ORIGINAL RESEARCH PAPER
Volume - 12 | Issue - 04 | April - 2023 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A LITERATURE REVIEW ON OCCUPATIONAL EXPOSURES AND THEIR ASSOCIATED DISEASES.

Occupational Health

Barira Siddiqui MPT final year Student, Department of Physiotherapy, School of Health Sciences, Garden City University.

Dr. Anjali Suresh Professor, and HOD, Department of Physiotherapy, School of Health Sciences, Garden City University.

Dr. R. Sedhunivas Assistant Professor, Department of Physiotherapy, School of Health Sciences, Garden City University.

ABSTRACT

Lung diseases are called work-related when they are caused, worsened, or exacerbated by workplace exposure. They include an extensive range of long-lasting pathologies occurring from the upper respiratory tract to the lower respiratory tract. Online search engines such as Google Scholar, PubMed, and PEDro were searched. We have included articles from 2006-2021. In this review, we are going to discuss the conditions, the causative factors, the clinical manifestations, and the management.

KEYWORDS

hypersensitivity pneumonitis, obstructive lung disease, occupational COPD, occupational lung disease, pneumoconiosis, pulmonary fibrosis, restrictive lung disease, silicosis, and work-related asthma.

INTRODUCTION

Work-related or occupational lung diseases are lung conditions caused, worsened, or exacerbated by exposure to workplace pollutants. Work-related lung disease (WRLD) and non-work-related lung diseases are clinically synonymous but are misinterpreted by one another based on etiology and are usually identified as work-related illnesses, however, withinside the absence of occupational records, they're indistinguishable from the similar conditions that are usually seen in the overall population[1].

Increased industrialization and modernization in the mining industries and the execution of proficient preventive exposure control measures then reduced dust exposure levels and thus the accompanying respiratory disease burden[2].

WRLD includes a broad range of persisting pathologies occurring from the upper to the lower respiratory tract. They encompass an extensive range of pulmonary diseases comprising obstructive diseases such as work-related asthma and COPD; restrictive diseases such as pulmonary fibrosis and silicosis; and mixed lung diseases such as pneumoconiosis and hypersensitivity pneumonitis[1].

WRLD should be evaluated for respiratory complaints by focusing on crucial questions, for instance, the patient's existing and past occupations, home environment, hobbies, para-occupational exposure, history of presenting illness, and timing of the onset of symptoms[3].

1. Obstructive WRLD

a. Work-related asthma (WRA)

Asthma is the most predominant respiratory illness affecting about 7.7% of employed adults. Exacerbation of asthma is common, with reports from the latest surveys demonstrating that each year approximately half of the individuals with asthma experience a minimum of one episode of deterioration of their asthma symptoms, and 0.17% require hospitalization[4].

WRA is a broad diagnosis covering heterogeneous conditions, with two things in common, the first being the clinical features of asthma and the second the exposure in the workplace, and incorporates both occupational asthma (OA) and work-exacerbated asthma (WEA)[5]. OA is a disease exemplified by inconsistent airflow limitation and inflammation because of the causes and conditions accountable to specific occupational surroundings rather than the stimuli encountered outside of the workplace[6]. While Work-exacerbated asthma is exacerbated by workplace conditions[7].

The assemblage of complaints encountered by individuals with WRA comprises coughing, wheezing, exertional dyspnea, and chest tightness, and is indistinguishable from that seen in patients with asthma without occupational exposures[3].

b. Chronic obstructive pulmonary disorder (COPD)

COPD is characterized by progressive airflow limitation and is related to the pathological inflammatory response when exposed to noxious particles and gases[8]. The most recognized hazardous elements for COPD are cigarette smoking, workplace exposure (wood, coal, cotton, rubber, welding, and cadmium), air pollution, and fumes from burning biomass fuels[9]. The Global Initiative on Obstructive Lung Disease (GOLD) has insinuated that the standard FEV1/FVC ratio of <70% is a suggestion of the presence of COPD[10].

The inhalation of gases and particulates might also initiate nearby inflammatory processes within the airways and lungs, resulting in disorders such as chronic bronchitis, small airway disease, and emphysema. Long-term outcomes are systemic illness and pulmonary hypertension[11].

2. Restrictive WRLD

a. Pulmonary fibrosis (PF)

PF encompasses an extensive collection of lung diseases where the lung parenchyma experiences an irreversible progression of overgrowth, hardening, and scarring usually accredited to excess deposition of extracellular matrix components[12]. The most common form is idiopathic pulmonary fibrosis (IPF) which has the worst prognosis with a survival rate of 2-5 years after the diagnosis[13,14].

