Amphibole asbestos as a public health risk in 2025: Autoimmune disease

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Despite efforts to minimize exposure, deaths from asbestos-related diseases remain high. A recent review emphasized the importance of evaluating different mineral groups and pointed out that exposure to amphibole asbestos is linked to higher rates of autoimmune diseases. Jean C. Pfau and Brenda J. Buck discuss the urgent need to address this public health risk more effectively

Despite decades of work to reduce asbestos exposure, tens of thousands of people die globally from asbestos-related diseases, including 30,000 deaths from mesothelioma. [1] Death rates from mesothelioma have remained fairly constant since 1999, both globally and in most countries, after having risen dramatically during the decades when asbestos exposures were not regulated. [2] In the United States, mesothelioma deaths were found to be disproportionately high. Because mesothelioma occurs specifically with exposure to asbestos and similar minerals, this constancy suggests that, despite current regulations, asbestos exposures continue to occur at a rate that poses a serious public health problem. In addition, the rates of asbestos-caused cancers of the trachea, bronchus, and lung in the United States are twice that of the global average during the same period. [2] Lastly, systemic autoimmune diseases (SAID) are on the rise globally, with evidence that much of that rise is due to environmental factors, including asbestos. [3,5]

One complicating factor is that asbestos is treated as one substance when, in fact, six regulated minerals come from two very different mineral groups: [1] Serpentine group (chrysotile) and [2] Amphibole group (tremolite, actinolite, riebeckite (crocidolite), anthophyllite, cummingtonite-grunerite (amosite)). The chrysotile and amphibole minerals have very different mineral structures and chemistries, which control their physical and chemical properties. Regulations are focused on chrysotile characteristics, thus resulting in many amphibole particles not being counted/ regulated as asbestos. For example, chrysotile is always fibrous, whereas amphibole may occur in both fibrous and non-fibrous forms. The latter are excluded in regulations but may still have adverse health effects. The definition of asbestos focuses on the characteristics of high tensile strength and flexibility, which are only two of several marketable characteristics of asbestos. These two are more common with chrysotile than amphibole minerals. Additionally, high tensile strength and flexibility cannot be measured in a microscope, nor have these characteristics been correlated to health outcomes. Regulations should be focused on the measurable characteristics that control toxicity. The confusion of considering asbestos as one entity also has resulted in academic and media publications stating that asbestos has been banned when, in fact, the recent ban enacted in the US only applies to chrysotile

and isn't an immediate or total ban. [4] These two mineral groups not only have distinct physical and chemical characteristics, but they also, not surprisingly, behave differently in the body. [5] Both research and regulations surrounding asbestos should treat these two mineral groups separately.

The risk of asbestos-induced systemic autoimmune disease

Last year, a small but diverse group of experts collaborated on a broad review of asbestos as a public health risk in the prestigious journal Autoimmunity Reviews. [5] The review focused on a non-cancer outcome, systemic autoimmune diseases (SAID), including Rheumatoid Arthritis, Systemic Lupus Erythematosus, and Systemic Sclerosis. The authors' first topic was identifying and mapping the source of exposure to amphibole asbestos. Several places where amphibole asbestos exposure occurs have reported elevated rates of positive tests for SAID. These sites include Libby, Montana (mining and use of asbestos-containing vermiculite), New York City (collapse of the World Trade Center towers), regions of Italy (mining of, and construction use of, fluoroedenite), and Australia (mining and commercial use of crocidolite). To illustrate the extent of possible exposure to fibrous minerals, the authors created maps of the United States that pinpoint sources of exposure within rocks and soils across large areas of the country. Many source areas are near large populations and recreation areas, such as Southern Nevada. As urban development exposes bedrock areas and climate change expand arid regions, producing dust from wind erosion, exposure increases. [6]

The 2024 review examined the published literature for articles studying asbestos and autoimmune disease and lists 48 epidemiologic studies in humans and 15 mechanistic studies in rodents. From 31 of the human studies, data regarding the development of antinuclear autoantibodies (ANA, diagnostic markers for SAID) enabled calculation of the risk of ANA from asbestos exposure in the form of odds ratios (OR) and 95% confidence intervals. The overall OR for all 31 studies was 2.78 (95% CI 2.39-3.23). Interestingly, the OR were much stronger, indicating higher risk, when the exposure contained amphibole compared to chrysotile asbestos. This difference between asbestos fiber types has previously been reported in animal studies. [7] The authors emphasized that most studies in rodents and humans support the hypothesis that exposure to amphibole asbestos impacts the immune system and drives autoimmune responses. Despite this strong evidence, a perusal of eight major papers from 2023-2024 on the health effects of asbestos from the US, Central Asia, Italy, Scandinavia, and South America and global reviews found no reference to the risk for autoimmune disease. We encourage policymakers, academics, healthcare workers, and governments to ask WHY.

The article in Autoimmunity Reviews also analyzes the mechanistic data for developing SAID, comparing crystalline silica (long recognized as a trigger for SAID) and asbestos. They found that all the mechanisms involved in driving silica-induced SAID are also activated by amphibole asbestos, not chrysotile. [5] These include oxidative stress, chronic inflammation and NALP3 inflammasome activation, dysregulated cell death pathways, impaired ability to safely dispose of dead cell debris, and activation of a non-resolving inflammatory response characteristic of autoimmune responses.

Currently, to our knowledge, there are only a few places in the world that are actively screening for SAID among people exposed to asbestos. In the interest of global public health, expanding SAID monitoring much more broadly through simple and inexpensive ANA testing could enhance the early detection of disease and, critically, the detection and remediation of current exposure sources.

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