



Occupational respiratory and skin diseases among workers exposed to metalworking fluids

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Purpose of review

To examine respiratory and skin diseases that occur among workers exposed to metalworking fluids (MWFs) used during machining processes.

Recent findings

Five cases of a severe and previously unrecognized lung disease characterized by B-cell bronchiolitis and alveolar ductitis with emphysema (BADE) were identified among workers at a machining facility that used MWFs, although MWF exposure could not be confirmed as the etiology. In the United Kingdom, MWF is now the predominant cause of occupational hypersensitivity pneumonitis (HP). Under continuous conditions associated with respiratory disease outbreaks, over a working lifetime of 45 years, workers exposed to MWF at 0.1 mg/m³ are estimated to have a 45.3% risk of acquiring HP or occupational asthma under outbreak conditions and a 3.0% risk assuming outbreak conditions exist in 5% of MWF environments. In addition to respiratory outcomes, skin diseases such as allergic and irritant contact dermatitis persist as frequent causes of occupational disease following MWF exposure.

Summary

Healthcare providers need to consider MWF exposure as a potential cause for work-related respiratory and skin diseases. Additional work is necessary to more definitively characterize any potential association between MWF exposures and BADE. Medical surveillance should be implemented for workers regularly exposed to MWF.

Keywords

B-cell bronchiolitis and alveolar ductitis with emphysema, dermatitis, hypersensitivity pneumonitis, metalworking fluids, work-related asthma

INTRODUCTION

Respiratory and dermatologic diseases associated with metalworking fluid (MWF) exposure were last reviewed in this journal in 2015 [1]. An estimated 1.2 million US workers are potentially exposed to MWFs [2]. MWFs are used during machining processes for the purposes of reducing friction, cooling, preventing corrosion, and removing metal debris [1,3^{***}]. There are several types of MWFs, referred to by different names, which can create confusion in the medical literature (Table 1). Workers are exposed to MWFs through direct skin contact when handling bulk fluids or by inhalation of vapors, mists, or dusts [3^{***}]. MWFs are typically recycled and can become contaminated with microorganisms, which is associated with both a degradation in performance and potentially harmful inhalational exposures to microorganisms and their byproducts [3^{***}]. Biocides can be added to prevent microbial contamination, but exposures to biocides are also associated with adverse health events. The

purpose of this review is to describe a previously unrecognized severe lung disease that occurred among workers exposed to MWF; provide updated data regarding conditions related to MWF exposure, including hypersensitivity pneumonitis (HP), occupational asthma (OA), and dermatitis; and describe controls that can be used to reduce worker exposure to MWF. This article does not review any potential associations between MWF and cancer.

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KEY POINTS

- Five cases of a severe and previously unrecognized lung disease characterized by B-cell bronchiolitis and alveolar ductitis with emphysema (BADE) were identified among never-smoking production workers at a machining facility that used metalworking fluids (MWF) designed to preferentially grow *Pseudomonas pseudoalcaligenes*, although MWF exposure could not be identified as the etiology of the disease cluster.
- A risk assessment for occupational MWF exposure and hypersensitivity pneumonitis (HP) and occupational asthma (OA) determined that if workers were continuously exposed to conditions associated with HP or OA outbreaks, over a working lifetime of 45 years, workers exposed to MWF at 0.1 mg/m³ would have a 45.3% risk of acquiring OA or HP under outbreak conditions and an overall 3.0% risk of OA or HP assuming that outbreak conditions exist in 5% of MWF environments.
- The predominant skin disease related to MWF exposure is occupational contact dermatitis, and because of the number of potential chemicals and compositions of MWFs, identification of the causal irritants or allergens can be difficult.
- Facilities should limit potentially harmful exposures to MWF by reducing airborne mists, maintaining fluid quality, limiting bacterial contamination, and minimizing skin exposures, and implementing a medical monitoring program that includes symptom evaluation and possibly spirometry.

