ORAL ARGUMENT NOT YET SCHEDULED

No. 16-1105 and consolidated cases

UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT

NORTH AMERICA'S BUILDING TRADES UNIONS,
Petitioner,

v.

OCCUPATIONAL SAFETY & HEALTH ADMINISTRATION,
UNITED STATES DEPARTMENT OF LABOR,
Respondents.

On Petitions for Review of Final Rule of the
Occupational Safety & Health Administration

BRIEF OF THE AMERICAN THORACIC SOCIETY AND AMERICAN COLLEGE OF OCCUPATIONAL AND ENVIRONMENTAL MEDICINE AS AMICI CURIAE IN SUPPORT OF RESPONDENT AND SUPPORTING AFFIRMANCE OF AGENCY RULE

Lisa W. Jordan, D.C. Circuit Bar No. 59940
TULANE ENVIRONMENTAL
LAW CLINIC
6329 Freret St., Suite 130
New Orleans, LA 70118
(504) 865-5789
Counsel for the American Thoracic Society and the American College of Occupational and Environmental Medicine

DATED FEBRUARY 10, 2017

CERTIFICATE AS TO PARTIES, RULINGS, AND RELATED CASES

Pursuant to D.C. Circuit Rules 28(a)(1) and 29(d), Amici Curiae, in support of Respondent, certify as follows:

A. Parties, Intervenors, and Amici

All parties, intervenors, and amici appearing before this Court are listed in the Brief for Respondents (filed January 19, 2017).

Currently, there are no other amici; therefore, this brief complies with D.C. Circuit Rule 29(d).

B. Rulings Under Review

References to the rulings at issue appear in the Brief for Respondents.

C. Related Cases

The consolidated cases on review have not previously been before this Court or any other court. The undersigned counsel is not aware of any other related cases currently pending in this Court or any other court.

Dated: February 10, 2017

Respectfully submitted,

/s/ Lisa Jordan

Lisa W. Jordan
TULANE ENVIRONMENTAL LAW CLINIC
6329 Freret Street, Suite 130
New Orleans, LA 70118
(504) 865-5789

Counsel for the American Thoracic Society and the American College of Occupational and Environmental Medicine

CORPORATE DISCLOSURE STATEMENT

Pursuant to Rule 26.1 of the Federal Rules of Appellate Procedure and D.C. Circuit Rule 26.1, Amici Curiae state the following:

The American Thoracic Society and the American College of Occupational and Environmental Medicine are both incorporated organizations of medical and health professionals with an interest in avoiding death and disease from, inter alia, air pollution. Neither of these organizations has a parent company and no publicly-held entity owns an interest of more than ten percent in either of them.

Respectfully submitted 10th day of February, 2017,

/s/ Lisa Jordan
Lisa Jordan, D.C. Bar No. 59940
TULANE ENVIRONMENTAL
LAW CLINIC
6329 Freret St., Suite 130
New Orleans, LA 70118
(504) 865-5789

Counsel for the American Thoracic Society and the American College of Occupational and Environmental Medicine

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GLOSSARY

ACOEM	American College of Occupational and Environmental Medicine
ATS	American Thoracic Society
IARC	World Health Organization's International Agency for Research on Cancer
NIOSH	National Institute for Occupational Safety and Health
OEM	Occupational and Environmental Medicine
OSHA	Occupational Safety & Health Administration
PEL	Permissible exposure limit
$\mu g/m^3$	Micrograms per cubic meter of air

STATUTES AND REGULATIONS

Except for the following, all applicable statutory and regulatory provisions are contained in the Addenda to the Joint Opening Brief of Industry Petitioners and the Joint Brief of Union Petitioners:

Occupational Safety and Health Act § 3(8), 29 U.S.C. § 652(8): "For the purposes of this chapter . . . (8) The term 'occupational safety and health standard' means a standard which requires conditions, or the adoption or use of one or more practices, means, methods, operations, or processes, reasonably necessary or appropriate to provide safe or healthful employment and places of employment."

SUMMARY OF ARGUMENT AND STATEMENT AS TO IDENTITY OF AMICI CURIAE, THEIR INTERESTS IN THE CASE, AND THE SOURCE OF THEIR AUTHORITY TO FILE

Amici, the American Thoracic Society and the American College of Occupational and Environmental Medicine, support OSHA's findings that reduction from the previous permissible exposure limit (PEL) of 100 µg/m^3 to a more protective PEL of 50 µg/m^3, as well as the establishment of an action level of 25 µg/m^3 for medical surveillance, will significantly reduce the material impairment of health caused by exposure to respirable crystalline silica for workers in industries regulated by the rule. OSHA's rule is based on the best available scientific evidence and a thorough review of the record.[1]

The American Thoracic Society (ATS) is an international educational and scientific organization founded in 1905 that represents more than 16,000 physicians, scientists, nurses, respiratory therapists, and allied health care professionals. These members are leaders in the prevention, detection, treatment, cure, and research of respiratory diseases, including respiratory disease caused by occupational exposure to silica. The ATS works to prevent and fight respiratory disease around the globe through research, education, patient care, and advocacy.

[1] Throughout this brief, documents in the rulemaking record are referred to as "OSHA-2010-0034-XXXX," where XXXX reflects the last four digits of the full document number.

Our membership includes experts on respiratory occupational health. The ATS publishes three peer-reviewed scientific journals which disseminate groundbreaking research, including studies on respiratory occupational exposures and pulmonary occupational disease. The ATS has published treatment guidelines for the diagnosis and management of patients with occupational silicosis.

