocally distinguish between particulate and gaseous matter, such as the transition from gaseous materials to particles or from particles to gaseous materials, depending on atmospheric conditions [8]. Typical air pollutants are composed of particulate matter such as $PM_{2.5}$ or PM_{10}; gases such as SO_2, NO_2, O_3, CO, reactive hydrocarbons, and polycyclic aromatic hydrocarbons (PAHs) such as naphthalene or anthracene; and volatile organic compounds (VOCs) such as toluene, xylene, and ethyl acetate, which are generally produced by industrial or transportation sources [1,6,9]. Over the last few decades, while emissions and air concentrations of traditional industrial air pollutants such as SO_2 and smoke particles have declined in developed regions, problems with the emissions of vehicle engine combustion products such as VOCs, NO, and fine particulates from vehicle exhaust and secondary ozone are persisting and even increasing [7].

$PM_{2.5}$ is considered to be the most harmful PM and is subject to several air pollution regulations [1,10]. $PM_{2.5}$ is a PM with an aerodynamic diameter of <2.5 μm [11] and includes several pollutants: heavy metals, PAHs, unburned or incompletely burned fuel gases, sulfur compounds, and nitric compounds [12]. $PM_{2.5}$ includes PMs that are directly produced by combustion (so-called 'primary particulates') and those that are produced, largely from primary pollutants, by chemical reactions in the atmosphere (known as 'secondary particulates'). Sources of primary particulates include facilities that generate soot; smoke induced by boilers and incinerators; and dust (fine dust) and mineral deposits created by coke ovens, automobiles, ships, aircraft, soil, and oceans. There are also natural gaseous and dust materials created by volcanoes and their cross-border pollution. Secondary particulates are produced from sulfur oxides (SO_x) and nitrogen oxides (NO_x) emitted by fuel combustion in thermal power plants, factories, business establishments, automobiles, ships, aircraft, and households, and from gaseous substances, such as VOCs emitted from evaporation from solvents, petroleum handling facilities, forests, and fuel combustion facilities, by reacting with light and ozone in the atmosphere [8]. Gaseous and particulate materials are also produced from smoking, cooking, and stoves in indoor environments.

$PM_{2.5}$ can deeply penetrate the lungs with a size threshold of deposition in the alveoli [1,10], which may initiate harmful airway effects, including airway oxidative stress,

lung and systemic inflammation, cilia dysfunction that increases the risk of infectious diseases, and increased airway hyperresponsiveness [10]. The harmful effect of PM generally depends not only on the particle size, but also on its structure and composition. There may also be toxic components on the surface of the particles, which can induce tissue damage. Toxic elements (e.g., arsenic, lead, or cadmium) or compounds (e.g., sulfuric acid or polycyclic aromatic hydrocarbons) can adhere to the surface of the PM during the combustion process and be carried deep into the lung; ultrafine particles are of particular concern. This situation is most closely related to the particles that emerge from coal combustion, which contain many heavy metal components and high levels of sulfur compounds. In addition, air pollution, including PM, may interact with airborne allergens to induce and exacerbate allergic asthma [13].

The biological effects of $PM_{2.5}$ have generally been reported as short-term or long-term exposure effects (Table 1). Short-term (hours to days) exposures have been reported to increase hospital and emergency visits due to asthma attacks or COPD in many meta-analyses [14–17]. In asthma, with an increase in the $PM_{2.5}$ concentration of 10 $\mu g/m^3$, the risk of hospital visits has been shown to increase by 1.5% [14]; Zheng et al. [15] and Lim et al. [16] also indicated a relative risk of hospital or emergency consultation of 1.023 and 1.048, respectively. Similarly, in COPD, a 10 $\mu g/m^3$ increase in the $PM_{2.5}$ concentration resulted in a significant increase in the rate of hospital visits (0.31%) [16], and also significantly increased the relative risk of acute exacerbations of COPD (1.031) [17]. In addition, long-term (months to years) exposure to $PM_{2.5}$ has been shown to affect lung function development in children. Over an 8-year period (from 10 to 18 years old), the decreased gain in forced expiratory volume in one second ($FEV_{1.0}$) was significantly associated with exposure to $PM_{2.5}$ ($p = 0.04$), and the estimated proportion of 18-year-old subjects with a low $FEV_{1.0}$ (defined as <80% of the predicted $FEV_{1.0}$) was approximately five-fold greater in those exposed to the highest level of $PM_{2.5}$ than in those exposed to the lowest level ($p = 0.002$) [18].

PM was classified as a group 1 carcinogen by the IARC [19], and it has been shown that long-term exposure to $PM_{2.5}$ is associated with the development and mortality of lung cancer. In a large cohort study, the mortality risk ratio of lung cancer increased significantly to 8% [20] and by 1.23-fold [21] when the average concentration of $PM_{2.5}$ increased by 10 $\mu g/m^3$. In addition, a global meta-analysis revealed that $PM_{2.5}$ significantly increased the risk of lung cancer development [22].

Each country makes efforts to control the $PM_{2.5}$ level via emission regulation. However, the level may also be affected by climate change and drastic fluctuations during a day or season [23]; thus, it may be difficult to exert control via regulation alone. Greater attention should be paid to the $PM_{2.5}$ level, particularly regarding the populations susceptible to air pollution, for example, children, elderly people, pregnant women, those with cardiovascular or respiratory diseases or diabetes [24–26].

Table 1. Health hazards to the respiratory system of $PM_{2.5}$.

Adverse Health Effects		Reference Number
Short-term exposure (hours to days)		
Asthma attacks	Increased risk of hospital visits	[14–16]
COPD	Increased risk of hospital visits,	[16]
	Increased the relative risk of acute exacerbations	[17]
Long-term exposure (month to years)		
Lung function development	Suppression of lung function development in children	[18]
Lung cancer	Increased the development and mortality of lung cancer	[20–22]
SARS-CoV-2 associated events	Increased the risk of SARS-CoV-2-associated respiratory distress, respiratory failure, and mortality	[27–32]

SARS-CoV-2: severe acute respiratory syndrome coronavirus 2.