The causes include long-term exposure to environmental and occupational hazards such as asbestos, silica, beryllium, coal dust, hard metals, and radiation treatments. Some chemotherapy drugs (methotrexate, bleomycin), anti-inflammatory (rituximab, sulfasalazine), heart medications (amiodarone, propranolol), and antibiotics (nitrofurantoin, ethambutol) in addition to exposure to molds, bacteria, and animal protein[15,16].

Clinical manifestation includes inexplicable chronic exertional dyspnea, cough, inspiratory crackles, and/or clubbing that occur without legitimate or other symptoms. Patients ordinarily reported the gradual onset of dyspnea on exertion and non-productive cough over numerous months[17].

b. Silicosis

Silicosis is occupational pneumoconiosis instigated by exposure to respirable silica-based particles, leading to an inflammatory cascade, gradual lung fibrosis, and hence respiratory failure[18]. The processes that further influence the development of silicosis are lasting inflammation, epithelial-mesenchymal transition (EMT), fibroblast proliferation and activation, and excessive deposition of extracellular matrix[19].

According to Occupational Safety and Health Administration (OSHA), the occupations and industries that cause silica exposure are sandblasting, mining, miller, ceramic working, glassmaker, granite

quarry worker, sand and stone grinding, casting, shakeout, and blasting[20].

Clinical features include the risk of mycobacterial infection, enlarged hilar or mediastinal lymphadenopathy, pleural thickening, spontaneous pneumothorax, weight loss, dyspnea, and cough[21]

The diagnosis confirmation is monitored by clinical examination accompanied by a detailed history of occupational exposure to silica dust. Chest X-ray illustrates the bilateral patch nodular opacities; however, the confirmed diagnosis will be made at a moderate or advanced stage [22].

3. Mixed Obstructive and Restrictive WRLD

a. Pneumoconiosis

Pneumoconiosis is a cluster of diverse fibrotic lung diseases that progresses through the inhalation of mineral dust such as free silica dust, asbestos fibers, dust from coal mines, and mixed silicate dust. The clinical features of the disease are chronic pulmonary inflammation and fibrosis[23]. The main pathological features include chronic pulmonary inflammation and progressive pulmonary fibrosis, which can eventually lead to death caused by respiratory and/or heart failure[24].

The management includes a thorough history related to dust exposure, physical examination, chest radiographs, pulmonary function tests, and an examination for occupational complications by a certified physician [25].

b. Hypersensitivity Pneumonitis

Hypersensitivity pneumonitis (HP) is an inflammation of the lung alveoli and bronchi due to inhaled inorganic bags of dust. The causative agents are mold, paint catalyst, sugar cane and hay dust, mushroom, tobacco, heating and cooling system water, cork dust, beer brewing, plastic residue, etc [26].

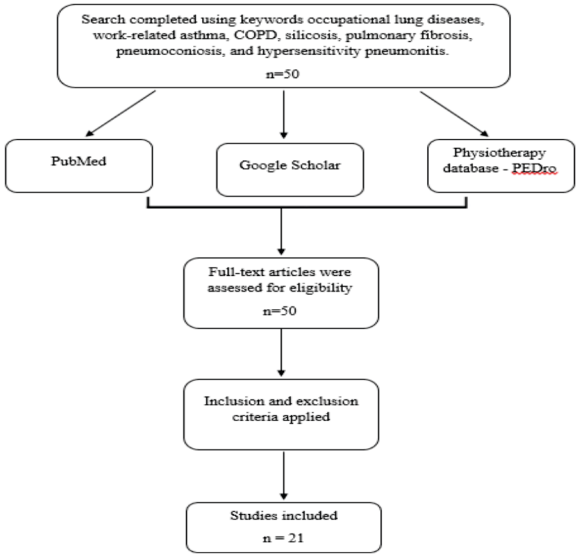
Patients with the non-fibrotic disease will frequently show progressive dyspnea for a few weeks/months collectively with fever, chills, wheezing, chest tightness, and weight loss[27]. While in patients with fibrotic disease, progressive exertional dyspnea and chronic cough will advance in months to years[28].

Diagnosis of hypersensitivity pneumonitis will be based on thorough environmental and occupational history, CT appearances, bronchioloalveolar cell count, and validation of sensitization to an antigen, by specific IGg or triggering antibodies[29].