Table 1. Metalworking fluid nomenclature and classifications

Other names	
Metalworking fluid	
Machining fluid	
Metal removal fluid	
Mineral oil	
Cutting fluid	
Cutting oil	
Working fluid	
Type	Definition
Straight mineral oil	
Soluble or emulsified	Combination of oil and water
Semi-synthetic	Combination of oil and water but lower concentration of oil than soluble type
Synthetic	No mineral oils

RESPIRATORY DISEASE

B-cell lymphocytic bronchiolitis, alveolar ductitis and emphysema (BADE)

During 2012–2016, the National Institute for Occupational Safety and Health (NIOSH) evaluated the environmental conditions and respiratory health of workers in a machine-manufacturing facility where MWFs were used [4,5[¶],6]. The NIOSH Health Hazard Evaluation was initiated after an occupational medicine provider realized four patients receiving treatment in a local clinic for severe lung disease were employed at the same workplace, including one worker who underwent lung transplantation [5[¶]]. Careful examination of lung tissue biopsy and explanted lung specimens resulted in the identification of a previously unrecognized occupational lung disease with a unique histopathologic pattern described as B-cell lymphocytic bronchiolitis, alveolar ductitis, and emphysema (BADE) (Fig. 1) [5[¶]]. During the evaluation, a fifth employee who had been employed for < 5 years at the time of initial spirometry in 2013 sought treatment after serial spirometry conducted in 2016 identified a severe decline in forced expiratory volume in one second (FEV1).

At the time of diagnosis, each of the five workers worked in the production areas of the facility and were young (27–50 years), never-smokers, with a constellation of symptoms that included cough, wheeze, exertional dyspnea, spirometric obstruction, and reduced diffusing capacity. Pathological features of the surgical biopsies from four workers and explanted lung tissue specimen from one worker included bronchiolocentric lymphocytic infiltrates with CD20⁺ B-cell primary lymphoid follicles in the bronchioles and alveolar ducts (Fig. 1) [5[¶]]. One of the most striking features shared by all five workers was prevalent centrilobular emphysema on high-resolution computed tomography. Bronchiectasis, bronchial wall thickening, and air trapping were also present. Although the presence of emphysema was an unusual finding among these nonsmoking production workers, emphysematous changes have been reported in other potentially work-related conditions, including chronic HP [7]. Emphysema is also a common component in those with chronic obstructive pulmonary disease or chronic obstructive pulmonary disease (COPD); recent work by Lytras *et al.* concluded that occupational exposures accounted for 21% of COPD cases [8].

The microbial composition of both environmental samples and specimens collected from workers at the machining facility where BADE was identified among production workers was evaluated