Recently, chronic exposure to air pollution has intensified severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)-associated respiratory distress, respiratory failure, and mortality [27–32]. Chronic exposure to air pollutants such as $PM_{2.5}$ and NO may promote SARS-CoV-2 entry and lifecycle in the body via several mechanisms, including the prevention of antiviral activity by antimicrobial peptides and surfactant protein D, increasing angiotensin-converting enzyme 2 receptor expression levels on the surface of airway epithelial cells, and inhibiting mucociliary clearance [27,33]. Severe acute respiratory illness due to SARS-CoV-2 can worsen Th1/Th17 proinflammatory cytokine production in humans, resulting in a cytokine storm. Air pollutants, such as $PM_{2.5}$, can also enhance the Th1/Th17 immune response and may exacerbate SARS-CoV-2-related respiratory distress, failure, and mortality [34].

3.2. Ambient Occupational Pollution

Occupational air pollution may cause or worsen various lung diseases, including malignant diseases such as lung cancer and malignant mesothelioma, WRA, occupational COPD, pneumoconiosis, and HP (Tables 2 and 3). It is estimated that 5–10% of global cases of lung cancer are due to occupational exposure to carcinogens [35]. Lung cancer is frequently associated with exposure to carcinogenic materials in the workplace [36,37]. Many occupational pollutants cause lung cancer in humans, including heavy metals such as arsenic, chromium [VI], and nickel and radiation [38], but the most common is asbestos. Asbestos comprises natural silicate mineral fibers that can be categorized into two groups: the serpentine group (e.g., chrysotile) and the amphibole group (e.g., crocidolite, amosite, tremolite, and others). The serpentine group has a winding standard structure, whereas the amphibole group is composed of straight, rod-like fibers, and both forms are related to the development of lung cancer and malignant mesothelioma [39]. These fibers are used in construction and manufacturing, such as brake linings, pads, and insulation. By the mid-20th century, asbestos exposure had drastically increased the risk of lung and pleural diseases, including non-malignant (e.g., pleural effusion, pleural plaque, rounded atelectasis, and asbestosis) and malignant (lung cancer and mesothelioma) diseases [40–42]. Occupational asbestos exposure is associated with a five-fold increase in the risk of lung cancer [43,44], and the risk of lung cancer has been noted to increase with increased exposure to asbestos [44]. Malignant mesothelioma (mainly malignant pleural mesothelioma) is the most common malignant occupational respiratory disease, and 23,000 cases of malignant mesothelioma were caused by asbestos exposure in 2015 [45]. Although all forms of asbestos fibers can also cause pulmonary fibrosis [39], which is referred to as asbestosis, the risk of asbestosis is higher following exposure to the amphibole group than to the serpentine group [46]. Asbestosis is a progressive disease and its severity is related to the total dose of asbestos exposure [46]. A recent study revealed that longer fibers are more powerful than shorter fibers in stimulating the nuclear factor (NF)-kB pathway and gene promoter activities, and the length of fibers is directly related to pathogenesis, with longer fibers generally being more harmful [47]. The fiber type at the time of initial exposure and physical properties of various asbestos fibers determine the toxic effects of asbestos [41,48–50]. Alveolar epithelial cells and alveolar macrophages internalize inhaled asbestos fibers, and consequently, oxidative stress is induced by these cells in the lung due to the release of reactive oxygen species (ROS) and reactive nitrogen species (RNS) [48,50,51]. Although the use of asbestos has been banned or strictly restricted in at least 40 countries [52], the health risks of asbestos remain relevant because the latent period between exposure and the development of asbestos-related diseases can span several decades [53].

Similar to asbestos, crystalline silicon dioxide or silica also induces pulmonary fibrosis, which is referred to as silicosis. People who work in the mining, quarrying, drilling, foundries, ceramics, and sandblasting industries are at high risk of developing silicosis [54]. Silicosis generally has a latent period of approximately 10–30 years; however, accelerated silicosis can develop earlier in people exposed to large quantities of fine silica dust during a relatively short period (typically months) [54–58]. Accelerated silicosis, also known as

silicoproteinosis, may result from the excessive production of proteinaceous materials and surfactant proteins by hypertrophic type II pneumocytes and excessive radicals by silica particles [59–62]. Similar to asbestosis, it has been reported that chronic silicosis is also related to the generation of ROS and RNS due to silica particles lodging in alveolar macrophages [63].

Some occupational pollutants, such as sensitizers or irritants, cause or exacerbate WRA (Table 3). Asthma has the second highest prevalence and mortality rate after COPD among chronic respiratory diseases worldwide [64]. The incidence of asthma was 43 million in 2017, and the estimated prevalence and mortality were 273 million (3.6% of the world population) and 500,000 deaths (mortality rate of 6.48/100,000 population) [64–66], and approximately 7% of these 500,000 deaths were attributed to WRA [67]. WRA can be classified as occupational asthma (OA) or work-exacerbated asthma (WEA) [68,69]. OA is defined as asthma caused by exposure to certain materials in the occupational environment, whereas WEA is defined as preexisting or concurrent asthma exacerbated by exposure at work [68–70], and the prevalence of OA and WEA has been estimated at 16.0% and 21.5%, respectively [71]. In a surveillance study conducted in the United States, 'miscellaneous chemicals and materials (including pesticides and glues)', 'mineral and inorganic dusts (including cement dust and copier toner)' and 'cleaning materials' were the top three agent categories attributable to WEA, which accounted for >40% of all 14 categories of the Association of Occupational and Environmental Clinics Exposure Code List [71,72]. OA is further categorized into sensitizer-induced OA and irritant-induced OA [69]. Although sensitizer-induced OA accounts for approximately 90% of all OA cases and 5–18% of OA cases are reported as irritant-induced asthma [73], there are variations across studies. Approximately 600 agents have been associated with OA, of which 400 cause asthma due to sensitization mechanisms [74]. In addition, >70% of cases of sensitizer-induced OA are attributable to only eight agents (flour, latex, isocyanates, persulfates, metals, quaternary ammonium compounds, acrylates, and wood) [75]. The onset of symptoms after a latency period (e.g., months or years) is characteristic of sensitizer-induced OA, and exposure at higher levels, sensitizing agents, and individual characteristics are related to an earlier onset of OA [76]. Sensitizers include high molecular weight (>5 kDa) and low molecular weight (≤4 kDa) agents [77,78]. The former are mostly proteins, such as those of animal or plant origin, and have an IgE-mediated immune mechanism, while the latter are composed of organic or inorganic compounds that can act as haptens but often have an unclear mechanism [69,70]. The phenotype of sensitizer-induced asthma has been revealed in recent investigations, including a large cohort study [75,79]. In the group of patients exposed to low molecular weight sensitizers, symptoms such as chest tightness and daily sputum at work, severe asthma exacerbations, and late asthmatic reactions during specific inhalation challenge (SIC), were more frequent [75,79]. In the group of patients exposed to high molecular weight sensitizers, there were more cases of work-related conjunctivitis and rhinitis, baseline airway obstruction, atopy, early asthmatic reactions to SIC, higher levels of fractional exhaled NO, and blood eosinophils [75,79]. Irritant-induced asthma (also known as nonimmunologic asthma), including reactive airway dysfunction syndrome (RADS), occurs without a latent period after high-level single or multiple exposures to an irritant [68,80]. RADS was first reported in 1985 [81] and best meets the definitive diagnosis of irritant-induced asthma, i.e., onset within 24 h of exposure to high concentration irritants such as gases, fumes, smoke, or vapors, with pulmonary functional changes (positive bronchodilator response or methacholine challenge) [80–83]. Additionally, there is another phenotype of RADS, which is characterized by a more insidious onset >24 h after exposure due to exposure to multiple agents or chronic exposure at low concentrations [82,84,85]. The reported causative agents of irritant-induced asthma include benzene-1,2,4-tricarboxylic acid, 1,2-anhydride (trimellitic anhydride), chlorine, cobalt, cement, environmental tobacco smoke, grain, welding fumes, construction work, swine or poultry confinement or farming, and the 2001 New York World Trade Center collapse [86]. Neurogenic inflammation and oxidative stress are suggested mechanisms of irritant-induced asthma [82], for example,