Established in 1916, the American College of Occupational and Environmental Medicine (ACOEM) is an international society of 4,500 occupational and environmental medicine (OEM) physicians. The OEM physician has the knowledge and skills to provide evidence-based clinical evaluation and treatment of injuries and illnesses that are occupationally and/or environmentally related. In addition, the OEM physician's skill and expertise includes understanding health risks, clinical practice guidelines for chronic disease management, and current practices in disease detection, prevention, and treatment. Members of ACOEM have the ability to assess the causes and occupational impact of respiratory disorders and pulmonary impairment.

On September 26, 2016, this Court granted the ATS and ACOEM's motion to participate as amici curiae in support of Respondents. ECF No. 1637793. ATS's and ACOEM's identities and interests in the case are also outlined in that motion.[2]

ARGUMENT

I. WORKERS IN INDUSTRIES REGULATED BY OSHA'S FINAL RULE ARE REGULARLY EXPOSED TO RESPIRABLE CRYSTALLINE SILICA AND SUFFER MATERIAL IMPAIRMENT OF HEALTH AND FUNCTIONAL CAPACITY AS A RESULT.

Silica, when inhaled as small particles, causes silicosis, lung cancer, certain kinds of autoimmune diseases including ones that damage the kidneys, susceptibility to deep lung infections, and a condition closely related to emphysema. Silicosis is an umbrella term for various related forms of lung scarring and is the most common occupational lung disease in the world. As health care professionals who regularly see patients with health impairments caused by respirable crystalline silica, ATS and ACOEM members can describe how silicosis and silica-related diseases develop from exposure to silica in the workplace.

A. The Method By Which Worker Inhalation of Respirable Crystalline Silica Causes Significant Adverse Health Effects is Well Known.

[2] No party's counsel authored this brief in whole or in part, and no person, other than Amici, their members, or their counsel, made a monetary contribution to the preparation or submission of this brief.

Silica is a naturally-occurring material, familiar as quartz or as a component of granite and many other rocks, sand, clays, and a variety of building materials. Large silica particles found in nature from coarse rock dust, soil, and sand are not respiratory health hazards because they fall out of the air quickly. Even when inhaled, most particles of this size never get into the lung itself because they are very large relative to the scale of the respiratory tract and are effectively removed without being absorbed. Silica becomes a hazard when it is in the form of a dust small enough to be inhaled. This commonly occurs in the workplace when rock or silica-containing material is cut, sawed, drilled, blasted, ground, or polished, or when silica is used as a powder or in dust from material containing silica (such as clays used for ceramics like porcelain). Such silica dust consists of very small particles that form clouds in air and are carried by air currents. Workers in industries, such as mining and construction, which involve handling silica-containing material often inhale silica dust because the dust cloud surrounds them and the source is close to their nose and mouth.

The hazard of silica dust is greatest for the smallest particles, because they penetrate most deeply into the lung and have more surface area relative to weight. They are even more hazardous when the silica is freshly cut (the term of art is “freshly-fractured” silica) because the cut surface exposes electrically-charged, chemically reactive sites on the surface of the particle. While all respirable silica

particles can cause silicosis, freshly-fractured particles are even more dangerous than weathered particles, because weathered particles have undergone a reaction with exposure to air that partially reduces their chemical reactivity. This is important because common industrial processes such as drilling and cutting stone and sandblasting create freshly-fractured silica in abundance. The greatest health hazard is from extremely small, freshly-fractured silica particles.

When a person inhales a silica particle, the particle is carried in air through the nose and mouth and throat. The very largest particles lodge there and may produce local irritation. Medium-sized particles enter the trachea (the first and largest airway of the lung) and are conducted into the branching tree of airways that leads to the deep lung. Some of them fall out in the airway and others stick to the sides, but many remain airborne. Most of these medium-sized particles cause local irritation and cough and can contribute to obstructive airways disease, even in non-smokers.

However, the greater problem is with the smallest particles, which penetrate deeply into the tissue of the lung itself. These particles set up a site of inflammation that is unusually severe and chronic and that results in pockets of scarring, called “nodules.” This inflammation and scarring is the beginning of the disease process of silicosis. These nodules are initially quite small and can only be detected on biopsied lung samples. However, they grow together over time and

eventually cause wider bands of scar tissue in the lung that become visible on chest X-ray. These nodules may progress to cause the entire lung or large parts of it to become rigid, pocked by holes, resistant to blood flow in such a way as to cause a strain on the heart, and, most importantly, ineffective in oxygen exchange. Sometimes this scar tissue forms large masses in the lung that pull at and essentially tear the fabric of normal tissue. Individuals vary in how susceptible they are to these effects, but everyone will develop scarring if enough silica is inhaled.

One of the reasons that silica particles produce these effects in the lungs is that they poison the guardian immune cells of the lung, which are large cells (called macrophages) that pick up and engulf bacteria, debris, and other types of particles. Once the cell has picked up a silica particle, its important immune functions become less effective and when it eventually dies another macrophage will ingest the silica particle, causing the cycle to repeat. The cycle results in more and more scar tissue of silicosis and impedes the capacity of the macrophage to fight off certain types of infection like tuberculosis.