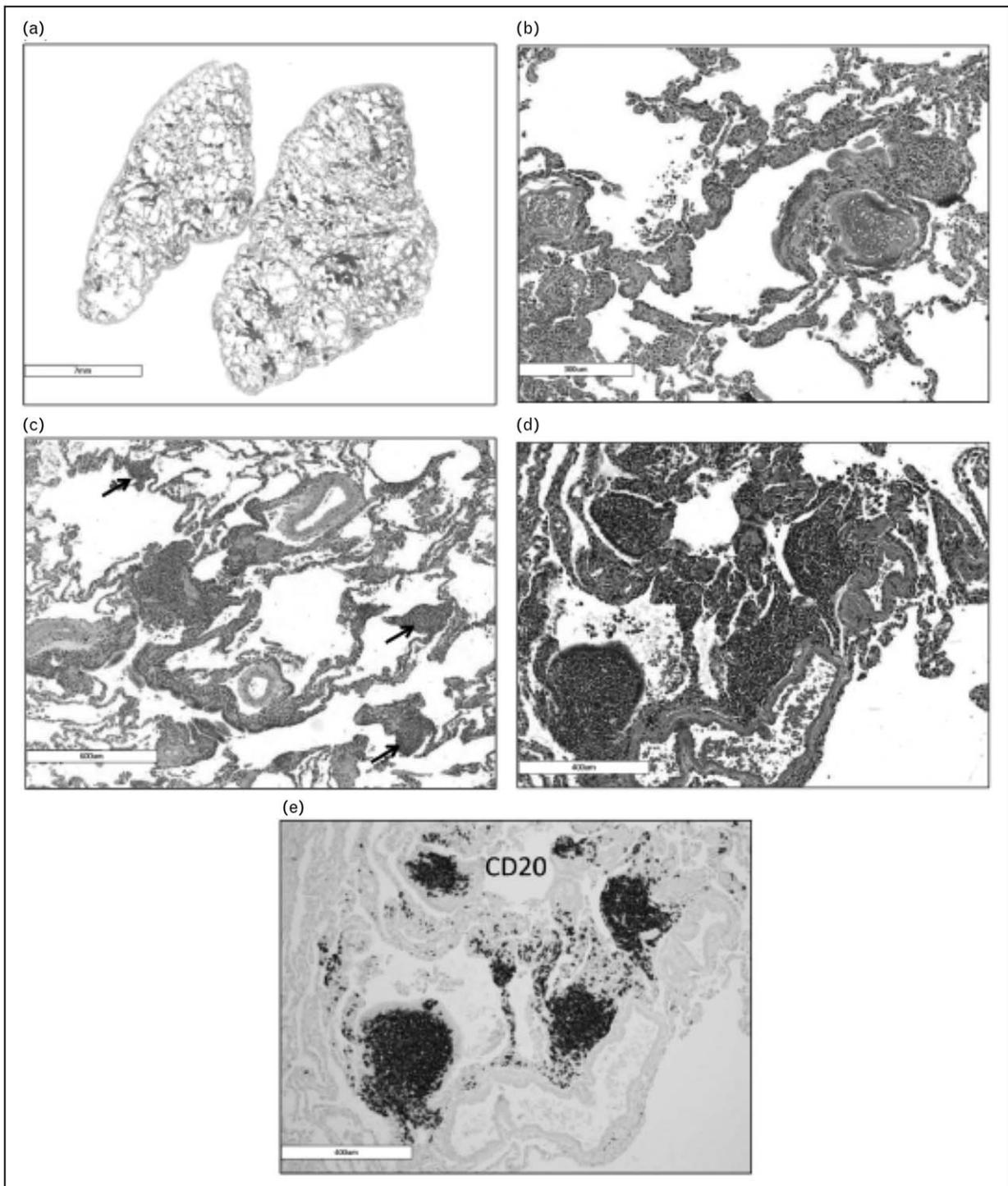


FIGURE 1. Representative hematoxylin and eosin stains of explanted lung tissue and surgical lung biopsies highlight the primary histological features of lymphoplasmacytic infiltrates with primary lymphoid follicles around the distal airways and notable involvement of respiratory bronchioles and alveolar ducts, in addition to diffuse emphysema. (a) Low-power view, highlighting peribronchiolar lymphoid aggregates with widespread emphysema. (b) Medium-power view, showing nonreactive lymphoid follicles with nodular extensions into the alveolar ducts (arrows); emphysema also appreciated. (c, d) High-power views of nodular lymphoid aggregates around a respiratory bronchiole and chronic inflammatory infiltrates expanding the walls of the alveolar ducts. (e) Immunohistochemical staining for CD20, a B-cell marker, demonstrating B cells make up the majority of the primary follicles. [Reprinted with permission from Cummings *et al.* [5[¶]]].

to gain a better understanding of the impact of workplace environment on health [9]. The main MWF used at the facility when samples were collected was a bioconcept MWF engineered to promote the growth of one organism, *Pseudomonas pseudoalcaligenes*, which was considered by the manufacturer to be a nonharmful bacterial species that outcompetes other potentially harmful bacteria and preserves the condition and stability of the MWF [10,11]. The nasal, oral, and skin microbiota of workers who worked in the machine shop where MWF were used, compared with workers from other areas of the facility, had greater similarity to the microbiota present in the MWF specimens, and *Pseudomonas* was the dominant taxon [9]. Further, exposure of murine B-cells to bulk fluid MWF samples obtained at the facility resulted in B-cell proliferation, which is of concern considering B-cell follicles is a characteristic of BADE. These findings indicate that further investigation is needed to characterize the relationship, if any, between inhalational exposure to MWF engineered to promote the growth of *P. pseudoalcaligenes* and BADE, including active surveillance for disease in multiple settings other than the facility where the BADE was described, along with animal exposure studies.