MATERIALS AND METHODS

Literature Search Methodology

Online search engines that are used to collect journals are Google Scholar, PubMed, and PEDro. The authors identified articles based on the keywords. The articles were collected in full text. A total of 50 articles were identified, out of which 21 articles were selected for review. [Fig-1]



Study Selection

Inclusion criteria:

(1) articles published in English language only; (2) obstructive, restrictive, and mixed occupational lung diseases; (3) articles from 2006-2021 have been taken.

Exclusion criteria:

(1) articles published in other languages; (2) articles published below the year 2006.

S. NO	AUTHORS	TITLE	YEAR	STUDY DESIGN	CONCLUSION
1.	Kevin P. Vlahovich, et al., (1)	A 2019 Update on Occupational Lung Disease: A Narrative Review	2021	Narrative Review	As the occupational burden increases respiratory ailments, the crucial need to increase awareness about occupational lung diseases. With present knowledge, we may be able to forestall the complications from occupational hazards.
2.	Mohd Iskandar Jumat, et al., (3)	Occupational Lung Diseases: A Narrative Review of Lung Conditions from the Workplace	2021	Narrative Review	Prevention and constraint of occupational lung diseases necessitate a collective effort of the employers, workers, occupational and pulmonary physicians, industrial hygienists, and other disciplines members.
3.	Lavinia Clara Del Roio, et al., (30)	Work-related asthma	2021	Review Article	Work-related asthma is underdiagnosed and inefficiently treated. To palliate the weight of the illness, priority should be given to the health care system, worker protection, and the social security system.
4.	Chiara Giacomelli, et al., (12)	Pulmonary fibrosis from molecular mechanisms to therapeutic interventions: lessons from post-COVID-19 patients	2021	Review	Despite current knowledge of fibrosis and covid-19, more research should focus on efficient clinical parameters and biomarkers to stratify ILD and covid-19 patients.
5.	Demin Cheng, et al., (13)	Metformin attenuates silica-induced pulmonary fibrosis via AMPK signaling	2021	Research Article	Metformin helps in decreasing cell toxicity, inflammation, oxidative stress, EMT, and fibroblast activation processes, hence, is a potential therapy for fibrotic diseases.

6.	Jana Adamcakova, et al., (32)	New Insights into Pathomechanisms and Treatment Possibilities for Lung Silicosis	2021	Review	A better understanding of the crucial role of the NLRP3 inflammasome, and its associated changes, cleavage of IL-1β, and the activation of caspase-1 has helped the treatment protocol for silicosis.
7.	Shyam Sundar Nandi, et al., (22)	A rapid point-of-care CC16 kit for screening occupational silica dust-exposed workers for early detection of silicosis/ silico-tuberculosis	2021	Manuscript	Early detection of silicosis can be done by the performance evaluation of lateral flow assay that helps in secondary prevention and financial compensation per the country's guidelines.
8.	Xian-Mei Qi, et al., (24)	Pneumoconiosis: current status and prospects	2021	Review Article	The review abridged the present clinical status along with the research prospects. As the existing research lacks effective prevention protocols, early diagnosis, and disease-specific drug treatments, future research should focus more on these methods.
9.	Maria Laura Alberti, et al., (33)	Hypersensitivity neumonitis: Diagnostic and Therapeutic Challenges	2021	Review Article	As the diagnosis and management of hypersensitivity pneumonitis are a complicated and puzzling due to the absence of diagnostic gold standard protocols, it is needed to focus on large-scale clinical trials.
10.	N. Kongsupon, et al., (29)	Occupational causes of hypersensitivity pneumonitis: a systematic review and compendium	2021	Systematic Review and Compendium	The review has illustrated the list of occupational causative agents and exposures which causes hypersensitivity pneumonitis. They listed a total of 14 occupation categories that will result in hypersensitivity pneumonitis.
11.	Piero Maestrelli, et al., (5)	Causes and Phenotypes of Work-Related Asthma	2020	Review	Work-related asthma jeopardizes long-term health but it is possibly preventable. Clinical, pathological, and functional modifications are analogous tocommon asthma, consequently, assessment and occupational history are necessary. Precise recognition of the illness is vital for managing and preventing the disease.
12.	Silvia Ranzieri, et al., (17)	Idiopathic pulmonary fibrosis and occupational risk factors	2019	Reviw-Article	Numerous pieces of evidence support the hypothesis which states that environmental agents can cause idiopathic pulmonary fibrosis. Assembling pidemiological data may be helpful in better understanding the disease.
13.	Sara De Matteis, et al., (2)	Current and New Challenges in Occupational Lung Diseases.	2017	Author Manuscript	While some studies proposed that work-related allergies can be prevented by limited exposure to the allergen, no schemes are there to promote the occupational safety guidelines. Contributing to occupational disease burden and public and private costs can hasten the occupational exposure standards.
14.	Oziom a S. Chioma, et al., (34)	Role of Microbial Agents in Pulmonary Fibrosis	2017	Review Article	Evidence has shown that infectious pathogens can prompt and exacerbate pulmonary fibrosis, and antimicrobials may be used to improve patients' quality of life. However, further studies are needed to assess the viability of infectious agents after the initiation of pulmonary fibrosis and outline the importance and role of antimicrobials in the management.
15.	Naw Awn, et al., (35)	Japanese workplace health management in pneumoconiosis prevention	2017	Review Article	To increase the efficiency of protecting workers' health, the Pneumoconiosis Law of 1960 posed a comprehensive health management routine for workers employed in dust exposure.
16.	F. Limongi, et al., (26)	Hypersensitivity pneumonitis and alpha-chemokines	2017	Review Article	The difference in the expression of CXC chemokines and Th1 cytokines may interpose to the varied immunopathogenesis, clinical course, and treatment of hypersensitivity pneumonitis.
17.	George Friedman-Jimenez, et al., (36)	Occupational Asthma and Work-Exacerbated Asthma	2015	Review	Management and prevention are highly-effective for work-related asthma. Management includes both pharmacological and non-pharmacological interventions. Prevention includes early diagnosis, education, and decreasing exposure duration.
18.	Anne Kristin Møller Fell, et al., (37)	Work-related COPD	2014	Review Article	Current studies show an increased risk of COPD when exposed to quartz and for employers in cement production and metallurgic smelting industries. Previous studies have long-established that a sequence of organic and inorganic occupational exposures may cause COPD.