activated transient receptor potential channels [87], or irritant agents that enhance the airway response to allergens [88,89]. In addition, genetic factors, such as polymorphisms of genes related to inflammation via the NF-κB pathway, may also play a role in irritant-induced asthma [90].

In recent decades, it has been demonstrated that cleaning agents may cause asthma. The cleaning products frequently used around the world contain well-known sensitizers (e.g., quaternary ammonium compounds, amines, and fragrances) and irritants (e.g., sodium hypochlorite, hydrochloric acid, and alkaline agents (ammonia and caustic soda)). Therefore, these cleaning agents can be attributed to sensitizer-induced and irritant-induced asthma, in addition to WEA [91,92].

Certain organic chemicals and low molecular weight compounds can cause HP [93]. The incidence of HP ranges from to 1–3 to 30 per 100,000 people per annum [94–96]. It is estimated that HP due to occupational exposure accounts for 12–28% of all HP cases [5]. Most cases of HP are reported in farmers (1.3–12.9% [97,98]), bird breeders (3.7–10.4% [99,100]), and mushroom producers (3.5–29% [101–104]). Isocyanates are low-molecular-weight organic chemicals that are well-known HP-causing agents [105], and are often used as surface protective materials, in paints, and in polyurethane production [106]. Although isocyanates are not immunogenic per se, haptens consisting of human proteins may induce HP [105]. Although the immunopathogenesis involved in HP have not been fully elucidated, it is suggested that aberrant immunological mechanisms are involved. Inhaled antigens interact with antigen-presenting cells (APCs), such as macrophages and dendritic cells via pattern recognition receptors, leading to stimulation of the Th1 response of APCs. The Th1 response of APCs is enhanced by cytokine and chemokine production, which promotes the influx of neutrophils into the lungs; subsequently, the complement cascade is initiated and macrophages are stimulated, forming granulomas as a result of fusion with multinucleated giant cells and epithelioid cells. Concurrently, antibodies, particularly immunoglobulin G (IgG), are produced by stimulated B cells that recognize antigens and form antigen–antibody complexes. During the transition from acute, predominantly inflammatory HP to chronic or fibrous HP, fibroblast proliferation is promoted by chemotactic factors produced by granulomas, switching from a Th1 to Th2 inflammatory response, a decrease in regulatory T cells, production of cytotoxic $CD8^+$ T cells, and differentiation of Th17 cells. Fibroblasts then differentiate into myofibroblasts, producing collagen and extracellular matrix components, which promotes pulmonary fibrosis [93]. In addition, gene variants, including the tumor necrosis factor (TNF)-gene promotor region [107], overexpression of mucin 5 subtype B (MUC5B) [108], and telomere dysfunctions [109], are involved as genetic risk factors in individuals susceptible to fibrotic HP.

In recent years, new toxic materials have emerged, such as cross-linked acrylic acid-based polymers (CL-PAAs), which cause serious lung disorders, including pneumoconiosis. CL-PAAs are polymers that contain acrylic acid as a monomer. CL-PAAs are widely used as intermediates in the manufacture of pharmaceuticals and cosmetics [106]. According to reports from the Working Group on Occupational Accident Diseases of the Ministry of Health, Labor and Welfare in Japan, all affected workers with lung diseases were relatively young (aged 20–40 years), and pneumoconiosis developed within a short period (several years) from the initial CL-PAA exposure before progressing. The radiological and clinical findings in these workers were pulmonary fibrosis, interstitial pneumonia, emphysema, and pneumothorax [110]. Since CL-PAAs have not been reported to be harmful to the lung, they should be considered a new hazard and further investigations should be conducted to determine the mechanism of pulmonary toxicity following CL-PAA exposure.

Int. J. Environ. Res. Public Health **2022**, 19, 2788

7 of 12

Table 2. Representative occupational pollutants with health effects on the respiratory system (except work-related asthma) in this review.

Pollutants	Occupational Exposure Circumstances	Respiratory Diseases	Reference Number
Inorganic substances Mineral dusts			
Asbestos Chrysotile (Serpentine group) Crocidolite, amosite, tremolite, and others (Amphibole group)	Construction, manufacturing brake lining and pads, and handling insulation	Lung cancer, mesothelioma, pleural effusion, pleural plaque, rounded atelectasis, and asbestosis	[38–42]
Crystalline silicon dioxide, Crystalline silica Heavy metals	Mining, quarrying, drilling, foundries, ceramics, and sandblasting industries	Silicosis, Lung cancer	[54–58]
Arsenic	Hot smelting, fur handling, manufacturing sheep-dip compounds and pesticides, and vineyard working	Lung cancer	[7,38]
Chromium [VI]	Producing chromate, chromium platers and ferrochromium, and manufacturing chromate pigment	Lung cancer	[7,38]
Nickel Radiation	Mining, smelting, and electrolyzing	Lung cancer	[7,38]
Low-LET radiation; X-rays, and γ-rays	Medical professions and nuclear industry	Lung cancer	[7,38]
High-LET radiation and Radon Organic substances	Underground mining	Lung cancer	[7,38]
Saccharomyces spp., *Aspergillus* spp.	Farming	Hypersensitivity pneumonitis	[93]
Bird bloom, feather, droppings, serum, and down products	Bird breeding, Manufacturing down products	Hypersensitivity pneumonitis	[93]
Shitake, bunashimeji, nameko, eringi, and thermophilic *Actinomycetes*	Mushroom working	Hypersensitivity pneumonitis	[93,111]
Isocyanates	Surface protective materials, painting, and producing polyurethane	Hypersensitivity pneumonitis	[93,106]
Cross-linked acrylic acid-based polymers	Manufacturing pharmaceuticals and cosmetics	Pneumoconiosis, emphysema, and pneumothorax	[106,110]

LET: liner energy transfer.