Hypersensitivity pneumonitis

MWF is now the predominant cause of occupational HP in the United Kingdom [12]. Approximately 35% of the 202 cases of occupational HP reported to surveillance of work-related and occupational respiratory disease during January 1996–2015 were attributed to MWF, with the majority associated with outbreaks [13]; this equated to an estimated 818 cases during the study timeframe after accounting for cases submitted by chest physicians using a sampling approach that included a core group of physicians with an interest in occupational respiratory diseases who submitted all eligible cases and the other participating physicians who submitted all eligible cases for one month of the year only [14]. Using the estimated 818 cases that occurred during the study timeframe and assuming 35% were caused by MWF, the annual incidence in the United Kingdom of occupational HP attributed to MWF during the first three 5-year intervals was ~0.5 cases per 1 million workers and decreased to 0.4 cases per 1 million workers during 2011–2015. Further, 36 (17%) of 206 total cases of HP identified at a United Kingdom tertiary care center were caused by MWF exposure [12]. Compared with HP cases from other causes, MWF-related HP was significantly associated with male sex, younger age, acute/subacute disease onset, fever, weight loss, systemic features or recurrent symptoms, crackles on examination, higher median

percentage predicted values for forced vital capacity (FVC) and diffusing capacity of the lung for carbon monoxide (DLCO), more frequent lymphocytes on bronchoalveolar lavage cytology, and less reticulation on computed tomography.

Two case reports of HP were published that demonstrated substantial growth of *P. pseudoalcaligenes* in contaminated MWF recovered from each of the patient's worksites [15,16]. One patient also underwent precipitin testing for 20 antigens; the only strong positive was to *P. pseudoalcaligenes* [15]. These two cases are noteworthy considering the five cases of BADE presented above occurred in a facility that used MWF designed to preferentially allow growth of *P. pseudoalcaligenes* [5[■]], and brings into question the safety of inhalational exposures to MWF contaminated with this bacteria. Using specific inhalational challenge (SIC) tests is the only method for clearly establishing a particular antigen or agent is the cause of HP [17]. Unfortunately, SICs must be performed in a controlled setting and are only available at select tertiary care settings. Additionally, it can be challenging to establish a specific organism is the cause of HP considering antigens produced under stress in contaminated MWF in a workplace differ from those produced in a laboratory setting [17]. The use of recombinant antigens specific to MWF can be used for SIC to aid in establishing a definitive diagnosis [17,18].

In 2019, Park published a risk assessment for MWF exposure and respiratory disease [3[■]]. Incidence rates for HP and OA were combined because both diseases often occur together in MWF-related outbreaks. The assessment estimated that if workers were continuously exposed to conditions associated with HP or OA outbreaks over a working lifetime of 45 years, which includes MWF exposure at 0.1 mg/m³, their risk of acquiring OA or HP would be 45.3%, and their overall risk of OA or HP would be 3.0% assuming a 5% prevalence of outbreak conditions. These findings underscore the need to conduct medical surveillance for workers exposed to MWF, even at levels below occupational exposure limits. MWF-related HP should be considered in any worker exposed to MWF who develops interstitial lung disease of unknown etiology; establishing a diagnosis requires a multidisciplinary team approach [19]. Each case of occupational HP should be treated as a sentinel event prompting the evaluation of the workplace and assessment of the workforce's respiratory health.