19.	Øyvind Omland, et al., (38)	Occupational chronic obstructive pulmonary disease: a systematic literature review	2014	Systematic Review	The studies that were examined were based on different designs, and derivatives of different populations, and applied differing exposure and outcome measures however the result shows that there was a reliable correlation between occupational exposures and COPD.
20.	Kyle Steenland, et al., (39)	Silica: A Lung Carcinogen	2014	Review Article	As silica has established a cause of lung cancer appropriate history-taking is necessary to rule out if the cancer is caused by silica exposure. Preventive measures are required to be followed according to the Occupational Safety and Health Administration (OSHA) guidelines.
21.	Paul K. Henneberger, et al., (7)	An Official American Thoracic Society Statement: Work-Exacerbated Asthma	2011	American Thoracic Society Statement	Work-related asthma is common, however, is also an obscure illness resulting from occupational exposure. Further research should be done to better understand the mechanism, risk factors, and outcomes of Work-related asthma.

DISCUSSION

Occupational lung diseases place major economic burdens on employees, employers, insurance companies, physicians, and the general population. Even though elimination from workplace exposure may help improve the condition, it is not practical to remove the employee.

As the disease is progressing, it is necessary to have evidence-based occupational health standards and laws to protect the workers from developing occupational lung diseases. However, health-based evidence and occupational health standards and laws will never fully protect workers to develop occupational lung diseases.

In this scenario, awareness of OLDs is significant for the workers as well as the employers. Awareness can be raised by proper education about the OLDs and their most common causative agents which include cigarette smoking, metal fumes, gases, coal mine dust, exposure to asbestos and silica, cementing industries, smelting industries, gardeners, carpenters, etc.

A thorough assessment is crucial as it helps with the early diagnosis and distinguishing between the various OLDs. With the help of assessment, it will be effortless to identify the occupational triggers, condition of the patient, and elimination of the exposed particles.

With the advancing medical science technology, it has become unproblematic for the radiologist to identify the condition better alongside other investigations, such as chest X-rays, chest CTs, serological findings, etc. Treatment should focus both on pharma cological and non-pharmacological aspects.

CONCLUSION

Occupational lung diseases remain a major concern for workers across the globe affecting health and economic impacts in industries even with increased occupational safety measures. There is a crucial need to spread awareness among clinicians as well as workers. Education of the workers and employers is necessary to identify the disease. Regular screening is helpful for early diagnosis, treatment, and prevention strategies. Prevention and control of OLDs necessitate a collective effort among employees, employers, physicians, pulmonologists, and members from other disciplines.

