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Linda Regis

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From the Sandbox to Sandblasting: Regulation of Crystalline Silica

LINDA REGIS*

I. Introduction

In 1997, the International Agency for Research on Cancer (IARC) reclassified the most common mineral in the earth’s crust as a known carcinogen. For many years, excessive exposure to crystalline silica in the workplace has been recognized as causing adverse health effects. Consequently, the Occupational Safety and Health Administration (OSHA) has established exposure limits for workers in dusty jobs such as sandblasting, stone cutting, construction, and auto repair. These exposure limits have substantially reduced the risks of developing the health-related problems that were prevalent during the period of non-regulation. OSHA now plans to propose a more comprehensive crystalline silica standard in an attempt to further reduce the risk of disease. Additionally, many states are considering regulations of crystalline silica in ambient air because of the IARC reclassification.

* B.S., University of Illinois; J.D. candidate 2000, Pace University School of Law. The author would like to thank the editors and associates of the Pace Environmental Law Review for editing this comment.

1. Quartz is the most common mineral in the earth’s crust and is the most prevalent form of crystalline silica. See NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH, U.S. DEP’T OF HEALTH AND HUMAN SERVICES, PUB. NO. 96-112, ALERT: REQUEST FOR ASSISTANCE IN PREVENTING SILICOSIS AND DEATHS IN CONSTRUCTION WORKERS 1 (1996).

2. See INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, WORLD HEALTH ORGANIZATION, IARC MONOGRAPHS ON THE EVALUATION OF CARCINOGENIC RISKS TO HUMANS, SILICA, SOME SILICATES, COAL DUST AND PARA-ARAMID FIBRILLS 210 (1997) [hereinafter IARC]. IARC had previously classified crystalline silica as “possibly carcinogenic to humans” and had concluded that there was limited evidence of carcinogenicity. See id. at 31.

3. Silica also occurs in an amorphous form, but this form, as opposed to the crystalline form, is much less likely to cause silicosis. See Janet M. Hughes et al., RADIOGRAPHIC EVIDENCE OF SILICOSIS RISKS IN THE DIATOMACEOUS EARTH INDUSTRY, 158 AM. J. RESPIR. CRIT. CARE MED. 812 (1998). See also IARC, supra note 2, at 210 (concluding that there is inadequate evidence that amorphous silica may cause cancer).


5. See Hughes, supra note 3, at 812.

tion and because of the known dangers associated with excessive exposure to dust in the workplace.

This comment explains why a more stringent exposure limit, the central issue in OSHA's proposed regulations, is not necessary to prevent cancer and other adverse health effects in the workplace. This comment analyzes why regulations of crystalline silica in the ambient environment are also unnecessary. Part II of this comment examines the occupational hazards of overexposure to crystalline silica. Part II also discusses the scientific basis for the current and recommended exposure limit of crystalline silica, as well as for the IARC reclassification of crystalline silica as a "known carcinogen." Part III outlines the uncertainties and inconsistencies in the scientific data. The substantial evidence required to revise OSHA's current exposure limit is questionable because of these inconsistencies. Part III discusses what OSHA should focus on to effectively control crystalline silica in the workplace. Part IV examines the states' concerns, and their attempts to regulate silica in ambient air. Part V analyzes the slippery slope of unnecessary regulations of crystalline silica in the ambient air and the harm that can be created from over-regulation.

Lastly, Part VI concludes that until more thorough studies are complete, OSHA should not alter occupational regulations. Additionally, Part VI concludes that regulating crystalline silica in the ambient air is a wasted effort. All efforts, at both the federal and state level, should focus on more stringent enforcement of the current regulations.

