mining exposure had more than threefold odds of reporting a diagnosis of RA, leading to a population attributable fraction of 33% of those studied (2). Military inorganic dust exposure has also been associated with the development of RA compared with other occupations within the armed forces (3). Prolonged exposure to the World Trade Center disaster site has also been associated with an increased rate of autoimmune disease, the most prevalent of which was RA (4). Other occupations have demonstrated gender-specific associations with RA: bricklayers and concrete workers have an increased risk of RA among men, whereas nurses and medical attendants have increased risk among women (5).

Occupational-associated lung disease in patients with connective tissue disease is increasingly recognized (6), and the well-known association between occupational exposures and asthma, COPD, and bronchiolitis suggest a shared causative environmental antigen may exist among patients with RA and airway disease. Ascertaining the association between RA and occupational exposures has enormous implications for assessment of those at high risk of disease development, such as individuals with a family history of autoimmune disease. Furthermore, more novel occupational exposure–pulmonary disease dyads in women may reveal more exposure–autoimmune disease connections in the future, given the female-predominant nature of connective tissue disease. Systematic assessment of lifetime occupational exposures, starting in clinic and continuing in research registries, is the key to further codifying exposure–disease relationships and early identification and ultimately prevention of airway disease in patients with RA.

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

Reply: Occupational Exposures in Rheumatoid Arthritis-related Airway Disease: A Missing Link?

From the Authors:

We thank Drs. Lee and Strek for their thoughtful reading of our review highlighting the current knowledge of airway disease manifestations in patients with rheumatoid arthritis (RA) (1). In their response to our review, Drs. Lee and Strek aptly highlight an unexplored part of this discussion: the association between occupational and inhalational exposures beyond cigarette smoking and the development of RA. In fact, this paradigm of exposure to concentrated inhalational exposure such as seen in World Trade Center disaster site workers, followed by the development of RA, fitfully supports one of the conceptual models we presented in our review article. In our proposed model of RA autoimmunity development, bronchus-associated lymphoid tissue responds to local inflammatory pressures that can be triggered by a variety of inhaled factors with the production of RA autoantibodies such as antibodies to citrullinated peptide antigens. In this review, we highlighted the potential role of neutrophil extracellular trap formation (NETosis) in connecting chronic lung inflammation to development of antibodies to citrullinated peptide antigens, and although cigarette smoking has been shown to induce NETosis in models, there is a paucity of data regarding the role of other inhalational injuries on NETosis (2).

We read the recent publication from Drs. Lee and Strek regarding the spectrum of occupational and inhalational exposures found in a large interstitial lung disease registry with great interest (3). We believe their data highlight the underexplored role of inhalational exposures in chronic lung disease regardless of etiology, including RA lung manifestations such as interstitial lung disease (3). The themes in Drs. Lee and Strek’s response highlight the need to understand the role of inhalational injury in NET formation and subsequent development of autoimmunity across chronic lung disease states. Importantly, NET cargo release varies based on the antigenic trigger (4); therefore, it would follow that unique inhalational risks may confer specific autoimmune risk based on the nature of the NET response induced.

We believe, as Dr. Lee and Strek point out, that ongoing clinical exploration of lung disease in patients with RA, including inhalational exposure assessment, remains paramount when approaching the many questions remaining in this realm. Airway disease in RA...
represents a broad set of manifestations with several potential pathways of development and many unanswered questions for screening, diagnosis, and treatment.

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Possible Alternate Explanation for Cases of Idiopathic Pulmonary Fibrosis

To the Editor:

In their paper on idiopathic pulmonary fibrosis (IPF) among U.S. veterans, Kaul and colleagues may have missed another set of factors possibly leading to an incorrect diagnosis among those found to have IPF (1). Exposure to various dusts, including asbestos, has been well documented as looking both radiologically and pathologically like IPF.

It would have been of interest to examine if navy veterans, most likely to have had significant exposure to asbestos, had a higher rate of disease than members of other services.

Also, from the map provided in the paper, three states with extensive mining activities, Montana, Kentucky, and West Virginia, had among the highest rates of IPF reported. This raises the question of the possible role of pneumoconiosis-producing dusts as being a possible more correct reason for the fibrotic changes.

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Reply: Possible Alternate Explanation for Cases of Idiopathic Pulmonary Fibrosis

From the Authors:

We thank Dr. Frank for his thoughtful comments. Our study (1) excluded patients with codes for pneumoconiosis, so veterans with diagnoses such as asbestosis or coal workers’ pneumoconiosis would have been excluded from our idiopathic pulmonary fibrosis (IPF) cohort. Per Dr. Frank’s suggestion, we incorporated military service line into our multivariable regression model to examine whether service in the navy was associated with higher odds of IPF and found no significant difference between the odds of IPF among male navy veterans and the odds of IPF among male army veterans (odds ratio, 1.0; 95% confidence interval, 0.98–1.02; \(P = 0.58\)).

We agree that more work is needed to better understand the role that exposures play in the pathobiology and etiology of fibrotic lung diseases, and collecting such data has been proposed (2). As highlighted by Dr. Frank, exposures are of particular importance to the veteran population. In addition to environmental risk factors, military exposures may also increase the risk of fibrotic lung disease.

Our intention in this study was to use the strength of the Veterans Affairs learning healthcare system to identify a population-based cohort to better understand the epidemiology of IPF in this unique population. Quantifying disease burden is challenging, and