REFERENCES

1. Vlahovich, K.P.; Sood, A. A 2019 Update on Occupational Lung Diseases: A Narrative Review. Pulm. Ther. 2021; 7:75-87.
2. Sara De Matteis, Dick Heederik, Alex Burdorf, Claudio Colosio, Paul Cullinan, Paul K. Henneberger, Ann Olsson, Anne Raynal, Jos Rooijackers, Tina Santonen, Joaquin Sastre, Vivi Schlussen, Martie Van Tongeren, and Torben Sigsgaard on behalf of the European Respiratory Society Environment and Health Committee. Eur Respir Rev. Current and new challenges in occupational lung diseases. 2017 December 31; 26(146).
3. Mohd Iskandar Jumat, Firdaus Hayati, Syed Sharizman Syed Abdul Rahim, Sahipudin Saupin, Khamisah Awang Lukman, Mohammad Saffree Jeffree, Helen Benedict Lasimbang, Fairrul Kadir. Occupational lung disease: A narrative review of lung conditions from the workplace. Annals of Medicine and Surgery 64 (2021) 102245.
4. National Center for Health Statistics. Asthma prevalence, health care use, and mortality: the United States, 2003–05. Department of Health and Human Services; 2007.
5. Piero Maestrelli, Paul K. Henneberger, Susan Tarlo, Paolo Mason, Piera Boschetto. Causes and phenotypes of work-related asthma. Int. J. Environ. Res. Public Health 2020, 17, 4713.
6. Malo JL, Chan-Yeung M, Bernstein DI. Asthma in the Workplace. 4th ed. Boca Raton, London, New York: Taylor & Francis Group; 2013.
7. Henneberger PK, Redlich CA, Callahan DB, Harber P, Lemière C, Martin J, et al. An official American thoracic society statement: work-exacerbated asthma. Am J Respir Crit Care Med. 2011;184(3):368- 378.
8. Global Strategy for the Diagnosis, Management, and Prevention of COPD, Global Initiative for Chronic Obstructive Lung Disease. 2013.
9. Salvi SS, Barnes PJ. Chronic obstructive pulmonary disease in non-smokers. Lancet 2009; 374: 733 – 43.
10. Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the diagnosis, management, and prevention of COPD. GOLD; 2008.
11. Hogg JC. Pathophysiology of airflow limitation in chronic obstructive pulmonary disease. Lancet. 2004 Aug 21- 27;364(9435):709–21.
12. Chiara Giacomelli, Rebecca Piccarducci, Laura Marchetti, Chiara Romei, and Claudia Martini. Pulmonary fibrosis from molecular mechanisms to therapeutic interventions: lessons from post-covid 19 patients. Biochemical Pharmacology 193 (2021) 114812.
13. T.M. Maher, A.U. Wells, G.J. Laurent, Idiopathic Pulmonary Fibrosis: Multiple Causes and Multiple Mechanisms? Eur Respir J 30 (5) (2007) 835–839.
14. P.W. Noble, C.E. Barkauskas, D. Jiang, Pulmonary Fibrosis: Patterns and Perpetrators, J Clin Invest 122 (8) (2012) 2756–2762.
15. Nalysnyk L, Cid-Ruzafa J, Rotella P, Esser D. Incidence and prevalence of idiopathic pulmonary fibrosis: a review of the literature. Eur Respir Rev. 2012;21(126):355-61.
16. King TE, Jr., Pardo A, Selman M. Idiopathic pulmonary fibrosis. Lancet. 2011;378(9807):1949-61.
17. Silvia Ranzieri, Elisa Illica Magrini, Paola Mozzoni, Roberta Andreoli, Giovanna Pelà, Giuseppina Bertorelli, Massimo Corradi. Idiopathic pulmonary fibrosis and occupational risk factors. Med Lav 2019; 110, 6: 407-436.
18. Cao Z, et al. A novel pathophysiological classification of silicosis models provides some new insights into the progression of the disease. Ecotoxicol Environ Saf. 2020;202: 110834.
19. Xie Y, Ma J, Yang M, Fan L, Chen W. Extracellular signal-regulated kinase signaling pathway and silicosis. Toxicol Res. 2021;10(3):487–94.