II. Occupational Background

A. Adverse Health Effects

Crystalline silica is a generic term referring to the family of minerals including quartz, cristobalite, and tridymite. When workers inhale excessive amounts of crystalline silica, or silica dust, they may risk contracting silicosis, a lung disease characterized by the formation of nodular regions in the lung tissue. Thus,

7. The proposed comprehensive silica standard also includes provisions for "exposure monitoring, engineering and work practice controls, training and education, respiratory protection, and medical surveillance." Id.
8. See 3 Clifford Frondel, The System Of Mineralogy: Silica Minerals 241 (7th ed. 1962). The overwhelming majority of crystalline silica exists as quartz, which is abundant in most rocks, soils, and sands. See id.
workers in dusty trades are at the greatest risk of silicosis from exposure to crystalline silica.¹⁰

The relationship between inhaling dust containing crystalline silica and silicosis is well-established.¹¹ In the 1930s, approximately 700 workers died of acute silicosis after drilling tunnels in West Virginia.¹² Also in the 1930s, numerous lawsuits¹³ involving silicosis made silicosis the "king of occupational diseases."¹⁴ Despite controls to curb the disease, 4,313 death certificates in the United States listed silicosis as the cause of death from 1979 to 1990.¹⁵ However, these deaths may be attributable to the pre-regulation period: chronic silicosis, the most common type of the disease, occurs over twenty to forty-five years.¹⁶

Acute silicosis is a rare and highly fatal disease and usually occurs when crystalline silica exposure is at extremely high levels over a short time. See U.S. ENVTL. PROTECTION AGENCY, REP. NO. EPA/600/r-95/115, AMBIENT LEVELS AND NONCANCER HEALTH EFFECTS OF INHALED CRYSTALLINE AND AMORPHOUS SILICA 5-3 (1996). Accelerated silicosis results from exposure to high concentrations of crystalline silica over five to ten years and is more prevalent in occupations without proper respiratory protection. See id. If the exposure to crystalline silica is high enough, respiratory failure may occur in ten years, even though the worker is removed from the source of the exposure. See id. Chronic silicosis, the most common type of silicosis, usually occurs after ten or more years of exposure at lower levels. See id. at 5-4. Diagnosis of silicosis is confirmed with an x-ray. See id. at 5-5.

¹⁰ See Beckett, supra note 9, at 761.
¹² See Gerald Markowitz & David Rosner, The Reawakening of National Concerns About Silicosis, 113 PUBLIC HEALTH REP. 302 (1998). Pneumatic rock drills, developed in the early 1900s were known as "widow makers" because of the severity of dust that resulted from these dry drills. See Stephen Voynick, The Making Of A Hard Rock Miner 50-52 (1978). Modern drills are wet to control the dust and have greatly reduced the health risks. See U.S. DEP'T OF HEALTH AND HUMAN SERVICES, NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH [hereinafter NIOSH], PUB. NO. 86-102, OCCUPATIONAL RESPIRATORY DISEASES 223 (1986).
¹³ See generally Rosacci v. United States Pipe & Foundry Co., 8 A.2d 707 (N.J. 1939) (holding that the employer failed to provide reasonable prevention from disease when the employee died from silicosis); Bellows v. Merchants Dispatch Transp. Co., 12 N.Y.S.2d 655 (App. Div. 1939) (holding that employer failed to provide a safe environment with proper ventilation when employee contracted silicosis); Michigan Quartz Silica Co. v. Syring, 252 N.W. 682 (Wis. 1934) (holding that employee suffered compensable loss during employment due to silicosis).
¹⁴ See Markowitz & Rosner, supra note 12, at 303.
¹⁵ See Beckett, supra note 9, at 761.
¹⁶ See NIOSH, PUB. NO. 86-102, OCCUPATIONAL RESPIRATORY DISEASES 219 (1936).
A link between tuberculosis and silicosis has also been recognized. Some studies have associated crystalline silica exposure with extrapulmonary disease such as sclerderma, renal disease, and rheumatoid arthritis, because of the possibility that inhaled silica particles can disseminate throughout the body. Like tuberculosis, however, these extrapulmonary diseases are most prevalent in exposures resulting in silicosis. Thus, if the risk of silicosis can be controlled, the risk of the diseases that are associated with silicosis will also be controlled.