Table 3. Work-related asthma (WRA) and causative agents.

Classification of WRA	Causative Agents	Reference Number
Work-exacerbated asthma (WEA)	Miscellaneous chemicals and materials (including pesticides and glues), mineral and inorganic dusts (including cement dust and copier toner), and cleaning materials	[71,72,91,92]
Occupational asthma (OA)		
Sensitizer-induced OA	High molecular weight (≧5 kDa); flour, latex Low molecular weight (<5 kDa); isocyanates, persulfates, metals, cleaning materials (quaternary ammonium compounds, amines, and fragrances), acrylates, and wood	[75,91,92]
Irritant-induced OA (including RADS)	Benzene-1,2,4-tricarboxylic acid, 1,2-anhydride (trimellitic anhydride), sodium hypochlorite, hydrochloric acid, alkaline agents (ammonia and caustic soda), chlorine, cobalt, cement, environmental tobacco smoke, grain, welding fumes, construction work, swine or poultry confinement or farming, and the 2001 New York World Trade Center collapse	[68,80,86,91,92]

4. Conclusions

Respiratory diseases related to ambient or occupational pollution can be avoided, and it is crucial to avoid exposure to all harmful materials in order to prevent and control such

diseases. Adequate awareness of the risk of exposure to hazardous materials should be mandatory for all employers, occupational and pulmonary physicians, and researchers. Adequate education on occupational health and safety and awareness of environmental quality may lead to better health promotion and behavioral changes to prevent related diseases and disease exacerbations, as well as have an impact on social behavior to reduce air pollutants.

References

1. Thatcher, T.H.; Woeller, C.; McCarthy, C.E.; Sime, P.J. Quenching the fires: Pro-resolving mediators, air pollution, and smoking. *Pharmacol. Ther.* **2019**, *197*, 212–224. [CrossRef] [PubMed]
2. WHO. Releases Country Estimates on Air Pollution Exposure and Health Impact. Available online: https://www.who.int/news/item/27-09-2016-who-releases-country-estimates-on-air-pollution-exposure-and-health-impact (accessed on 23 December 2021).
3. Lelieveld, J.; Evans, J.S.; Fnais, M.; Giannadaki, D.; Pozzer, A. The contribution of outdoor air pollution sources to premature mortality on a global scale. *Nature* **2015**, *525*, 367–371. [CrossRef] [PubMed]
4. Vlahovich, K.P.; Sood, A. A 2019 Update on Occupational Lung Diseases: A Narrative Review. *Pulm. Ther.* **2021**, *7*, 1–13. [CrossRef]
5. Blanc, P.D.; Annesi-Maesano, I.; Balmes, J.R.; Cummings, K.J.; Fishwick, D.; Miedinger, D.; Murgia, N.; Naidoo, R.N.; Reynolds, C.J.; Sigsgaard, T.; et al. The Occupational Burden of Nonmalignant Respiratory Diseases. An Official American Thoracic Society and European Respiratory Society Statement. *Am. J. Respir. Crit. Care Med.* **2019**, *199*, 1312–1334. [CrossRef]
6. Schraufnagel, D.E.; Balmes, J.R. Air Pollution and Noncommunicable Diseases: A Review by the Forum of International Respir-atory Societies' Environmental Committee, Part 1: The Damaging Effects of Air Pollution. *Chest* **2019**, *155*, 409–416. [CrossRef]
7. Boffetta, P. Epidemiology of environmental and occupational cancer. *Oncogene* **2004**, *23*, 6392–6403. [CrossRef]
8. Ministry of the Environment Goverment of Japan. Available online: https://www.env.go.jp/ (accessed on 5 December 2021).
9. Pearson, C.; Littlewood, E. Exposures and health outcomes in relation to bioaerosol emissions from composting facilities: A sys-tematic review of occupational and community studies. *J. Toxicol. Environ. Health B Crit. Rev.* **2015**, *18*, 43–69. [CrossRef]
10. Kc, R.; Shukla, S.D.; Gautam, S.S.; Hansbro, P.M.; O'Toole, R.F. The role of environmental exposure to non-cigarette smoke in lung disease. *Clin. Transl. Med.* **2018**, *7*, 39. [CrossRef]
11. Schraufnagel, D.E.; Balmes, J.R. Air Pollution and Noncommunicable Diseases: A Review by the Forum of International Respir-atory Societies' Environmental Committee, Part 2: Air Pollution and Organ Systems. *Chest* **2019**, *155*, 417–426. [CrossRef]
12. Shamsollahi, H.R.; Jahanbin, B.; Rafieian, S.; Yunesian, M. Particulates induced lung inflammation and its consequences in the development of restrictive and obstructive lung diseases: A systematic review. *Environ. Sci. Pollut. Res.* **2021**, *28*, 25035–25050. [CrossRef]
13. Baldacci, S.; Maio, S.; Cerrai, S.; Sarno, G.; Baïz, N.; Simoni, M.; Annesi-Maesano, I.; Viegi, G.; on behalf of the HEALS Study. Allergy and asthma: Effects of the exposure to particulate matter and biological allergens. *Respir. Med.* **2015**, *109*, 1089–1104. [CrossRef] [PubMed]
14. Fan, J.; Li, S.; Fan, C.; Bai, Z.; Yang, K. The impact of $PM_{2.5}$ on asthma emergency department visits: A systematic review and meta-analysis. *Environ. Sci. Pollut. Res.* **2015**, *23*, 843–850. [CrossRef] [PubMed]
15. Zheng, X.-Y.; Ding, H.; Jiang, L.-N.; Chen, S.-W.; Zheng, J.-P.; Qiu, M.; Zhou, Y.-X.; Chen, Q.; Guan, W.-J. Association between Air Pollutants and Asthma Emergency Room Visits and Hospital Admissions in Time Series Studies: A Systematic Review and Meta-Analysis. *PLoS ONE* **2015**, *10*, e0138146. [CrossRef] [PubMed]
16. Lim, H.; Kwon, H.-J.; Lim, J.-A.; Choi, J.H.; Ha, M.; Hwang, S.-S.; Choi, W.-J. Short-term Effect of Fine Particulate Matter on Children's Hospital Admissions and Emergency Department Visits for Asthma: A Systematic Review and Meta-analysis. *J. Prev. Med. Public Health* **2016**, *49*, 205–219. [CrossRef] [PubMed]