The damage done by the "sick" macrophage's ineffective immune response also leaves damaged and distorted cellular debris, which the body may not recognize as its own tissue. When that happens, the body mounts an immune response to itself (called an "autoimmune response") and this can lead to serious arthritis-like disease throughout the body (including joints and skin), additional

lung disorders on top of the scar tissue, and two types of autoimmune kidney disease. Individuals vary in how strong their autoimmune response is.

For all these reasons, the nature, extent, and severity of silica-induced lung disease can vary a great deal from person to person but still result in very characteristic disorders that appear whether or not the person also smoked. All forms of silicosis, without exception, are "chronic" because they are permanently present for the duration of the person's life and there is no recovery and no specific treatment.[3]

B. The Diseases Caused by Silica Exposure and Inhalation Are Well Recognized.

Worker exposure to respirable crystalline silica causes a number of diseases, including various forms of silicosis, silicotuberculosis, various forms of autoimmune disease, kidney disease, airways disease, and lung cancer. Individually and collectively, these diseases take a significant toll on the health of exposed workers.

[3] It should be noted that the terminology in many sources is somewhat confused and requires clarification. When "silicosis" is used without qualification, it usually means what is referred to below as either "simple silicosis" or "conglomerative silicosis." Many writers, even experts, use the term "chronic silicosis" to refer to "conglomerative silicosis." These variations in nomenclature should be considered terms of art, not inconsistencies.

- Simple silicosis describes the disease of silica-induced scar tissue in the lung with no other complicating factors; the nodules are relatively small and widely scattered. Some patients with simple silicosis have no chest symptoms, but they may have mild breathing test abnormalities. Simple silicosis can progress over time, even after exposure ends, and places a person at risk of other silica-related conditions, infection, and lung cancer. With progression, simple silicosis can cause shortness of breath and can complicate other lung diseases.
- Conglomerative silicosis describes a more advanced form of silicosis which always begins as simple silicosis. Over time, the nodules grow together into larger nodules and masses and extensive scar tissue. Large tumor-like masses of scar tissue may displace normal lung. This kind of silicosis causes shortness of breath (sometimes called “air hunger” and often severe and panic-inducing), disabling exercise limitation, difficulty maintaining enough oxygen in the blood, and strain on the heart trying to pump blood through the scarred lung masses. It is often fatal because eventually the lungs cannot supply the body with adequate oxygen. Death in this way is particularly excruciating because of the strong and constant feeling of suffocation. Assisted breathing using a ventilator does not help or extend life. When exposure levels are high and these effects occur in years instead of decades,

the disease may be considered "accelerated." Once it reaches the conglomerative stage silicosis progresses on its own without further exposure and there is nothing the patient can do to prevent its progression; it is unrelated to smoking.

- Acute silicosis (sometimes called silicoproteinosis) is a disorder in which the lung is essentially in shock from the response to rapid deposition of large amounts of dust in a short time. The air sacs in the lung flood with thick fluid that cannot be removed by the large cells that normally clean the lungs of particles and other debris. Instead of forming scars, the patient drowns in this air sac fluid, and the condition is usually rapidly fatal within weeks or months of high exposure, such has historically occurred in sandblasting and in workers making abrasive soaps with finely divided (small particle size) silica flour.
- Silicotuberculosis occurs when a worker infected with tuberculosis or a similar type of bacteria is exposed to silica. Most persons who are exposed to tuberculosis bacteria wall off the infection with large immune cells that prevent tuberculosis from developing. This condition, called "latent tuberculosis infection," is not contagious and does not cause lung damage or spread to other organs. However, silica poisons these immune cells so that they are unable to perform this function and, therefore, tuberculosis becomes

an active infection. With this compromise of normal immune defense, treatment with antibiotics is very difficult. In addition, antibiotics do not efficiently penetrate the dense bundles of scar tissue in the nodules of silicotic patients. The patients with silicotuberculosis commonly die of the silicosis part of the disease while also suffering the effects of advanced tuberculosis, which may include weight loss (wasting away), coughing up blood, shortness of breath, and lung failure to supply oxygen to the body. In addition, their uncontrolled tuberculosis puts family and health care workers at risk of developing tuberculosis.

- Silica-related autoimmune disorders are of several types, including systemic sclerosis (also known as scleroderma), which can come with its own complication of kidney disease, rheumatoid arthritis, Caplan's syndrome (which causes masses in the lung of a different type), a different type of kidney disease (usually less severe), and other manifestations we are still learning about. These cases may develop symptoms of arthritis ranging from minor to severe and get worse with time.
- Airways disease is a general term for problems that affect the tubes that conduct air into the lungs, such as a form of emphysema, an irritation-induced form of chronic bronchitis, or chronic obstructive airways disease (not to be confused with the disease related to smoking, although it can

make it and other pre-existing airways diseases worse). Silica dust by itself can produce an airways disorder through irritation and inflammation in lung tissue. The airways disease caused by silica dust causes people to cough and feel short of breath.

- Silica exposure causes lung cancer, even in the absence of silicosis or cigarette smoking. Silica also increases the risk of lung cancer associated with cigarette smoking.

In sum, disease outcomes from silica dust exposure are diverse, serious, sometimes fatal, and always preventable.

C. Case Studies of Preventable Silicosis and Other Respiratory Diseases Reflect the Diversity and Devastation of These Diseases.

ATS and ACOEM members regularly care for patients with silica-related diseases. The following patient case reports highlight the impact of these diseases.

