ORCID IDs: 0000-0003-0337-406X (R.B.); 0000-0002-6416-1466 (J.H.); 0000-0002-3761-2778 (G.B.); 0000-0002-4688-4927 (I.M.); 0000-0002-0051-9107 (M.W.); 0000-0002-4288-5973 (I.D.P.).

*Corresponding author (e-mail: richard.beasley@mrinz.ac.nz).

Reference

1. Beasley R, Harper J, Bird G, Maijers I, Weatherall M, Pavord ID. Inhaled corticosteroid therapy in adult asthma: time for a new therapeutic dose terminology. *Am J Respir Crit Care Med* 2019;199: 1471–1477.

Occupational Burden in Chronic Respiratory Disease: Call for Recognition, Training, and Data Capture

To the Editor:

We read with great interest the recently published work entitled "The Occupational Burden of Nonmalignant Respiratory Diseases. An Official American Thoracic Society and European Respiratory Society Statement" (1). In this statement, Blanc and colleagues summarize the literature regarding the contribution of occupational exposures to a variety of chronic lung disorders. The authors pool previously published data to derive the population-attributable fraction (PAF) and estimate the contribution of meaningful exposures to individual pulmonary diseases. The proportion of patients with interstitial lung disease (ILD) and occupational exposures was of particular interest to us. In patients with idiopathic pulmonary fibrosis (IPF), the pooled PAF was highest for vapors, gas, dust, or fumes (at 26%), followed by metal dusts or fumes (8%), wood dusts (4%), and silica (3%). The PAF for work-related exposures was 19% for hypersensitivity pneumonitis (HP) and 30% for sarcoidosis. Although they were not addressed in the statement, inhalational exposures and environmental antigens have been known to stimulate the immune system and contribute to the development of autoimmune disease, most notably in rheumatoid arthritis from cigarette smoking and coal dust exposures, as well as systemic sclerosis from inhalation of silica particles (2, 3). At our tertiary referral center, systematic history taking has elicited inhalational exposures in the majority of patients with ILD, not only in those with IPF and HP but also in patients with connective tissue disease–related ILD and unclassifiable ILD (4).

Although occupational asthma has long been known to constitute a significant minority of all-comers with adult asthma, and workplace exposures are now recognized to cause chronic obstructive pulmonary disease, especially in nonsmokers, the environmental antigens that are usually believed to be causative in ILD, such as birds and mold, are often attributed to exposures in the home (5, 6). In contrast, the relatively high PAFs for occupational exposures in IPF and HP reported in this statement correlate with our own experience in our clinic, where we frequently see firefighters, construction workers, and metalworkers who have received multidisciplinary diagnoses of IPF or HP. A systematic review at our center showed that 45% of patients with IPF and 48% of patients with HP had a self-reported history of an exposure classified as occupational. Beyond the diagnoses mentioned in the consensus statement, 41% of the patients in that review had a connective tissue disease–related ILD and a history of potentially relevant work-related exposures. In addition, we see patients across all ILD diagnoses who are employed in workplaces that are not typically thought of as high risk, such as schools, hospitals, and hotels, which may be old, poorly maintained, or frequently remodeled, and thus are a potential source of dust and microbial antigen exposure.

The American Thoracic Society/European Respiratory Society statement is an important step toward increasing the pulmonary community's understanding of the role of inhalational exposures in chronic nonmalignant lung diseases. It is likely that increased awareness will confirm our personal clinical observation that the occupational burden in diffuse parenchymal lung disease is greater than was previously recognized and may extend to patients with all types of ILD, including that associated with autoimmunity. The data support the routine assessment of exposures in a systematic and comprehensive fashion in patients with chronic lung disease and necessitate formal training in occupational medicine for all pulmonary fellows and additional education for current clinical practitioners. For the research community, the identification of inhalational exposures in the home and workplace provides insight into disease pathogenesis and suggests future investigations, but is dependent on the data being elicited and captured in multicenter studies and national registries. The goal of this training and research should be effective interventions to modify the disease course, with prevention of disease onset being the ultimate holy grail. ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

Cathryn T. Lee, M.D.*
Mary E. Strek, M.D.
University of Chicago
Chicago, Illinois

*Corresponding author (e-mail: cathryn.lee@uchospitals.edu).