17. Ko, F.W.S.; Tam, W.; Wong, T.W.; Chan, D.P.S.; Tung, A.H.; Lai, C.K.W.; Hui, D.S.C. Temporal relationship between air pollutants and hospital admissions for chronic obstructive pulmonary disease in Hong Kong. *Thorax* **2007**, *62*, 780–785. [CrossRef]

18. Gauderman, W.J.; Avol, E.; Gilliland, F.; Vora, H.; Thomas, D.; Berhane, K.; McConnell, R.; Künzli, N.; Lurmann, F.; Rappaport, E.; et al. The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age. *N. Engl. J. Med.* **2004**, *351*, 1057–1067. [CrossRef]

19. Agents Classified by the IARC Monographs, Volumes 1–130—IARC Monographs on the Identification of Carcinogenic Haz-ards to Humans. Available online: https://monographs.iarc.who.int/agents-classified-by-the-iarc/ (accessed on 7 December 2021).

20. Pope, C.A., 3rd; Burnett, R.T. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA* **2002**, *287*, 1132–1141. [CrossRef]

21. Katanoda, K.; Sobue, T.; Satoh, H.; Tajima, K.; Suzuki, T.; Nakatsuka, H.; Takezaki, T.; Nakayama, T.; Nitta, H.; Tanabe, K.; et al. An Association Between Long-Term Exposure to Ambient Air Pollution and Mortality from Lung Cancer and Respiratory Diseases in Japan. *J. Epidemiol.* **2011**, *21*, 132–143. [CrossRef]

22. Cui, P.; Huang, Y.; Han, J.; Song, F.; Chen, K. Ambient particulate matter and lung cancer incidence and mortality: A meta-analysis of prospective studies. *Eur. J. Public Health* **2015**, *25*, 324–329. [CrossRef]

23. Samet, J.M.; Utel, M.J. The environment and the lung. Changing perspectives. *JAMA* **1991**, *266*, 670–675. [CrossRef]

24. Chen, Y.; Craig, L.; Krewski, D. Air Quality Risk Assessment and Management. *J. Toxicol. Environ. Health Part A* **2008**, *71*, 24–39. [CrossRef] [PubMed]

25. Li, W.; Dorans, K.S.; Wilker, E.H.; Rice, M.B.; Schwartz, J.; Coull, B.A.; Koutrakis, P.; Gold, D.R.; Fox, C.S.; Mittleman, M. Residential proximity to major roadways, fine particulate matter, and adiposity: The framingham heart study. *Obesity* **2016**, *24*, 2593–2599. [CrossRef] [PubMed]

26. Sheehan, W.J.; Gaffin, J.M.; Peden, D.; Bush, R.K.; Phipatanakul, W. Advances in environmental and occupational disorders. *J. Allergy Clin. Immunol.* **2017**, *140*, 1683–1692. [CrossRef]

27. Frontera, A.; Cianfanelli, L.; Vlachos, K.; Landoni, G.; Cremona, G. Severe air pollution links to higher mortality in COVID-19 patients: The "double-hit" hypothesis. *J. Infect.* **2020**, *81*, 255–259. [CrossRef] [PubMed]

28. Zuo, T.; Zhang, F.; Lui, G.C.Y.; Yeoh, Y.K.; Li, A.Y.L.; Zhan, H.; Wan, Y.; Chung, A.C.K.; Cheung, C.P.; Chen, N.; et al. Alterations in Gut Microbiota of Patients With COVID-19 During Time of Hospitalization. *Gastroenterology* **2020**, *159*, 944–955.e948. [CrossRef] [PubMed]

29. Wu, X.; Nethery, R.C. Exposure to air pollution and COVID-19 mortality in the United States: A nationwide cross-sectional study. *medRxiv* **2020**.

30. Konstantinoudis, G.; Padellini, T.; Bennett, J.; Davies, B.; Ezzati, M.; Blangiardo, M. Long-term exposure to air-pollution and COVID-19 mortality in England: A hierarchical spatial analysis. *Environ. Int.* **2021**, *146*, 106316. [CrossRef]

31. Bashir, M.F.; Ma, B.J. Correlation between environmental pollution indicators and COVID-19 pandemic: A brief study in Cali-fornian context. *Environ. Res.* **2020**, *187*, 109652. [CrossRef]

32. Domingo, J.L.; Rovira, J. Effects of air pollutants on the transmission and severity of respiratory viral infections. *Environ. Res.* **2020**, *187*, 109650. [CrossRef]

33. Woodby, B. Arnold MM. SARS-CoV-2 infection, COVID-19 pathogenesis, and exposure to air pollution: What is the connection? *Ann. N. Y. Acad. Sci.* **2021**, *1486*, 15–38. [CrossRef]

34. Naidoo, P.; Ghazi, T.; Chuturgoon, A.A.; Naidoo, R.N.; Ramsuran, V.; Mpaka-Mbatha, M.N.; Bhengu, K.N.; Nembe, N.; Duma, Z.; Pillay, R.; et al. SARS-CoV-2 and helminth co-infections, and environmental pollution exposure: An epidemiological and immunological perspective. *Environ. Int.* **2021**, *156*, 106695. [CrossRef] [PubMed]

35. Devesa, S.S.; Bray, F. International lung cancer trends by histologic type: Male:female differences diminishing and adenocarci-noma rates rising. *Int. J. Cancer* **2005**, *117*, 294–299. [CrossRef] [PubMed]

36. Doll, R.; Peto, R. The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today. *JNCI J. Natl. Cancer Inst.* **1981**, *66*, 1192–1308. [CrossRef]

37. Alberg, A.J.; Brock, M.V. Epidemiology of Lung Cancer: Diagnosis and Management of Lung Cancer, 3rd ed. American College of Chest Physicians evidence-based clinical practice guidelines. *Chest* **2013**, *143*, e1S–e29S. [CrossRef] [PubMed]

38. International Agency for Research on Cancer—IARC Monographs on the Identification of Carcinogenic Hazards to Humans. Available online: https://monographs.iarc.who.int/list-of-classifications (accessed on 21 September 2021).