The first example is that of a fifty-two-year-old silicosis patient. After ten years of working at a sheet metal plant, this never-smoker developed shortness of breath when he exerted himself and joint pain all over his body. Since 2003, he worked in a poorly ventilated room sandblasting metal parts for nine hours a day and swept the dusty room at the end of each work day. He had worn increasingly protective respirators during his employment: a disposable respirator in his first two years, then a half-face cartridge respirator, and finally an air-supplied respirator. After a year of progressive shortness of breath and cough, in 2014 he

consulted a physician. His lung test for ability to transfer oxygen to his blood was severely abnormal at 48% of predicted. At surgery, he had acute silicosis on a background of chronic silicosis. His doctor removed him from his job and treated him with oxygen, an anti-inflammatory drug, and a lung washing, which did not improve his health. While he was waiting for an expedited lung transplant, he developed an infection related to tuberculosis-like bacteria to which silicosis patients have poor resistance. Despite intensive medical treatment, his lung disease progressed rapidly and he died when his lungs failed in 2015.

The second example is that of a 30-year-old mason. This man worked repairing exterior brickwork on two public apartment buildings by cutting out mortar between bricks, a process known as "tuck-pointing," over several months with a powered hand grinder. Two months after completing this job he became progressively short of breath. In 2005, he underwent lung biopsy and whole lung wash under general anesthesia, but subsequently went into respiratory failure requiring mechanical ventilation for several days. He was referred for lung transplant, but developed a collapsed lung requiring a chest tube that prevented the lung transplant surgery. Analysis of minerals in lung biopsy tissue showed silica and other mineral particles. Although he improved somewhat following repeated aggressive treatment to wash the deep lung with saline solution, his oxygen levels remained low and he still requires oxygen for any exertion.

The final example was recorded by the Texas Department of State Health Services.[4] A twenty-seven-year-old Hispanic man who had never smoked started working in an industry making manmade stone countertops that contained 70-90 percent silica. Eight years into this employment as a polisher, laminator, and fabricator, he developed shortness of breath on exertion and persistent cough. Two years later in 2010, he first sought medical care for these symptoms. His physical examination, breathing tests, and test for oxygen transfer to the blood were all abnormal. His chest X-ray showed that he had large spots on his lung consistent with progressive massive fibrosis (conglomerative silicosis), with small silicotic nodules in the top two-thirds of his lungs. Tests showed that his lung disease had strained the right side of his heart and had elevated blood pressure in the blood vessels carrying blood to his lungs. He is on oxygen and being followed for lung transplantation at the age of 37.

II. THE BEST AVAILABLE SCIENTIFIC EVIDENCE SUPPORTS OSHA'S FINDING THAT THERE IS A SIGNIFICANT RISK OF MATERIAL IMPAIRMENT OF HEALTH AT THE PREVIOUS PERMISSIBLE EXPOSURE LIMITS AND THAT THE MORE PROTECTIVE PERMISSIBLE EXPOSURE LIMIT WILL SUBSTANTIALLY REDUCE THE RISK OF THE ADVERSE HEALTH EFFECTS CAUSED BY EXPOSURE TO RESPIRABLE CRYSTALLINE SILICA.

[4] Gary K. Friedman et al., *Silicosis in a Countertop Fabricator – Texas, 2014*, 64 Morbidity and Mortality Weekly Report 118, 129 (2015).

Congress mandated that, in setting and modifying occupational safety and health standards for toxic materials or harmful physical agents, OSHA "shall set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity . . . ." 29 U.S.C. § 655(b)(5), Occupational Safety and Health Act ("Act") § 6(b)(5). The U.S. Supreme Court emphasized the Act's focus on worker health and safety: "When Congress passed the Occupational Safety and Health Act in 1970, it chose to place pre-eminent value on assuring employees a safe and healthful working environment, limited only by the feasibility of achieving such an environment." *Am. Textile Mfrs. Inst., Inc.* v. *Donovan*, 452 U.S. 490, 540 (1981).

Congress defined occupational safety and health standards as those "reasonably necessary or appropriate to provide safe or healthful employment and places of employment." Occupational Safety and Health Act § 3(8), 29 U.S.C. § 652(8). Pursuant to this definition, in promulgation of a standard, OSHA must find that "significant risks are present and can be eliminated or lessened by a change in practices." *Indus. Union Dep't, AFL–CIO v. Am. Petroleum Inst.*, 448 U.S. 607, 642 (1980) (plurality opinion).

The best available evidence supports OSHA's finding that there is a significant risk of material impairment of health at the previous Permissible

Exposure Limit of 100 micrograms per cubic meter (µg/m^3) for general industry and 250 µg/m^3 for construction and maritime industries. The best available evidence also supports OSHA's conclusion that a reduction of the Permissible Exposure Limit to 50 µg/m^3 will significantly decrease that risk. OSHA selected the best-designed scientific studies to prepare its Preliminary Quantitative Risk Assessment. Further, the agency's response to public comment in preparation of the Final Rule was thorough and reasonable.

A. The Best Available Scientific Evidences Proves a Significant Risk of Carcinogenic Effects from Exposure to Respirable Crystalline Silica at the Previous Permissible Exposure Limits and That the Reduction of the Limits to 50 µg/m^3 Will Substantially Reduce the Risk.