20. Kyle Steenland, Elizabeth Ward. Silica: A Lung Carcinogen. CA CANCER J CLIN 2014; 64:63–69.
21. Antao, V.C.; Pinheiro, G.A.; Terra-Filho, M.; Kavakama, J.; Müller, N.L. High-resolution CT in silicosis: Correlation with radiographic findings and functional impairment. J. Comput. Assist. Tomogr. 2005, 29, 350–356.
22. Shyam Sundar Nandi, Upendra P. Lambe, Kamalesh Sarkar, Sonali Sawant, Jagadish Deshpande. A rapid point-of-care CC16 kit for screening occupational silica dust-exposed workers for early detection of silicosis/ silico-tuberculosis. Scientific reports. 2021; 11:23485.
23. Perret JL, Plush B, Lachapelle P, Hinks TS, Walter C, Clarke P, et al. Coal mine dust lung disease in the modern era. Respirology 2017; 22:662–670.
24. Xian-Mei Qi, Ya Luo, Mei-Yue Song, Ying Liu, Ting Shu, Ying Liu, Jun-Ling Pang, Jing Wang, Chen Wang. Pneumoconiosis: current status and future prospects. Chinese Medical Journal. 2021; 134(8).
25. Ministry of Labour. Handbook of pneumoconiosis health examination. Labor board of health, Industrial safety, and health department, Ministry of Labour. Tokyo, Japan: Government of Japan.
26. F. Limongi, P. Fallahi. Hypersensitivity pneumonitis and alpha-chemokines. Clin Ter 2017; 168 (2): e140-145.
27. Selman M, Pardo A, King TE Jr. Hypersensitivity pneumonitis: insights in diagnosis and pathobiology. Am J Respir Crit Care Med. (2012) 186:314– 24.
28. Varone F, Iovene B, Sgalla G, Calvello M, Calabrese A, Larici AR, et al. Fibrotic hypersensitivity pneumonitis: diagnosis and management. Lung. (2020) 198:429–40.
29. N. Kongsupon, G. I. Walters, S. S. Sadhra. Occupational causes of hypersensitivity pneumonitis: a systemic review and compendium. Occupational Medicine 2021; 71:262–266.
30. Lavinia Clara Del Roio, Rafael Futoshi Mizutani, Regina Carvalho Pinto, Mário Terra-Filho, Ubiratan Paula Santos. Work-related asthma. J Bras Pneumol. 2021;47(4): e20200577.
31. Demin Cheng, Qi Xu, Yue Wang, Guanru Li, Wenqing Sun, Dongyu Ma, Siyun Zhou, Yi Liu, Lei Han, Chunhui Ni. Metformin attenuates silica-induced pulmonary fibrosis via AMPK signaling. J Transl Med (2021) 19:349.
32. Jana Adamcakova, Daniela Mokra. New Insights into Pathomechanisms and Treatment Possibilities for Lung Silicosis. International Journal of Molecular Sciences. 2021, 22, 4162.
33. Maria Laura Alberti, Emily Rincon-Alvarez, Ivette Buendia-Roldan, Moises Selman. Hypersensitivity Pneumonitis: Diagnostic and Therapeutic Challenges. Frontiers in Medicine. (2021) 8: 718299.
34. Ozioma S. Chiomaa, Wonder P. Drake. Role of Microbial Agents in Pulmonary Fibrosis. Yale Journal of Biology and Medicine 90 (2017): pp.219-227.
35. Naw Awn, Momo Imanaka, Narufumi Suganuma. Japanese workplace health management in pneumoconiosis prevention. J Occup Health 2017; 59: 91-103.
36. George Friedman-Jimenez, Denise Harrison, Honghong Luo. Occupational Asthma and Work-Exacerbated Asthma. Semin Respir Crit Care Med 2015;36: 388–407.
37. Anne Kristin Møller Fell, Tor Olav Brøvig Aasen, Johnny Kongerud. Work-related COPD. Journal of the Norwegian Medical Association. 2014; 134: 2158 – 63.
38. Omland Ø, Würtz, Aasen TB, Blanc P, Brisman J, Miller MR, Pedersen OF, Schlünssen V, Sigsgaard T, Ulrik CS, Viskum S. Occupational chronic obstructive pulmonary disease: a systematic literature review. Scand J Work Environ Health 2014;40(1):19-35.
39. Kyle Steenland, Elizabeth Ward. Silica: A Lung Carcinogen. Ca Cancer J Clin 2014; 64:63–69.