39. Hosoda, Y.; Hiraga, Y. Railways and asbestos in Japan (1928–1987)—Epidemiology of pleural plaques, malignancies and pneu-moconioses. *J. Occup. Health* **2008**, *50*, 297–307. [CrossRef]

40. Roach, H.D.; Davies, G.J.; Attanoos, R.; Crane, M.; Adams, H.; Phillips, S. Asbestos: When the Dust Settles—An Imaging Review of Asbestos-related Disease. *RadioGraphics* **2002**, *22*, S167–S184. [CrossRef]

41. Guidotti, T.L.; Miller, A.; Christiani, D.; Wagner, G.; Balmes, J.; Harber, P.; Brodkin, C.A.; Rom, W.; Hillerdal, G.; Harbut, M.; et al. Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos. *Am. J. Respir. Crit. Care Med.* **2004**, *170*, 691–715.

42. Hein, M.J.; Stayner, L.T.; Lehman, E.; Dement, J.M. Follow-up study of chrysotile textile workers: Cohort mortality and exposure-response. *Occup. Environ. Med.* **2007**, *64*, 616–625. [CrossRef]

43. Knox, J.F.; Holmes, S.; Doll, R.; Hill, I.D. Mortality from Lung Cancer and Other Causes among Workers in an Asbestos Textile Factory. *Occup. Environ. Med.* **1968**, *25*, 293–303. [CrossRef]

44. Newhouse, M.L.; Berry, G. Patterns of mortality in asbestos factory workers in London. *Ann. N. Y. Acad. Sci.* **1979**, *330*, 53–60. [CrossRef]
45. GBD 2013 Risk Factors Collaborators; Forouzanfar, M.H.; Alexander, L. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990-2013: A systematic analysis for the Global Burden of Disease Study 2013. *Lancet* **2015**, *386*, 2287–2323. [CrossRef]
46. Bhandari, J.; Thada, P.K. *Asbestosis*; StatPearls Publishing: Treasure Island, FL, USA, 2021. Available online: https://www.ncbi.nlm.nih.gov/books/NBK555985/ (accessed on 19 December 2021).
47. Fujimura, N. Pathology and pathophysiology of pneumoconiosis. *Curr. Opin. Pulm. Med.* **2000**, *6*, 140–144. [CrossRef] [PubMed]
48. Kamp, D.W.; Weitzman, S.A. Asbestosis: Clinical Spectrum and Pathogenic Mechanisms. *Exp. Biol. Med.* **1997**, *214*, 12–26. [CrossRef] [PubMed]
49. Cugell, D.W.; Kamp, D.W. Asbestos and the pleura: A review. *Chest* **2004**, *125*, 1103–1117. [CrossRef]
50. Mossman, B.T.; Churg, A. Mechanisms in the Pathogenesis of Asbestosis and Silicosis. *Am. J. Respir. Crit. Care Med.* **1998**, *157 Pt 1*, 1666–1680. [CrossRef]
51. Shukla, A.; Gulumian, M.; Hei, T.K.; Kamp, D.; Rahman, Q.; Mossman, B.T. Multiple roles of oxidants in the pathogenesis of asbestos-induced diseases. *Free Radic. Biol. Med.* **2003**, *34*, 1117–1129. [CrossRef]
52. Wagner, G.R. The fallout from asbestos. *Lancet* **2007**, *369*, 973–974. [CrossRef]
53. Jamrozik, E.; de Klerk, N. Asbestos-related disease. *Intern. Med. J.* **2011**, *41*, 372–380. [CrossRef]
54. Bang, K.M.; Attfield, M.D.; Wood, J.M.; Syamlal, G. National trends in silicosis mortality in the United States, 1981–2004. *Am. J. Ind. Med.* **2008**, *51*, 633–639. [CrossRef]
55. Akira, M. High-resolution CT in the evaluation of occupational and environmental disease. *Radiol. Clin. N. Am.* **2002**, *40*, 43–59. [CrossRef]
56. Antao, V.C.; Pinheiro, G.A. High-resolution CT in silicosis: Correlation with radiographic findings and functional impairment. *J. Comput. Assist. Tomogr.* **2005**, *29*, 350–356. [CrossRef] [PubMed]
57. Marchiori, E.; Souza, C.A.; Barbassa, T.G.; Escuissato, D.L.; Gasparetto, E.L.; Souza, A.S. Silicoproteinosis: High-Resolution CT Findings in 13 Patients. *Am. J. Roentgenol.* **2007**, *189*, 1402–1406. [CrossRef]
58. Chong, S.; Lee, K.S.; Chung, M.J.; Han, J.; Kwon, O.J.; Kim, T.S. Pneumoconiosis: Comparison of Imaging and Pathologic Findings. *RadioGraphics* **2006**, *26*, 59–77. [CrossRef] [PubMed]
59. Hoffmann, E.O.; Lamberty, J.; Pizzolato, P.; Coover, J. The ultrastructure of acute silicosis. *Arch. Pathol.* **1973**, *96*, 104–107.
60. Dalal, N.S.; Shi, X.; Vallyathan, V. Esr Spin Trapping and Cytotoxicity Investigations of Freshly Fractured Quartz: Mechanism of Acute Silicosis. *Free Radic. Res. Commun.* **1990**, *9*, 259–266. [CrossRef]
61. Heffner, J.E.; Repine, J.E. Pulmonary Strategies of Antioxidant Defense. *Am. Rev. Respir. Dis.* **1989**, *140*, 531–554. [CrossRef]
62. Fubini, B.; Hubbard, A. Reactive oxygen species (ROS) and reactive nitrogen species (RNS) generation by silica in inflammation and fibrosis. *Free Radic. Biol. Med.* **2003**, *34*, 1507–1516. [CrossRef]
63. Dubois, C.M.; Bissonnette, E.; Rola-Pleszczynski, M. Asbestos Fibers and Silica Particles Stimulate Rat Alveolar Macrophages to Release Tumor Necrosis Factor: Autoregulatory Role of Leukotriene B4. *Am. Rev. Respir. Dis.* **1989**, *139*, 1257–1264. [CrossRef] [PubMed]
64. GBD Chronic Respiratory Disease Collaborators; Soriano, J.B.; Kendrick, P.J. Prevalence and attributable health burden of chronic respiratory diseases, 1990–2017: A systematic analysis for the Global Burden of Disease Study 2017. *Lancet Respir. Med.* **2020**, *8*, 585–596. [CrossRef]
65. GBD 2017 Disease and Injury Incidence and Prevalence Collaborators; James, S.L.; Abate, D. Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: A systematic analysis for the Global Burden of Disease Study 2017. *Lancet* **2018**, *392*, 1789–1858. [CrossRef]
66. GBD 2017 Causes of Death Collaborators; Roth, G.A.; Abate, D. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: A systematic analysis for the Global Burden of Disease Study 2017. *Lancet* **2018**, *392*, 1736–1788. [CrossRef]
67. GBD 2017 Risk Factor Collaborators; Stanaway, J.D.; Afshin, A. Global, regional, and national comparative risk assessment of 84 behavioural, environ-mental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990–2017: A systematic analysis for the Global Burden of Disease Study 2017. *Lancet* **2018**, *392*, 1923–1994.
68. Chan-Yeung, M. Assessment of Asthma in the Workplace. *Chest* **1995**, *108*, 1084–1117. [CrossRef] [PubMed]
69. Mapp, C.E.; Boschetto, P. Occupational asthma. *Am. J. Respir. Crit. Care Med.* **2005**, *172*, 280–305. [CrossRef]
70. Roio, L.C.D.; Mizutani, R.F. Work-related asthma. *J. Bras. Pneumol.* **2021**, *47*, e20200577.
71. Henneberger, P.K.; Redlich, C.A.; Callahan, D.B.; Harber, P.; Lemière, C.; Martin, J.; Tarlo, S.M.; Vandenplas, O.; Torén, K. An Official American Thoracic Society Statement: Work-Exacerbated Asthma. *Am. J. Respir. Crit. Care Med.* **2011**, *184*, 368–378. [CrossRef]
72. AOEC—Epidemiology Tools. Available online: http://www.aoec.org/tools.htm (accessed on 23 December 2021).
73. Cormier, M.; Lemière, C. Occupational asthma. *Int. J. Tuberc. Lung Dis.* **2020**, *24*, 8–21. [CrossRef]
74. Baur, X.; Akdis, C.A. Immunological methods for diagnosis and monitoring of IgE-mediated allergy caused by industrial sensi-tizing agents (IMExAllergy). *Allergy* **2019**, *74*, 1885–1897. [CrossRef]