Asthma

In recent years, reporting of OA following MWF exposure has decreased. Rosenman posited that

improved ventilation controls and enclosure of newer machines with local exhaust ventilation reduced the level of exposure to chemical sensitizers and thus the potential risk of sensitization [1].

Some studies have evaluated peak expiratory flow (PEF) measurements following an OA outbreak attributed to MWFs. Burge *et al.* examined serial PEF measurements following exposure to MWF in a UK factory [20]. Workers identified as having OA or allergic alveolitis underwent serial PEF measurements and were found to have significant reductions in PEF. However, PEF measurements could not distinguish workers with OA from those with alveolitis. Immediate declines in PEF were notable in OA, whereas late reactions were more consistent with a diagnosis of alveolitis. Workers with both OA and alveolitis also exhibited decrements in FEV₁, FVC, and DLCO. Ilgaz *et al.* performed serial PEF measurements on workers with sensitizer-induced OA caused by MWF aerosols for the purpose of evaluating trends in PEF performance before and after work following the implemented use of respiratory protective equipment [21]. The authors demonstrated that following sensitization, respiratory protective equipment prevented significant reductions in PEF measurements in the majority of workers with OA, but only eliminated work-related decreases in PEF in 25% of workers. These findings highlight the need for source control in industrial settings that use MWF and the need for ongoing monitoring of symptoms and spirometry after respiratory protection is implemented.

Respiratory irritation and symptoms consistent with asthma (e.g., shortness of breath, chest tightness, wheeze) are common among workers who experience MWF exposures. In addition to the lifetime risk of adverse respiratory health effects from HP and OA studied by Park, exposure to MWF at 0.1 mg/m³ also resulted in excess risk of FEV₁ decline [3^{••}]. Therefore, use of spirometry as an adjunct to medical surveillance might be warranted because early identification of changes in FEV₁ could indicate sensitization or potentially COPD.

DERMATOLOGIC DISEASE

Skin contact with MWF can occur before, during, or after machining processes. Exposures to either petroleum-based or water-based MWFs are well-known causes of occupational skin disease, most commonly resulting in contact dermatitis. Occupational contact dermatitis, primarily irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD), is thought to cause 90–95% of all occupational skin diseases [22].

MWFs are a broad class of chemicals and can contain a variety of chemical ingredients with

variable composition. This varied chemical exposure often makes it difficult to distinguish the exact causal agent resulting in skin disease. However, allergens responsible for ACD can be identified using patch testing. Since 2014, a number of case studies have described occupational dermatitis in metalworkers. These chemicals constitute recognized occupational allergens within MWFs, and include N-butyl-1; 2-benzisothiazolin-3-one; 2-butyl-1,2-benzisothiazol-3 (2H)-one; 2-amino-2-methyl-1-propanol; colophonium; and methylchloroisothiazolinone/methylisothiazolinone [23–26]. A study by Alinaghi *et al.* (2020) found that considerable concentrations of metals that can cause ACD, including chromium, nickel, and cobalt, can be found in both used and unused MWFs, presenting an additional potential allergen exposure for metalworkers [27].

Skin diseases continue to be the predominant diagnoses related to occupational exposure to MWF. A retrospective analysis of 39,331 patch-tested production workers in North America revealed that the prevalence of ACD and ICD was significantly higher in production workers than nonproduction workers [28]. Warshaw *et al.* identified MWF as a common allergen source resulting in 6.8% of positive patch test results among production workers.

In Germany, a cohort study used questionnaire data to identify MWF exposures in 230 metalworkers during 2012–2017 [29]. Of the 230 metalworkers identified, 188 (81.7%) were suspected to have occupational dermatitis, of which 138 had self-reported work exposure to MWF. Machining activities were the predominant activities reported among this cohort of metalworkers. The most common diagnosis among the 230 metalworkers was chronic ICD (39.6%), followed by atopic dermatitis (23.5%) and ACD (14.8%). Of the 58 metalworkers with occupational hand dermatitis that underwent patch testing, 11 (18.7%) tested positive for reactivity to water-based MWFs.