The evidence that silica is a carcinogen is compelling, not uncertain. Many august international and national scientific and policy organizations have concluded that silica causes human cancer, including the World Health Organization's International Agency for Research on Cancer (IARC) in both 1997[5] and 2012,[6] California's Proposition 65 in 1988,[7] the U.S. National Toxicology

[5] IARC, *Silica, Some Silicates, Coal Dust and Para-Aramid Fibrils*, 68 IARC Monographs on Carcinogenic Risk to Humans 41 (1997) (OSHA-2010-0034-1301).

[6] IARC, *Arsenic, Metals, Fibres, and Dusts*, 100C IARC Monographs on the Evaluation of Carcinogenic Risk to Humans 355 (2012) (OSHA-2010-0034-1473).

[7] Safe Drinking Water and Toxic Enforcement Act of 1986, Cal. Health and Safety Code § 25249.5 et seq. (West, Westlaw through Ch. 8 of the 2015-2016 Second Extraordinary Session, and all propositions on 2016 ballot).

Program in 2011;[8] and the U.S. National Institute for Occupational Safety and Health (NIOSH) in 2002.[9]

Additionally, studies by many different investigators across the world have demonstrated increased lung cancer risk in diverse industries with silica exposure. A 2001 study by Kyle Steenland and others aggregated and updated ten studies of workers in different industries to produce stable and robust calculations of the degree of lung cancer risk in relation to levels of exposure to silica over working lifetimes.[10] In this study, Steenland and his co-authors divided the 65,980 workers from these ten studies – who were from many different countries – into five equally-sized subgroups with increasing cumulative silica exposure. The subgroups had progressively higher lung cancer death rates compared to the lowest exposure subgroup.[11] The authors concluded that workers in the United States exposed to 100 µg/m^3 of respirable crystalline silica from ages 20-65 would have 1.7% excess lung cancer deaths through age 75, causing material impairment to health for 17 of

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[8] Nat'l Toxicology Program, Pub. Health Serv., U.S. Dep't of Health and Human Servs., Report on Carcinogens 377, 12th ed. (2011) (referenced in OSHA-2010-0034-1417).

[9] NIOSH, Ctrs. for Disease Control and Prevention, U.S. Dep't of Health and Human Servs., Health Effects of Occupational Exposure to Respirable Crystalline Silica (2002) (OSHA-2010-0034-1110).

[10] Kyle Steenland et al., *Pooled Exposure-Response Analyses and Risk Assessment for Lung Cancer in 10 Cohorts of Silica-Exposed Workers: An IARC Multicentre Study*, 12 Cancer Causes and Control 773 (2001) (OSHA-2010-0034-0452).

[11] *Id.* at 778-779.

1000 exposed workers from lung cancer alone.[12] This excess risk alone justifies lowering the permissible exposure limit.

After OSHA appropriately prepared its quantitative risk assessment, a more recent study – included in the record and available during the public comment period for refutation – confirmed the accuracy of OSHA's risk assessment finding that silica causes lung cancer. This excellent study by Liu and others analyzed data for the period 1960-2003 among 34,018 tungsten and iron miners and ceramic workers who had not been exposed to other occupational causes of lung cancer.[13] The researchers estimated cumulative silica exposure for each individual in the study by linking average silica exposure by job with work history and summing exposure across all jobs held.[14] The average length of follow-up for the 34,018 workers was 34.5 years.[15] Using similar methods to the Steenland pooled analysis above, the study then divided the workers into four equal groups with increasing cumulative silica exposure.[16] As average silica exposure over employment

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[12] *Id.* at 780.

[13] Yuewei Liu et al., *Exposure-Response Analysis and Risk Assessment for Lung Cancer in Relationship to Silica Exposure: A 44-Year Cohort Study of 34,018 Workers*, 178(9) Am. J. Epidemiology 1424 (2013) (article included in OSHA-2010-0034-2340).

[14] *Id.* at 1425.

[15] *Id.*

[16] *Id.* at 1426.

increased in the four groups, the risk of lung cancer increased, compared to the unexposed group.[17]

Both the historical studies used in OSHA's risk assessment and the more recent Liu study demonstrated exposure-response relationships between the cumulative amount of silica exposure over time and the risk of lung cancer. This demonstrated dose-response relationship supports OSHA's finding that lowering the permissible exposure to 50 µg/m^3 of respirable silica over a working lifetime will result in fewer lung cancer cases. According to Liu et al, for every 1000 workers exposed to the prior OSHA PEL for a working lifetime (45 years), five will die from lung cancer.[18] At lower exposure levels, the risk is reduced, falling to one excess death for 1000 workers at a working lifetime exposure of 20 µg/m^3.[19]

The 2013 Liu study also refuted the arguments that silicosis is a required precursor to a finding of lung cancer, and that any lung cancer finding is the result of fibrotic processes and not a function of the silica dust. The study was large enough that analyses of silica-exposed workers without silicosis could be performed and showed that lung cancer risk increased by cumulative silica exposure group, even when workers did not have scarring due to silicosis.[20] This

[17] *Id.* at 1426-1428.
[18] *Id.* at 1426.
[19] *Id.*
[20] *Id.* at 1429, 1431.

finding that more exposure correlates with increased lung cancer risk among those without silicosis scarring confirms that silica is a carcinogen in itself and not just because it causes scarring that can also be associated with lung cancer.