B. Current Standards Regulating Crystalline Silica

Approximately 1.7 million workers and 200,000 miners are exposed to crystalline silica. Because there is no effective treatment once a worker has developed silicosis, the best approach to combat the disease is to prevent it from occurring. Thus, numerous agencies have developed regulations to control a worker’s exposure to crystalline silica. OSHA has a current permissible exposure limit (PEL) of ten mg/m³ divided by (%SiO₂ + 2) for an eight hour time-weighted average. This approximates 0.1 mg/m³ for quartz and 0.05 mg/m³ for cristobolite. The Mine Safety and Health Administration (MSHA), which establishes exposure levels for the mining industry, uses the same PEL as established by OSHA. Two other groups, the American Conference of Governmental Industrial Hygiene (ACGIH) and the National Institute for Occupational Safety and Health (NIOSH) have created rec-

17. See Beckett, supra note 9, at 762. Specifically, a higher mortality from tuberculosis associated with silicosis has been documented. See id. Those with silicosis have three times a greater incidence of tuberculosis than those exposed to crystalline silica and who do not have silicosis. Tuberculosis is highest in those workers with accelerated or acute silicosis. See id.
18. See id. at 764.
19. See id. at 762.
21. See Beckett, supra note 9, at 764.
23. See id.
25. The American Conference of Governmental Industrial Hygienists [hereinafter ACGIH] is a private industrial hygiene group. It recommends Threshold Limit Values (TLVs) for many substances, including crystalline silica. ACGIH’s adopted TLV for respirable crystalline silica is 0.1mg/m³. See ACGIH, 1999 TLVs and BEIs: Threshold Limit Values for Chemical Substances and Physical Agents Biological Exposure Indices 61 (1999).
26. The National Institute for Occupational Safety and Health [hereinafter NIOSH] is part of the Center for Disease Control and Prevention, which is a unit of the
ommended exposure limits, which may be submitted to OSHA for consideration in establishing the PEL.\textsuperscript{27} In 1974, NIOSH recommended a stricter exposure limit of 0.05 mg/m$^3$, reducing the limit by half of that set by OSHA.\textsuperscript{28} NIOSH continues to adhere to this recommendation.\textsuperscript{29} Part of OSHA’s proposed regulation is to revise the PEL to NIOSH’s recommended exposure limit.\textsuperscript{30} The ACGIH is proposing to categorize crystalline silica as an A2, “Suspected Human Carcinogen,” and is also proposing to modify the accepted exposure value of 0.1 mg/m$^3$.\textsuperscript{31}

OSHA may receive the recommendations of interested groups or persons regarding a regulation, but OSHA may not modify the current regulation based solely on those recommendations.\textsuperscript{32} OSHA’s decision to modify a regulation must be supported by substantial evidence.\textsuperscript{33} “Substantial evidence is such relevant evidence as a reasonable mind might accept as adequate to support a conclusion.”\textsuperscript{34} The burden of proving the validity of the decision rests on OSHA.\textsuperscript{35} Furthermore, the court gives a harder look at

\begin{footnotes}
\item<sup>27</sup> See 29 U.S.C.A. § 655(b)(1) (West 1998).
\item<sup>28</sup> See NIOSH, Doc. No. 75-120, Criteria for a Recommended Standard: Occupational Exposure to Crystalline Silica 76 (1974).
\item<sup>29</sup> See id.
\item<sup>30</sup> See Occupational Exposure to Crystalline Silica, 64 Fed. Reg. at 21,553.
\item<sup>31</sup> See ACGIH, supra note 25, at 61. An A2 designation by ACGIH is appropriate when:

Human data are accepted as adequate in quality but are conflicting or insufficient to classify the agent as a confirmed human carcinogen; or, the agent is carcinogenic in experimental animals at dose(s), by route(s) of exposure, at sites of histologic types, or by mechanism(s) considered relevant to worker exposure. The A2 is used primarily when there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals with relevance to humans.

\item<sup>32</sup> See 29 U.S.C.A. § 655(1) (West 1999).
\item<sup>33</sup> See Alabama Power Co. v. Occupational Safety & Health Admin., 89 F.3d 740, 743 (11th Cir. 1996).
\item<sup>34</sup> Id.
\item<sup>35</sup> See id.
\end{footnotes}
OSHA's actions than is required for other administrative agencies that are reviewed under the deferential, rational basis standard.\textsuperscript{36} Thus, for OSHA to modify the existing rule on crystalline silica, it cannot simply base its decision on the recommendation of NIOSH. OSHA must show substantial evidence that supports the regulation.\textsuperscript{37} OSHA should, therefore, examine the numerous, conflicting data on crystalline silica exposure to support its conclusion that the current regulation needs modification.