In Switzerland, Koller *et al.* carried out a descriptive analysis of occupational diseases associated with MWF that occurred during 2004–2013 [30]. The authors identified 1385 cases of occupational diseases attributable to MWF; of these, 1280 (92%) were skin disease. Consistent with other studies, contact dermatitis was the most commonly diagnosed skin disease ($n = 1261$, 98.5%). A trend analysis demonstrated the prevalence of occupational skin disease related to MWF decreased over the period of study. The authors attributed this decline to intensive efforts to improve skin protection in Swiss workplaces, and a likely reduction in skin exposure although attempting to prevent inhalational exposures and respiratory outcomes.

In a review of risk assessment and respiratory outcomes, Park noted a prevalence of dermal disorders of 0.10–0.85 per 1000 in studies following acute outbreaks of respiratory diseases from MWFs [3^{***}]. Park noted that regulating MWF exposures known to cause respiratory effects could also substantially reduce dermal exposures through the implementation of engineering controls and additional measures. As facilities strive to reduce MWF exposures and consider implementing medical surveillance programs for exposed workers, an assessment of skin symptoms should accompany respiratory symptoms as a part of the evaluation process.

EXPOSURE CONTROL

Machining activities in manufacturing facilities necessitate the use of MWFs. The identification of a previously unrecognized and severe lung disease like BADE, and continued reports of HP, asthma, work-related symptoms including nasal symptoms and wheeze, and dermatitis [1,6,17,29,31–34], reinforce the need for diligent exposure control in settings where MWF are used. Key components of an exposure control strategy include maintaining fluid quality, limiting bacterial contamination, limiting airborne mists, and minimizing skin exposure to MWF.

Facilities that use MWF should have a fluid management system that includes a fluid maintenance schedule. MWFs are commonly colonized with a variety of microorganisms requiring monitoring and control. MWFs with bacterial concentrations $> 10^6$ colony-forming units per milliliter (CFU/mL) are poorly controlled and should be addressed immediately, which might require draining and a thorough cleaning of the system [35]. Facilities using a bioconcept fluid should consult with the fluid manufacturer for guidance on proper management.

The proper design and operation of MWF delivery systems can minimize mist and vapor generation. Exhaust ventilation systems can prevent the recirculation of contaminants in workplace air. NIOSH recommends limiting exposures to MWF aerosols to 0.4 mg/m^3 for the thoracic particulate mass as a time-weighted average concentration for up to 10h per day during a 40-h workweek [36]. Employees should minimize skin contact with MWFs as much as possible to prevent skin disorders. Facilities that use MWFs should consider implementing medical monitoring that includes a regular assessment of skin and respiratory symptoms, and possibly spirometry.

CONCLUSION

Additional work is necessary to more definitively characterize any potential association between

BADE and inhalational exposures to MWF designed to allow for preferential growth of *P. pseudoalcaligenes*. Likewise, more work is also necessary to understand the role inhalational exposure plays regarding MWF contaminated with *P. pseudoalcaligenes* and HP. Contact dermatitis continues to persist as a common occupational disease following MWF exposure, with MWF components continually identified as causal allergens. Facilities should limit potentially harmful exposures to MWF by reducing airborne mists to as low as feasible, maintaining fluid quality, limiting bacterial contamination, and minimizing skin exposures.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

1. Rosenman K. Occupational diseases in individuals exposed to metal working fluids. *Curr Opin Allergy Clin Immunol* 2015; 15:131–136.
2. National Institute for Occupational Safety and Health (NIOSH). Metalworking fluids 2013. Available from: <https://www.cdc.gov/niosh/topics/metalworking/default.html> [Accessed 7 September 2020].
3. Park RM. Risk assessment for metal working fluids and respiratory outcomes. ■ *Saf Health Work* 2019; 10:428–436.
4. Cummings KJ, Stanton ML, Kreiss K, et al. Work-related adverse respiratory health outcomes at a machine manufacturing facility with a cluster of bronchiolitis, alveolar ductitis and emphysema (BADE). *Occup Environ Med* 2020; 77:386–392.
5. Cummings KJ, Stanton ML, Nett RJ, et al. Severe lung disease characterized by lymphocytic bronchiolitis, alveolar ductitis, and emphysema (BADE) in industrial machine-manufacturing workers. *Am J Ind Med* 2019; 62:927–937.