75. Vandenplas, O.; Godet, J.; Hurdubaea, L.; Rifflart, C.; Suojalehto, H.; Wiszniewska, M.; Munoz, X.; Sastre, J.; Klusackova, P.; Moore, V.; et al. Are high- and low-molecular-weight sensitizing agents associated with different clinical phenotypes of occupational asthma? *Allergy* **2019**, *74*, 261–272. [CrossRef]

76. Vandenplas, O. Occupational Asthma: Etiologies and Risk Factors. *Allergy Asthma Immunol. Res.* **2011**, *3*, 157–167. [CrossRef]

77. Mapp, C.E. Agents, old and new, causing occupational asthma. *Occup. Environ. Med.* **2001**, *58*, 354–360. [CrossRef] [PubMed]

78. Tarlo, S.M.; Balmes, J. Diagnosis and management of work-related asthma: American College of Chest Physicians Consensus Statement. *Chest* **2008**, *134*, 1S–41S. [CrossRef] [PubMed]

79. Meca, O.; Cruz, M.J. Do Low Molecular Weight Agents Cause More Severe Asthma than High Molecular Weight Agents? *PLoS ONE* **2016**, *11*, e0156141. Available online: https://pubmed.ncbi.nlm.nih.gov/27280473/ (accessed on 22 December 2021). [CrossRef] [PubMed]

80. Tarlo, S.M. Irritant-Induced Asthma in the Workplace. *Curr. Allergy Asthma Rep.* **2013**, *14*, 1–6. [CrossRef]

81. Brooks, S.M.; Weiss, M.A. Reactive airways dysfunction syndrome (RADS). Persistent asthma syndrome after high level irritant exposures. *Chest* **1985**, *88*, 376–384. [CrossRef]

82. Vandenplas, O.; Wiszniewska, M.; European Academy of Allergy and Clinical Immunology. EAACI position paper: Irritant-induced asthma. *Allergy* **2014**, *69*, 1141–1153. [CrossRef]

83. Banauch, G.I.; Dhala, A.; Alleyne, D.; Alva, R.; Santhyadka, G.; Krasko, A.; Weiden, M.; Kelly, K.J.; Prezant, D.J. Bronchial hyper-reactivity and other inhalation lung injuries in rescue/recovery workers after the World Trade Center collapse. *Crit. Care Med.* **2005**, *33*, S102–S106. [CrossRef] [PubMed]

84. Baur, X.; Bakehe, P.; Vellguth, H. Bronchial asthma and COPD due to irritants in the workplace—An evidence-based approach. *J. Occup. Med. Toxicol.* **2012**, *7*, 19. [CrossRef]

85. Harrison, D.; Luo, H.; Friedman-Jimenez, G. Occupational Asthma and Work-Exacerbated Asthma. *Semin. Respir. Crit. Care Med.* **2015**, *36*, 388–407. [CrossRef]

86. Baur, X. A compendium of causative agents of occupational asthma. *J. Occup. Med. Toxicol.* **2013**, *8*, 15. [CrossRef]

87. Zholos, A.V. TRP Channels in Respiratory Pathophysiology: The Role of Oxidative, Chemical Irritant and Temperature Stimuli. *Curr. Neuropharmacol.* **2015**, *13*, 279–291. [CrossRef] [PubMed]

88. Molfino, N.A.; Wright, S.C. Effect of low concentrations of ozone on inhaled allergen responses in asthmatic subjects. *Lancet* **1991**, *338*, 199–203. [CrossRef]

89. Witten, A.; Solomon, C.; Abbritti, E.; Arjomandi, M.; Zhai, W.; Kleinman, M.; Balmes, J. Effects of Nitrogen Dioxide on Allergic Airway Responses in Subjects with Asthma. *J. Occup. Environ. Med.* **2005**, *47*, 1250–1259. [CrossRef] [PubMed]

90. Rava, M.; Ahmed, I.; Kogevinas, M.; Le Moual, N.; Bouzigon, E.; Curjuric, I.; Dizier, M.-H.; Dumas, O.; Gonzalez, J.R.; Imboden, M.; et al. Genes Interacting with Occupational Exposures to Low Molecular Weight Agents and Irritants on Adult-Onset Asthma in Three European Studies. *Environ. Health Perspect.* **2017**, *125*, 207–214. [CrossRef]