C. Dose-Response Studies

The current and recommended exposure limits are based on varying epidemiological dose-analysis studies.\textsuperscript{38} OSHA primarily relied on a study of Ontario Gold and Uranium miners to establish the existing PEL.\textsuperscript{39} The Ontario study involved 2,109 miners who were first employed by the mine between 1940 and 1959.\textsuperscript{40} Twenty-one mines were the focus of the study because of their adequate records on dust exposure.\textsuperscript{41} Five x-ray readers were involved in reviewing the films, and if any one reader questioned the normalcy of a film, a more thorough evaluation was performed.\textsuperscript{42} The Ontario Study concluded that there was a 1.2% cumulative risk of a positive reading for a worker exposed to crystalline silica for forty years at 0.1 mg/m\textsuperscript{3}.\textsuperscript{43}

NIOSH, in its 1974 recommendation for an exposure limit that is half that of OSHA's PEL, relied primarily on a series of

\textsuperscript{36} See id. If, however, the choice is between two conflicting views, the court must uphold OSHA's decision even if the court believes it is the incorrect choice. See id. at 744.

\textsuperscript{37} In Asbestos Info. Assoc./North America v. Reich, 117 F.3d 891 (5th Cir. 1997), the plaintiff represented asbestos miners and manufacturers of the asbestos containing products of roofing sealants and coatings. See id. at 892. The plaintiff contended that there was no risk of inhaling the fibers because the fibers were encapsulated within the asphalt. See id. The court first found that the plaintiff, as representative of members who were adversely affected by the regulation, had standing. See id. at 893. OSHA argued that the regulation was necessary because the roofing sealant might break down and result in exposure. See id. The court, however, held that there was not substantial evidence of the risk and therefore struck down the regulation. See id. at 894.

\textsuperscript{38} See Beckett, supra note 9, at 762.

\textsuperscript{39} See D.C.F. Muir et al., Silica Exposure and Silicosis Among Ontario Hardrock miners: I. Methodology, II. Exposure Limits, III. Analysis & Risk Estimates, 16 AM. J. INDUS. MED. 5 (1989).

\textsuperscript{40} See id. at 6.

\textsuperscript{41} See id.

\textsuperscript{42} See id. at 7.

\textsuperscript{43} See Janet Hughes, Radiographic Evidence of Silicosis in Relation to Silica Exposure, 10 APPL. OCCUP. ENVTL. HYG. 1066 (1995).
studies from the 1920s to 1972 on Vermont granite workers. All of the Vermont studies, with the exception of the data from Therault et al., measured the exposure using impinger methods. These earlier studies concluded that a safe limit for dust exposure was approximately ten mppcf. Because of dust control methods created after these early studies, later studies concluded that few exposures exceeded five mppcf at the granite sheds. These reports found no new cases of silicosis in workers hired after 1937. Thus, NIOSH concluded that five mppcf was a safe level for crystalline silica and that five mppcf was equivalent to, in mass units, fifty ug/cu m of crystalline silica.

NIOSH also finds support for their recommendation in a study of South African gold miners employed after 1938. The South African miners were employed an average of 23.5 years. For forty years of exposure at OSHA's PEL, the study concluded that there would be a sixty percent cumulative risk of silicosis.

D. International Agency For Research on Cancer (IARC) Evaluation

The IARC monographs are recognized as an authoritative source of information on the carcinogenicity of a variety of exposures. They are designed to assist in formulating decisions and making risk assessments. The working group gathers all the available data on the topic, reviews them for scientific accuracy, and prepares an evaluation of the carcinogenicity from the rele-

44. See NIOSH, supra note 28, at 71.
46. See NIOSH, supra note 28, at 72. Impinger sampling, unlike the modern gravimetric methods, counts the dust particles without regard to size. See id. at 80. The concentration from impinger methods is reported in particle counts (mppcf) rather than mass units. See id. at 72.
47. See id. at 74.
48. See id.
49. See id.
50. See id. at 74-75.
51. See Hughes, supra note 43, at 1065.
52. See id. at 1066.
53. See id. at 1064.
54. See IARC, supra note 2, at 10.
55. See id.
vant data. When IARC reviews the data, they must take into account the possibility of bias and confounding. Because of their prevalence within the crystalline silica exposure studies, bias and confounding should have been particularly important for IARC to recognize and to consider when drawing its conclusions.