The Liu study was unusually strong because smoking histories were available for the participants, and there were over 12,000 never-smoking workers.[21] The authors contrasted high and low silica-exposed non-smokers. Those with high exposure had 1.6 times the risk for lung cancer compared to the low exposed group.[22] Furthermore, cigarette smokers with silica exposure had much higher risk of lung cancer than was accounted for by smoking alone or silica exposure alone.[23] In fact, the risk of lung cancer in smoking silica-exposed workers was far greater than adding the risks conferred by either smoking alone or silica exposure alone, an effect that we call near-multiplicative risk.[24] This much higher risk of lung cancer in smoking silica-exposed workers is an additional justification for regular medical surveillance of silica-exposed workers, as an opportunity for a smoking cessation program to greatly decrease lung cancer risk.

B. Workers Exposed to Respirable Crystalline Silica at the Previous Permissible Exposure Limits Have a Significant Risk of Silicosis.

[21] *Id.* at 1431.
[22] *Id.*
[23] *Id.*
[24] *Id.*

In addition to the compelling evidence that silica causes lung cancer, there is overwhelming evidence that workers exposed to respirable crystalline silica at levels below the previous permissible exposure limit of 100 µg/m^3 are still at significant risk of developing silicosis (morbidity) and dying from silicosis (mortality). A solid body of well-conducted studies, relied on by OSHA, strongly supports this conclusion. Additionally, while silicosis is underreported and underestimated, it is not disappearing. In fact, emerging industries and technologies expose additional workers to respirable crystalline silica and the resulting significant health risks.

1. The Best Available Scientific Evidence Proves a Significant Risk of Silicosis at the Previous Permissible Exposure Limits and That the Reduction of the Limits to 50 µg/m^3 Will Substantially Reduce the Risk.

The strongest studies quantitating the risk of *death* from silicosis at different levels of silica exposure were published in the premier British journal in our field, Occupational and Environmental Medicine, a journal that has particularly rigorous peer review of potential articles for publication.[25]

[25] A 't Mannetje et al., *Exposure-Response Analysis and Risk Assessment for Silica and Silicosis Mortality in a Pooled Analysis of Six Cohorts*, 59 Occup. Environ. Med. 723 (2002) (OSHA-2010-0034-1089); R. Park, et al., *Exposure to Crystalline Silica, Silicosis, and Lung Disease Other than Cancer in Diatomaceous Earth Industry Workers: A Quantitative Risk Assessment*, 59 Occup. Environ. Med. 36 (2002) (OSHA-2010-0034-0405).

Like the studies analyzing the risks of dying from silicosis, studies which carefully evaluated the risk of *developing* silicosis have shown a significant risk associated with exposures at the levels allowable under the previous permissible exposure limit and even the contested lower permissible exposure limit of 50 $\mu g/m^3$. These robust studies describe significant risks of developing silicosis in American gold miners; Scottish coalminers; Chinese tin miners; Chinese tin, tungsten, and pottery workers; and South African gold miners.[26] The general agreement about the degree of silicosis risk in relation to amount of silica exposure of so many researcher groups in diverse populations and countries leaves no doubt about the strength of evidence supporting OSHA's regulation.

In all the studies discussed and cited above, which OSHA relied on to assess the risks of developing silicosis and dying from silicosis, air levels of silica dust in workplaces were measured or estimated with the best historical information

[26] Kyle Steenland & David Brown, *Silicosis Among Gold Miners: Exposure-Response Analyses and Risk Assessment*, 85 Am. J. Public Health, 1372 (1995) (OSHA-2010-0034-0451); D. Buchanan et al., *Quantitative Relations Between Exposure to Respirable Quartz and Risk of Silicosis*, 60 Occup. Environ. Med. 159 (2003) (OSHA-2010-0034-0306); W. Chen et al., *Exposure to Silica and Silicosis Among Tin Miners in China: Exposure-Response Analyses and Risk Assessment*, 58 Occup. Environ. Med. 31 (2001) (OSHA-2010-0034-0332); W. Chen et al., *Risk of Silicosis in Cohorts of Chinese Tin and Tungsten Miners, and Pottery Workers (I): An Epidemiological Study*, 48 Am. J. Ind. Med. 1 (2005) (OSHA-2010-0034-0985); Eva Hnizdo & G.K. Sluis-Cremer, *Risk of Silicosis in a Cohort of White South African Gold Miners*, 24 Am. J. Ind. Med., 447–457 (1993) (OSHA-2010-0034-1052).

available about process changes, control measures, and the correspondence of new with old methods of measuring dust and silica levels. Uncertainties always exist in trying to reconstruct past exposures, but we are persuaded of the conclusions about job-specific exposures made by experienced public health researchers such as those at national public health organizations in several countries, including Drs. Park, Steenland, Hnizdo, Soutar, Chen, and others from the cited references. These researchers followed workers exposed to historical silica air levels for many years, including after employment. Their studies showed that a working life of exposure at levels *even below the current OSHA permissible exposure limit of 50 $\mu g/m^3$* resulted in silicosis lung disease and silicosis deaths. These studies, among others relied on by OSHA, demonstrate the need to lower the OSHA permissible exposure limit, even though a small risk of silicosis will remain at the lowered limit.

When increasing level of exposure correlates with increasing risk of disease (called an exposure-response relationship), as these studies demonstrate, lowering of exposure prevents cases of occupational lung disease and death that would occur at higher exposure levels. OSHA appropriately conducted its risk assessment using the most robust studies of silica exposure in relation to risk of developing silicosis and dying from silicosis. In science, no single study is definitive by itself, but the lines of convergent evidence from many studies of workers in diverse industries

and countries by many researchers constitute sufficient evidence that requires lowering of allowable silica exposures to prevent unnecessary illness and premature deaths.