91. Folletti, I.; Siracusa, A. Update on asthma and cleaning agents. *Curr. Opin. Allergy Clin. Immunol.* **2017**, *17*, 90–95. [CrossRef]

92. Siracusa, A.; De Blay, F.; Folletti, I.; Moscato, G.; Olivieri, M.; Quirce, S.; Raulf-Heimsoth, M.; Sastre, J.; Tarlo, S.; Walusiak-Skorupa, J.; et al. Asthma and exposure to cleaning products—A European Academy of Allergy and Clinical Immunology task force consensus statement. *Allergy* **2013**, *68*, 1532–1545. [CrossRef]

93. Barnes, H.; Troy, L.; Lee, C.T.; Sperling, A.; Strek, M.; Glaspole, I. Hypersensitivity pneumonitis: Current concepts in pathogenesis, diagnosis, and treatment. *Allergy* **2021**, *77*, 442–453. [CrossRef]

94. Rittig, A.H.; Hilberg, O.; Ibsen, R.; Løkke, A. Incidence, comorbidity and survival rate of hypersensitivity pneumonitis: A national population-based study. *ERJ Open Res.* **2019**, *5*, 00259–02018. [CrossRef]

95. Fernández Pérez, E.R. Epidemiology of Hypersensitivity Pneumonitis among an Insured Population in the United States: A Claims-based Cohort Analysis. *Ann. Am. Thorac. Soc.* **2018**, *15*, 460–469. [CrossRef]

96. Coultas, D.B.; Zumwalt, R.E.; Black, W.C.; Sobonya, R.E. The epidemiology of interstitial lung diseases. *Am. J. Respir. Crit. Care Med.* **1994**, *150*, 967–972. [CrossRef]

97. Hoppin, J.A.; Umbach, D.M. Pesticides and other agricultural factors associated with self-reported farmer's lung among farm residents in the Agricultural Health Study. *Occup. Environ. Med.* **2007**, *64*, 334–341. [CrossRef] [PubMed]

98. Stanford, C.F.; Hall, G. Farmer's lung in Northern Ireland. *Br. J. Ind. Med.* **1990**, *47*, 314–316. [CrossRef] [PubMed]

99. Charitopoulos, K.; Gioulekas, D.; Sichletidis, L.; Chloros, D.; Vamvakopoulou, V.; Zarogoulidis, K. Hypoxemia: An early indication of pigeon breeders' disease. Clinical and laboratory findings among pigeon breeders in the Salonica area. *J. Investig. Allergy Clin. Immunol.* **2005**, *15*, 211–215.

100. Banham, S.W.; McSharry, C.; Lynch, P.P.; Boyd, G. Relationships between avian exposure, humoral immune response, and pigeon breeders' disease among Scottish pigeon fanciers. *Thorax* **1986**, *41*, 274–278. [CrossRef]

101. Betz, B. Respiratory tract diseases in oyster mushroom cultivators. *Pneumologie* **1990**, *44*, 339–340.

102. Konishi, K.; Mouri, T.; Chida, E.; Kubota, K.; Nakadate, T.; Tamura, M. Chronic type hypersensitivity pneumonitis. *Nihon Kyobu Shikkan Gakkai Zasshi* **1993**, *31*, 62–66. (In Japanese)

103. Tanaka, H.; Saikai, T.; Sugawara, H.; Tsunematsu, K.; Takeya, I.; Koba, H.; Matsuura, A.; Imai, K.; Abe, S. Three-year follow-up study of allergy in workers in a mushroom factory. *Respir. Med.* **2001**, *95*, 943–948. [CrossRef]

104. Tsushima, K.; Fujimoto, K.; Yoshikawa, S.; Kawakami, S.; Koizumi, T.; Kubo, K. Hypersensitivity Pneumonitis due to Bunashimeji Mushrooms in the Mushroom Industry. *Int. Arch. Allergy Immunol.* **2005**, *137*, 241–248. [CrossRef]

105. Selman, M.; Pardo, A. Hypersensitivity pneumonitis: Insights in diagnosis and pathobiology. *Am. J. Respir. Crit. Care Med.* **2012**, *186*, 314–324. [CrossRef]

106. Morimoto, Y.; Nishida, C.; Tomonaga, T.; Izumi, H.; Yatera, K.; Sakurai, K.; Kim, Y. Lung disorders induced by respirable organic chemicals. *J. Occup. Health* **2021**, *63*, e12240. [CrossRef]

107. Falfán-Valencia, R.; Camarena, Á.; Pineda, C.L.; Montaño, M.; Juárez, A.; Buendía-Roldán, I.; Pérez-Rubio, G.; Reséndiz-Hernández, J.M.; Páramo, I.; Vega, A.; et al. Genetic susceptibility to multicase hypersensitivity pneumonitis is associated with the TNF-238 GG genotype of the promoter region and HLA-DRB1*04 bearing HLA haplotypes. *Respir. Med.* **2014**, *108*, 211–217. [CrossRef] [PubMed]

108. Ley, B.; Newton, C.A.; Arnould, I. The MUC5B promoter polymorphism and telomere length in patients with chronic hypersensitivity pneumonitis: An observational cohort-control study. *Lancet Respir. Med.* **2017**, *5*, 639–647. [CrossRef]

109. Ley, B.; Torgerson, D.G.; Oldham, J.M.; Adegunsoye, A.; Liu, S.; Li, J.; Elicker, B.M.; Henry, T.S.; Golden, J.A.; Jones, K.D.; et al. Rare Protein-Altering Telomere-related Gene Variants in Patients with Chronic Hypersensitivity Pneumonitis. *Am. J. Respir. Crit. Care Med.* **2019**, *200*, 1154–1163. [CrossRef] [PubMed]

110. Working Group Report to Decide Whether Pulmonary Disorder That Occured at the Workplace of Production of Powder of Cross-Linked Polyacrylic Acid Is Work-Related Disease or Not (Japanese). Available online: https://www.mhlw.go.jp/content/11402000/000502982.pdf (accessed on 23 December 2021).

111. Saikai, T.; Tanaka, H.; Fuji, M.; Sugawara, H.; Takeya, I.; Tsunematsu, K.; Abe, S. Hypersensitivity Pneumonitis Induced by the Spore of Pleurotus Eryngii (Eringi). *Intern. Med.* **2002**, *41*, 571–573. [CrossRef]