For the evaluation of crystalline silica's relation to lung cancer, the working group of IARC relied on numerous human data and animal data. The IARC working group reviewed epidemiological studies including seventeen cohort and five case control studies on ore miners; six cohort studies of quarry and granite workers; two cohort studies of refractory brick workers; three studies of ceramic/pottery workers; one study of diatomaceous earth workers; three cohort studies on foundry workers; and numerous studies on silicotics. The working group concluded that the overall findings from these studies support increased lung cancer risk from occupational exposure to crystalline silica.

Crystalline silica exposure was also evaluated in rats, mice, and hamsters by inhalation and intratrachial instillation. In the absence of adequate data on humans, it is biologically plausible to regard evidence of carcinogenicity in animals as evidence of carcinogenicity in humans. There is, however, the possibility that the exposure causes cancer only in a specific species.

56. See id. at 11.
57. See id. at 15. Bias refers to factors present in the way the study is designed or carried out that erroneously lead to a stronger or weaker result than actually exists. See id. Confounding occurs when there are other factors present that may make the relationship between the exposure and cancer stronger than it truly is. See id.
58. See id. at 33.
59. See IARC, supra note 2, at 86. There are three types of human studies. See id. at 14. Case-control and cohort studies examine an individual's exposure and the relationship to cancer. See id. Correlation studies examine whole populations and the frequency of cancer. See id. Correlation studies are not considered as valuable of data as case control and cohort studies because the causal relationship is more difficult to establish. See id.
60. See id. at 149.
61. See id. at 86-102.
62. See id. at 102-112.
63. See id. at 112-116.
64. See IARC, supra note 2, at 116-120.
65. See id. at 120-127.
66. See id. at 128-131.
67. See id. at 131-139.
68. See id. at 208.
69. See id.
70. See IARC, supra note 2, at 17.
71. See id. at 18.
was apparent in the evaluation of crystalline silica: The IARC working group concluded from review of the animal data that exposure to crystalline silica caused tumors in rats, but did not cause tumors in mice and hamsters.\(^{72}\)

III. Analysis of the Occupational Studies

A. The Inconsistencies of the Dose-Response Studies

The dose response studies prove that the prevalence of silicosis increases with increasing exposure to silica dust, but the exact level that establishes a relationship between silicosis and exposure is controversial.\(^{73}\) A major obstacle to obtaining a sound exposure limit from the currently available data is the difficulty of converting measurements taken from the impinger method, expressed in millions of particles per cubic foot of air, to limits expressed in mass units.\(^{74}\) NIOSH, however, bases its recommended exposure limit on the Vermont granite studies – studies that used the impinger method and that therefore required this problematic conversion. This issue was apparent to NIOSH at the time it developed its exposure limit: “Because of variations in types, size, and density of particles in other industries, it is not clear that the same limit, in terms of number of particles, will properly describe safe exposures in other industries producing airborne free silica.”\(^{75}\) Notwithstanding the acknowledged limitation of these studies, NIOSH applied this limit to all other industries.\(^{76}\)

Another obstacle for many of the dose response studies is underestimating the exposure to crystalline silica.\(^{77}\) The study of South African miners found a sixty percent risk of silicosis at the existing exposure limit, thus lending support to NIOSH’s recommendation for a stricter limit.\(^{78}\) However, this data is unreliable. There is a probability that the study underestimated the exposure

\(^{72}\) See id. at 150.

\(^{73}\) See Beckett, supra note 9, at 762.

\(^{74}\) See Hughes, supra note 43, at 1064.

\(^{75}\) NIOSH, supra note 28, at 75. Moreover, NIOSH recognizes that “the impinger method falls short of the ideal with regard to relevance to health hazard, simplicity, reproducibility and unit cost.” Id. at 80 (emphasis added).

\(^{76}\) See id. at 76. The Vermont granite studies by Therault et al, supra note 45, did use gravimetric methods instead of impinger counts. However, NIOSH recognized that imperfections in the data prohibited using the analysis in establishing an environmental limit. See NIOSH, supra note 28, at 72-73.