2. Silicosis Is Still Prevalent and Is Underreported and Underestimated.

Our physician members who care for silica-exposed workers in our clinics and hospitals are well aware through their clinical practices that silicosis has not disappeared with the previous exposure limit. In fact, it is underreported and underrecognized, for several reasons. First, reliance on public health counts of silicosis cases to calculate their prevalence is an error, as they are woeful underestimates.[27] On the basis of underreporting of cases in Michigan, one public health researcher estimated that 3,600 to 7360 new cases of silicosis occur annually in the United States.[28]

As specialists in occupational and respiratory diseases, we find that many patients are referred to us by primary care physicians or other respiratory specialists who have not considered the diagnosis of silicosis because it usually occurs decades after exposure and requires obtaining and interpreting patients' occupational histories to ascertain likely silica exposure. Compounding this

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[27] Kenneth D. Rosenman et al., *Estimating the Total Number of Newly-Recognized Silicosis Cases in the United States*, 44(2) Am. J. Indus. Med. 141, at 141 (2003) (OSHA-2010-0034-0420).

[28] *Id.* at 145.

diagnosis issue is the fact that silicosis is often misdiagnosed as other diseases (e.g., sarcoidosis) because the disease is unfamiliar to most clinicians and because medical records usually do not contain an informative occupational history.

The second reason that silicosis is underreported and underestimated is because hospital discharge data and death certificates poorly reflect the prevalence of disease.[29] This is because most silicotic patients with simple silicosis are not admitted to hospital, and the patients with conglomerative silicosis are frequently misdiagnosed. Similarly, death certificates mentioning silicosis underestimate the burden of disease.[30] We concur that OSHA wisely did not rely on surveillance of silicosis death certificate data in estimating the burden of silica-related disease and need for lowered permissible exposure.

Further, new industries have arisen that confer risk of silicosis, such as installing artificial quartz countertops and hydraulic fracturing for production of oil and gas.

Finally, studies of *current* workers conducted by industry underestimate health effects and prevalence of silicosis because silica dust stays in the lungs and does damage decades after first exposure. Numerous studies show that the disease of silicosis is often not apparent during employment and arises and gets worse after

[29] *Id.*
[30] *Id.*

dust exposure ends when employment ceases.[31] Studies limited to current workers thus underestimate the disease burden that will eventually occur.

C. The Best Available Scientific Evidence Proves a Significant Risk of Other Diseases, Nonmalignant Respiratory Disease, and Lung Function Impairment at the Previous Permissible Exposure Limits and That Reduction to 50 µg/m^3 Will Substantially Reduce the Risk.

1. Autoimmune and Renal Effects

Silica is like some other toxins, such as cigarette smoke, that cause many diseases outside of the lung. Crystalline silica is associated with increased risk of autoimmune diseases including rheumatoid arthritis, systemic sclerosis, systemic lupus erythematosus, and autoimmune blood vessel inflammation.[32] Further, population-based studies have shown a positive relationship between occupational silica exposure and chronic kidney disease deaths.[33] Some of the kidney disease is likely caused by silica's autoimmune consequences, even in the absence of

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[31] P. A. Hessel et al., *Progression of Silicosis in Relation to Silica Dust Exposure*, 32 Annals of Occupational Hygiene 689 (1988) (OSHA-2010-0034-1042); Brian G. Miller et al., *Risks of Silicosis in Coalworkers Exposed to Unusual Concentrations of Respirable Quartz*, 55 Occup. Environ. Med. 52 (1998) (OSHA-2010-0034-0374); Haibing Yang et al., *Natural Course of Silicosis in Dust-Exposed Workers*, 26(2) J. Huazhong Univ. of Science and Technology [Med Sci], 257 (2006) (OSHA-2010-0034-1134); Suteo Ogawa et al., *A 40-year Follow-up of Whetstone Cutters on Silicosis*, 41 Indus. Health 69 (2003) (OSHA-2010-0034-0398).

[32] C. Parks et al., *Occupational Exposure to Crystalline Silica and Autoimmune Disease*, 107 Envtl. Health Persp. 793 (1999) (OSHA-2010-0034-0406).

[33] Kyle Steenland et al., *Pooled Analyses of Renal Disease Mortality and Crystalline Silica Exposure in Three Cohorts*, 46 Annals of Occupational Hygiene 4 (2002) (OSHA-2010-0034-0448).

silicosis. Comparison of deaths from silica-associated kidney disease with deaths from lung cancer and deaths from silicosis shows that these three diseases are about equally common in silica-exposed workers at the previous permissible exposure limit of 100 µg/m^3, each in excess of the usual OSHA acceptable excess risk of serious disease or death for workers of 0.1%.[34]

2. Chronic Obstructive Airway Diseases

Chronic bronchitis and emphysema are obstructive lung diseases that occur commonly among smokers. However, silica exposure also causes these diseases.[35] In silica-exposed smokers, the extent of breathing test impairment exceeds that attributable to cigarette smoking alone.[36] A strong dose-response relationship exists in several studies between cumulative silica exposure and breathing test

[34] Kyle Steenland, *One agent, many diseases: Exposure-response data and comparative risks of different outcomes following silica exposure*, 48(1) Am. J. Indus. Med. 16, 21-22 (2005) (OSHA-2010-0034-1123).