References

1. Blanc PD, Annesi-Maesano I, Balmes JR, Cummings KJ, Fishwick D, Miedinger D, *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.
2. Schmajuk G, Trupin L, Yelin E, Blanc PD. Prevalence of arthritis and rheumatoid arthritis in coal mining counties of the U.S. *Arthritis Care Res (Hoboken)* 2019;71:1209–1215.
3. De Decker E, Vanthuyne M, Blockmans D, Houssiau F, Lenaerts J, Westhovens R, *et al*. High prevalence of occupational exposure to solvents or silica in male systemic sclerosis patients: a Belgian cohort analysis. *Clin Rheumatol* 2018;37:1977–1982.

Originally Published in Press as DOI: 10.1164/rccm.201907-1288LE on July 25, 2019

4. Lee C, Adegunsoye AO, Chung J, Jablonski R, Pan I, Montner S, *et al*. Identification of predisposing environmental risk factors for interstitial lung disease [abstract]. *Am J Respir Crit Care Med* 2019;199:A1454.
5. Knoeller GE, Mazurek JM, Moorman JE. Work-related asthma among adults with current asthma in 33 states and DC: evidence from the Asthma Call-Back Survey, 2006-2007. *Public Health Rep* 2011;126: 603–611.
6. De Matteis S, Jarvis D, Hutchings S, Darnton A, Fishwick D, Sadhra S, *et al*. Occupations associated with COPD risk in the large population-based UK Biobank cohort study. *Occup Environ Med* 2016;73:378–384.

Reply to Lee and Strek

From the Authors:

We welcome the comments by Dr. Lee and Dr. Strek calling for increased recognition, training, and data capture to address unrecognized occupational attributions in response to the recently published work entitled "The Occupational Burden of Nonmalignant Respiratory Diseases. An Official American Thoracic Society and European Respiratory Society Statement." Indeed, those were implicit goals that led our international taskforce members to undertake this in-depth review (1). From the onset, we recognized that in focusing on selected nonmalignant respiratory conditions, there would be other occupational associations that we would not be able to address in depth. For example, Table E2 in the online supplement of Reference 1 describes a number of other pulmonary disorders associated with occupational exposures (with supporting citations in the main publication). These include acute eosinophilic pneumonia, bronchiolitis (obliterative, proliferative, and lymphocytic), cryptogenic organizing pneumonia, desquamative interstitial pneumonia, diffuse pulmonary hemorrhage, lipoid pneumonia, nonspecific interstitial pneumonia, and respiratory bronchiolitis interstitial lung disease (1). Many of these disorders fall into the interstitial lung disease group emphasized in the letter by Dr. Lee and Dr. Strek.

The authors also raise the important point that autoimmune conditions such as rheumatoid arthritis may not be appreciated as occupationally triggered by silica and other workplace exposures. In that context, it is important to note that concomitant lung disease may not be overt in such syndromes (2). It is our hope that further research and in-depth reviews will continue to shed light on underappreciated occupational contributors to disease and prioritize a reduction in the burden of these preventable conditions. ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

Paul D. Blanc, M.D., M.S.P.H*
University of California, San Francisco
San Francisco, California

Carrie A. Redlich, M.D., M.P.H
Yale University School of Medicine
New Haven, Connecticut

On behalf of all the authors

ORCID IDs: 0000-0001-9043-175X (P.D.B.); 0000-0001-6486-6725 (C.A.R.).

*Corresponding author (e-mail: paul.blanc@ucsf.edu).

References

1. Blanc PD, Annesi-Maesano I, Balmes JR, Cummings KJ, Fishwick D, Miedinger D, *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.
2. Murphy D, Hutchinson D. Is male rheumatoid arthritis an occupational disease? A review. *Open Rheumatol J* 2017;11: 88–105.

Expression of Concern: Inhaled Ethyl Nitrite Prevents Hyperoxia-impaired Postnatal Alveolar Development in Newborn Rats

The *Journal* is publishing this expression of concern about an article in the August 1, 2007, issue (1) because of potential problems with the reliability of its data. The authors have informed us that, although the data appear to be accurate, they have uncertainty about the validity of the data described in Figure 7 because of irregularities in the procedures of a lab that generated those data. ■

Reference

1. Auten RL, Mason SN, Whorton MH, Lampe WR, Foster WM, Goldberg RN, Li B, Stamler JS, Auten KM. Inhaled ethyl nitrite prevents hyperoxia-impaired postnatal alveolar development in newborn rats. *Am J Respir Crit Care Med* 2007;176:291–299.

Originally Published in Press as DOI: 10.1164/rccm.201907-1379LE on July 25, 2019

This article is open access and distributed under the terms of the Creative Commons Attribution Non-Commercial No Derivatives License 4.0 (http://creativecommons.org/licenses/by-nc-nd/4.0/). For commercial usage and reprints, please contact Diane Gern (dgern@thoracic.org).