\(^{77}\) See Hughes, supra note 43, at 1068.

\(^{78}\) See id.
to crystalline silica during the employment period. A previous report indicated that the exposure during the employment period was between 0.2 to 0.5 mg/m³, while the estimate used in the study was 0.09 mg/m³. This is significantly below the earlier estimate and substantially effects the silicosis risk estimates. Additionally, the South Africa study used only one x-ray reader as opposed to the more accepted use of multiple readers.

Two other studies lend support to the results of the South African data. The first involves South Dakota gold miners who worked underground at least one year between 1940 and 1965. The results are consistent with the South African study, finding a lifetime risk for silicosis of thirty-five percent to forty-seven percent at the OSHA standard. As in the South African study, however, uncertainties are present. Silicosis was determined by the death certificates of workers in 1960 and 1976, as opposed to repeated x-rays over time. This may have created a bias, as some individuals may have had silicosis, but were not accounted for in the study. Alternatively, physicians may have over diagnosed silicosis as a cause of death because of the known incidences of silicosis in the mines. Moreover, crystalline silica was measured in millions of particles per cubic foot of respirable dust, which required the questionable conversion to mass units and an estimate of the silica content of the dust.

The second study, of 149 Leadville, Colorado residents over the age of forty, found that thirty-two percent of workers had silicosis at an estimated level of 0.064 mg/m³. However, this study had two potential biases: (1) the risk estimate may have been overestimated because of the effect of altitude on Leadville resi-

79. See id.
80. See id. at 1066.
81. See id. "A twofold underestimation could account for more than a tenfold overestimation of risk." Id. at 1064.
82. See id.
83. See Steenland & Brown, supra note 20, at 1373.
84. See id. at 1376. OSHA cites these results as support for the need to revise the current regulations. See Occupational Exposure to Crystalline Silica, 64 Fed. Reg. 21,549 at 21,553 (1999).
85. See Steenland & Brown, supra note 20, at 1373.
86. See id. at 1376.
87. See id. Other limitations in the accuracy of the data include the conversion of the data to geometric measurements and the use of an estimate of the dust exposure rather than an actual measurement. See id.
88. See id. at 1373.
dents; and (2) the miners who did not have silicosis may have left Leadville after the mines closed to gain employment elsewhere. Most importantly, in both the South Dakota and Colorado studies, as in the South African study, the underestimation of the exposure to crystalline silica most likely had a significant effect on resulting silicosis risk estimates.

Thus, the best estimates of exposure and risk of silicosis are derived from the Ontario miners data. The study used five different x-ray readers, as opposed to just one in the South African study. Moreover, an underestimation of exposure to crystalline silica would change the risk estimate much less dramatically than the underestimation in the other studies. The Ontario study concluded that for forty hours/week exposure of 0.1 mg/m³, the silicosis risk is 1.2%. Thus, the most consistent and convincing study of dose response supports the current exposure limit. The most flawed studies are the very ones used by NIOSH.

It is unfair to require industries to adhere to new, more rigorous standards that are based on limited and disputable studies. OSHA should not rely on such flawed studies as the basis for establishing a more stringent PEL. Until further dose response studies are completed to accurately enforce the need for a more stringent exposure level, OSHA lacks the substantial evidence required for such a revision.

B. Critique of IARC’s Conclusions

1. The Human and Animal Data

The IARC reclassification affirms appreciation for the importance of controlling excessive exposure to crystalline silica dust in the workplace. The reclassification, however, does not necessarily indicate that OSHA and MSHA must adopt a more stringent PEL. First, IARC represents only one body of knowledge and does not represent a recommendation with respect to regulations or legisla-

90. See id.
91. See Hughes, supra note 43, at 1068.
92. See id. at 1066.
93. See id. at 1067. If exposures were underestimated by a factor of two, the risk estimate would change from 1.2% to 3.8%. See id. at 1068. In the South African study, if the exposure was underestimated by a factor of two, the risk estimate would change from sixty percent to twelve percent. See id.
94. See id.
Second, the relationship between exposure to crystalline silica and lung cancer is still a controversial topic. Of over forty studies IARC examined, for example, only eleven studies provided data that was not severely confounded. These eleven studies were inconsistent in concluding there is a cancer risk from exposure. IARC dismisses this consistency problem by stating that some "non-uniformity of results would be expected." Only one study showed significant excess risk and this study may have been confounded by the presence of asbestos exposure.