[35] R. Park et al., *Exposure to Crystalline Silica, Silicosis, and Lung Disease Other than Cancer in Diatomaceous Earth Industry Workers: A Quantitative Risk Assessment*, 59 Occupational & Envtl. Med. 36, at 36 (2002) (OSHA-2010-0034-0405).

[36] Xiaorong Wang et al., *Respiratory Impairments Due to Dust Exposure: A Comparative Study Among Workers Exposed to Silica, Asbestos, and Coalmine Dust*, 31 Am. J. Indust. Med. 495, 498-499 (1997) (OSHA-2010-0034-0478)

measurements of lung volume, ease of exhaling, and exchanging oxygen.[37] The damage to lung function caused by silica exposure occurs even in persons without the scarring of silicosis and indicates that silica exposure has a direct effect on the airways.[38] Silica exposure and smoking in combination have particularly deleterious effects, as has been shown for the lung cancer risk of silica exposure.[39] Mortality from lung disease other than cancer and infection, most of which is from the chronic obstructive lung diseases, is also related to the amount of exposure to silica dust.[40]

3. Active Tuberculosis From Latent Infection.

Silica dust impairs the body's defenses against tuberculosis and infections from similar bacteria. Most persons who are exposed to tuberculosis bacteria wall off the infection with immune cells so that the bacteria do not continue to multiply and spread in the body, causing active clinical tuberculosis. If the immune system fails later in life, this "latent" tuberculosis can then become contagious active

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[37] E. Hnizdo & V. Vallyathan, *Chronic Obstructive Pulmonary Disease Due to Occupational Exposure to Silica Dust: A Review of Epidemiological and Pathological Evidence*, 60 Occup. Environ. Med. 237 (2003) (OSHA-2010-0034-0405-1055); K. Kreiss et al., *Hard-rock Mining Exposure Affects Smokers and Non-Smokers Differently*, 143 Am. Rev. Respir. Dis. 1487 (1991) (OSHA-2010-0034-0405-1079).

[38] Wang, *supra* note 36, at 500-501.

[39] Liu, *supra* note 13, at 239; Hnizdo, *supra* note 37, at 58.

[40] Park, *supra* note 35, at 38-39.

tuberculosis. When persons with active tuberculosis talk, sing, or cough, tuberculosis bacteria become airborne and can infect those around them, including health care workers caring for them.

In silica-exposed workers, the immune system may be ineffective in containing tuberculosis bacteria. The consequences are increased rates of active tuberculosis, difficulty in treating tuberculosis, increased death rates from tuberculosis, and spread of tuberculosis to workers' families and communities.[41]

III. THE BEST AVAILABLE SCIENTIFIC EVIDENCE SUPPORTS OSHA'S ESTABLISHMENT OF AN ACTION LEVEL OF 25 µg/m^3 TRIGGERING MEDICAL SURVEILLANCE THAT WILL SUBSTANTIALLY REDUCE THE RISK OF ADVERSE HEALTH EFFECTS CAUSED BY EXPOSURE TO RESPIRABLE CRYSTALLINE SILICA.

The ATS and ACOEM strongly support OSHA's rule calling for medical surveillance triggered by exposure assessments showing respirable silica levels exceeding the new action level of 25 µg/m^3. The action level makes sense as even the new permissible exposure level does not prevent all silica-related diseases, and cases can be identified that should trigger attempts to lower silica exposures at the workplace and for the individual, and treat diseases like silicotuberculosis, lung

[41] Eva Hnizdo & Jill Murray, *Risk of Pulmonary Tuberculosis Relative to Silicosis and Exposure to Silica Dust in South African Gold Miners*, 55 Occupational and Envtl. Med. 496 (1998) (OSHA- 2010-0034-0360).

cancer, autoimmune diseases, and chronic obstructive lung diseases. Maintaining medical confidentiality for individual surveillance medical records is a long-honored ethical principle of occupational health surveillance and does not preclude a health professional sharing preventive counsel to the employer based on aggregate results of a silica-exposed workforce. The medical surveillance components proposed by OSHA are sound, reasonable and well-justified.

CONCLUSION

For all the foregoing reasons, OSHA's Final Rule on Occupational Exposure to Respirable Crystalline Silica should be UPHELD.

Respectfully submitted 10th day of February, 2017,

/s/ Lisa Jordan
Lisa Jordan, D.C. Bar No. 59940
TULANE ENVIRONMENTAL
LAW CLINIC
6329 Freret St., Suite 130
New Orleans, LA 70118
(504) 865-5789
Email: lwjordan@tulane.edu

Counsel for the American Thoracic Society and the American College of Occupational and Environmental Medicine

CERTIFICATE OF COMPLIANCE

Pursuant to Federal Rule of Appellate Procedure 32(g)(1) and D.C. Circuit Rule (e)(2)(C), I hereby certify that the foregoing brief complies with Court's Order dated October 13, 2016, as it contains 6360 words, excluding the parts of the brief exempted by Fed. R. App. P. 32(f) and D.C. Circuit Rule 32(e)(1).

Respectfully submitted 10th day of February, 2017,

/s/ Lisa Jordan
Lisa W. Jordan

CERTIFICATE OF SERVICE

I hereby certify that on February 10, 2017, I electronically filed the foregoing brief with the Clerk of the Court by using the appellate CM/ECF system, which will send a notice of electronic filing to all registered counsel.

Respectfully submitted 10th day of February, 2017,

/s/ Lisa Jordan
Lisa W. Jordan