First paper to describe a severe lung disease characterized by B-cell bronchiolitis and alveolar ductitis with emphysema (BADE) that occurred among five workers at a metal machining facility.

6. NIOSH. Health hazard evaluation report: evaluation of exposures and respiratory health concerns in a paper converting equipment manufacturing facility. 2019. By Stanton ML, Nett RJ. Morgantown, WV: U.S. Department of Health and Human Services (DHHS), Centers for Disease Control and Prevention (CDC), NIOSH, NIOSH HHE Report No. 2012-0055-3337.
7. Baqir M, White D, Ryu JH. Emphysematous changes in hypersensitivity pneumonitis: a retrospective analysis of 12 patients. *Respir Med Case Rep* 2018; 24:25–29.

8. Lytras T, Kogevinas M, Kromhout H, *et al.* Occupational exposures and 20-year incidence of COPD: the European Community Respiratory Health Survey. *Thorax* 2018; 73:1008–1015.
9. Wu BG, Kapoor B, Cummings KJ, *et al.* Evidence for environmental-human microbiota transfer at a manufacturing facility with novel work-related respiratory disease. *Am J Respir Crit Care Med* 2020; 202:1678–1688.
10. Dilger S, Fluri A, Sonntag HG. Bacterial contamination of preserved and nonpreserved metal working fluids. *Int J Hyg Environ Health* 2005; 208:467–476.
11. Swisslube B. Blasocut 2020. Available from: https://www.blaser.com/en_US/metalworking/our-solutions/water-miscible-coolants [Accessed 15 September 2020].
12. Walters GI, Mokhles JM, Moore VC, *et al.* Characteristics of hypersensitivity pneumonitis diagnosed by interstitial and occupational lung disease multidisciplinary team consensus. *Respir Med* 2019; 155:19–25.
13. Barber CM, Wiggans RE, Carder M, Agius R. Epidemiology of occupational hypersensitivity pneumonitis; reports from the SWORD scheme in the UK from 1996 to 2015. *Occup Environ Med* 2017; 74:528–530.
14. McDonald JC, Chen Y, Zekveld C, Cherry NM. Incidence by occupation and industry of acute work related respiratory diseases in the UK, 1992–2001. *Occup Environ Med* 2005; 62:836–842.
15. Bellanger AP, Morisse-Pradier H, Reboux G, *et al.* Hypersensitivity pneumonitis in a cystic fibrosis patient. *Occup Med* 2019; 69:632–634.
16. Moniodis A, Hamilton T, Racila E, *et al.* Hypersensitivity pneumonitis in a high school teacher. *Occup Med* 2015; 65:598–600.
17. Burge PS. Hypersensitivity pneumonitis due to metalworking fluid aerosols. *Curr Allergy Asthma Rep* 2016; 16:59.
18. Bellanger AP, Reboux G, Rouzet A, *et al.* Hypersensitivity pneumonitis: a new strategy for serodiagnosis and environmental surveys. *Respir Med* 2019; 150:101–106.
19. Quirce S, Vandenplas O, Campo P, *et al.* Occupational hypersensitivity pneumonitis: an EAACI position paper. *Allergy* 2016; 71:765–779.
20. Burge PS, Moore VC, Burge CB, *et al.* Can serial PEF measurements separate occupational asthma from allergic alveolitis? *Occup Med* 2015; 65:251–255.
21. Ilgaz A, Moore VC, Robertson AS, *et al.* Occupational asthma; the limited role of air-fed respiratory protective equipment. *Occup Med* 2019; 69:329–335.
22. Diepgen TL, Coenraads PJ. The epidemiology of occupational contact dermatitis. *Int Arch Occup Environ Health* 1999; 72:496–506.