Thus, the IARC working group essentially chose to rely on only one study out of forty to show that crystalline silica presents an excessive risk of cancer. "A single study suggesting an association between exposure to the agent and increased risk of cancer should not result in a finding of sufficient evidence of human carcinogenicity when other studies . . . do not show an association with exposure to the agent . . . ." IARC erred in basing their conclusion that crystalline silica is a known carcinogen on one study that shows an excess risk of cancer.

Moreover, as stated previously, when IARC reviews data, they must consider the effects of bias and confounding when drawing conclusions. It seems, however, that IARC chose to ignore the effects of bias and confounding. Their prevalence within the data contradicts IARC's reclassification of crystalline silica as a known carcinogen.

Smoking is a major confounding factor. There is a high presence of smoking among silicotics and blue collar workers in

95. See IARC, supra note 2, at 17.
97. See IARC, supra note 2, at 207.
99. IARC, supra note 2, at 208.
100. See Weill & McDonald, supra note 98, at 99.
101. See id. at 101.
103. See IARC, supra notes 55-57 and accompanying text.
104. See Gamble, supra note 102, at 12.
105. See id.
Heavy smokers are likely to have respiratory impairment and are more likely to be granted compensation than silicotics without such symptoms: smoking increases success of workers compensation claims for silicosis. Smoking-related illness actually triggered an examination and subsequent diagnosis of silicosis, which led to compensation for the silicosis and label of silicotic. The lung cancer risk, therefore, may be explained by smoking rather than by exposure to crystalline silica.

Although lung tumors were found in rats exposed to crystalline silica, the data did not establish a relationship between dose, duration, or method of exposure. Instead, most experiments involved an extremely high single dose, usually greater than 1.0 mg/m³, which resulted in lung overload. Thus, this high dose exposure does not establish that tumors may form at the low levels of cumulative exposure that a worker may encounter.

Most importantly, other animals tested, such as hamsters and mice, did not develop tumors. This suggests that rats have a greater propensity to develop tumors at high dosages and that rats differ from other species in this respect. “For inhaled particles, increasing evidence shows that responses of the rat lung to heavy, chronic exposures may not serve as good models for lung responses of humans to lesser exposures . . . .” Further evi-

106. See id. at 8. See also David Stipp, Cancer Scare: How Sand on a Beach Came to be Defined as Human Carcinogen, Wall St. J., Mar. 22, 1993, at A8 (noting that there is a higher rate of smoking among blue collar workers, such as sandblasters, quarry workers, and miners, than among the general population).

107. See Gamble, supra note 102, at 12.

108. See id. at 47.

109. See id. at 6. Smoking, the number one cause of lung disease, was rarely accounted for fully in the various studies. See Weill & McDonald, supra note 98, at 97.


111. See L. Martin Holland & Brooke Mossman, Crystalline Silica and Cancer, in SILICA COALITION, Comments Of The Silica Coalition on the 1998 Notice of Intended Change in the Carcinogenicity Notation for Crystalline Silica – Quartz (Appendix C) (1998). Lung overload is “an inflammatory response or impairment of normal defense mechanisms.” Id. at 3. It may be the determining factor in the rat studies that were positive for tumors. See id.

112. See id. at 10.

113. See IARC, supra note 2, at 208.


115. Id. at 9 (quoting J.L. Mauderly, Relevance of Particle-Induced Rat Lung Tumors for Assessing Lung Carcinogenic Hazard and Human Lung Cancer Risk, 105 Envtl. Health Persp. 1337 (1997)).
dence of the difference between rats and other species is the fact that female rats were found more susceptible to tumors than male rats, while there has been no finding of a similar gender bias for human lung cancer. The response in rats is unique to rats, and the danger levels in other species, such as humans, are exaggerated. The conclusion, therefore, that rats developed tumors from exposure to crystalline silica is not sufficient information to determine the risks for humans:

The finding that quartz can be carcinogenic in rats but not in other experimental animals is part of a pattern which indicates that the rat lung responds to widespread chronic damage and fibrosis with tumor production much more readily than other species. This makes the prediction of carcinogenic hazard to humans very difficult, because results from rats tend to exaggerate danger levels, particularly when extremely high doses are used.