23. Dahlin J, Isaksson M. Occupational contact dermatitis caused by N-butyl-1,2-benzisothiazolin-3-one in a cutting fluid. *Contact Dermatitis* 2015; 73:60–62.
24. Foti C, Romita P, Stufano A, *et al.* Occupational allergic contact dermatitis caused by 2-butyl-1,2-benzisothiazol-3(2H)-one in a metalworker. *Contact Dermatitis* 2019; 81:463–465.
25. Geier J, Forkel S, Heetfeld A, *et al.* Contact allergy to 2-amino-2-methyl-1-propanol in a metalworking fluid. *Contact Dermatitis* 2019; 80:323–324.
26. Pesonen M, Suuronen K, Suomela S, Aalto-Korte K. Occupational allergic contact dermatitis caused by colophonium. *Contact Dermatitis* 2019; 80:9–17.
27. Alinaghi F, Hedberg YS, Zachariae C, *et al.* Metals in used and unused metalworking fluids: X-ray fluorescence spectrometry as a screening test. *Contact Dermatitis* 2020; 83:83–87.
28. Warshaw EM, Hagen SL, DeKoven JG, *et al.* Occupational Contact Dermatitis in North American Production Workers Referred for Patch Testing: Retrospective Analysis of Cross-Sectional Data From the North American Contact Dermatitis Group 1998 to 2014. *Dermatitis* 2017; 28:183–194.
29. Schubert S, Brans R, Reich A, *et al.* Assessment of occupational exposure and spectrum of contact sensitization in metalworkers with occupational dermatitis: results of a cohort study within the OCCUDERM project. *J Eur Acad Dermatol Venereol* 2020; 34:1536–1544.
30. Koller MF, Pletscher C, Scholz SM, Schneuwly P. Metal working fluid exposure and diseases in Switzerland. *Int J Occup Environ Health* 2016; 22:193–200.
31. NIOSH. Evaluation of metalworking fluid exposure and dermatitis among rifle barrel manufacturing employees. 2019. By Tapp LC, Broadwater K, Mueller CA. Cincinnati, OH: U.S. DHHS, CDC, NIOSH, NIOSH Health Hazard Evaluation Report 2014-0170-3263, <http://www.cdc.gov/niosh/hhe/reports/pdfs/2014-0170-3263.pdf>.
32. NIOSH. Investigation of dermal and respiratory exposures to metalworking fluids at an automotive parts manufacturer. 2016. By Harney JM, Tapp L. Cincinnati, OH: U.S. DHHS, CDC, NIOSH, NIOSH Health Hazard Evaluation Report 2013-0075-3264, <http://www.cdc.gov/niosh/hhe/reports/pdfs/2013-0075-3264.pdf>.
33. NIOSH. Evaluation of metalworking fluid exposure, dermatitis, respiratory symptoms, and psychosocial factors in an engine machining plant. 2018. By Beaucham C, Tapp L, Wiegand D, Couch J, Mueller C. Cincinnati, OH: U.S. DHHS, CDC, NIOSH, NIOSH Health Hazard Evaluation Report 2015-0070-3304, <https://www.cdc.gov/niosh/hhe/reports/pdfs/2015-0070-3304.pdf>.
34. Wiggans RE, Barber CM. Metalworking fluids: a new cause of occupational nonasthmatic eosinophilic bronchitis. *Thorax* 2017; 72:579–580.
35. Health and Safety Executive. Metalworking fluids: bacterial contamination 2020. Available from: <https://www.hse.gov.uk/metalworking/bacterial.htm> [Accessed 6 September 2020].
36. NIOSH. Criteria for a recommended standard: occupational exposure to metalworking fluids. 1998. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, NIOSH, DHHS (NIOSH) Publication No. 98-102.