2. Silicosis/Lung Cancer Relationship

In all experiments where tumors occurred, fibrosis was also present. When lung fibrosis, or silicosis, is not present, the evidence that crystalline silica exposure induces lung cancer is weak and inconsistent. The increased risk of lung cancer could be directly related to silicosis or to the underlying exposure to silica. The "epidemiologic evidence on crystalline silica exposure per se inducing lung cancer in the absence of lung fibrosis must still be considered scanty and inconsistent, although biologically plausible." It appears, therefore, that at a minimum, silicosis must already be contracted before the risk of lung cancer can develop. However, a relationship between silicosis and a risk of lung cancer is also uncertain. It seems obvious that if there is a relationship between silicosis and lung cancer (silicosis/lung cancer hypothesis), silicotics should show the strongest increased risk of

116. See id.
117. See Holland & Mossman, supra note 111, at 10.
118. SILICA COALITION, supra note 114, at 10-11.
119. See id.
120. See Weill & McDonald, supra note 98, at 101.
121. See Smith, supra note 96, at 617.
123. See Gamble, supra note 102, at 2.
lung cancer when compared to nonsilicotics. Silicotics are the worst case situation, exposed to high levels of crystalline silica with evidence of adverse health effects. Thus, if any workers are to be at risk for lung cancer, it should be those exposed to such conditions. Similarly, a stronger risk of cancer should also be found in silicotics with a high severity compared to silicotics with a low severity, and in silicotics with high exposure to crystalline silica compared to silicotics with low exposure. No significant differences, however, were found in the risk of lung cancer in any of these comparisons. Thus, the relationship between silicosis and lung cancer is not established convincingly.

Even if there were studies that indicated silicosis is indeed a precursor to lung cancer and that smoking is not a confounding factor, a regulation that prevents the risk of silicosis will also prevent the risk of lung cancer. The dose response studies establish that the current regulation is sufficient to prevent silicosis. The existing regulation, therefore, is also sufficient to prevent lung cancer from exposure to crystalline silica.

3. The Requirement of Substantial Evidence Fails

There are numerous data on the effects of exposure to crystalline silica. There are also, however, numerous uncertainties and variations in the conclusions drawn from one study to another. The "paucity of scientific investigation of the exposure-response relationships is striking for an occupational disease with such worldwide morbidity and mortality." The concepts that silicosis might predispose cancer (if there is a relationship at all between cancer and silicosis) or that cigarette smoking may have been an unaccounted, predominant factor in those studies that were positive for lung cancer must also be examined. Furthermore, the working group ruled that carcinogenicity in humans was not detected in all industrial circumstances studied and that carcino-

124. See id. at 7.
125. See id. at 57.
126. See id. at 7-8.
127. See id. at 8.
128. See id.
129. See Holland, supra note 110, at 1102. See also Weill & McDonald, supra note 98, at 101; U.S. ENVTL. PROTECTION AGENCY, supra note 11, at 1-5 (noting an increased risk of cancer in those with silicosis has been shown, but the risk of cancer is unclear in healthy individuals).
130. Beckett, supra note 9, at 765.
131. See Weill & McDonald, supra note 98, at 101.
132. See IARC, supra note 2, at 210.
genicity may be dependent on inherent characteristics of crystalline silica or on external factors affecting its biological activity.\textsuperscript{133} OSHA, therefore, needs more complete studies to support its proposal for a stricter exposure limit. Presently, OSHA does not possess the substantial evidence required to modify the current regulation.

C. The Need For Compliance with Current Standards

Proponents of a more stringent PEL may assert that, even absent a lung cancer concern, silicosis is still a problem that must be addressed by revising the PEL. Indeed, workers in the dusty trades are still contracting silicosis notwithstanding the OSHA and MSHA regulations that are in place.\textsuperscript{134} The problem, however, lies in the lack of compliance with the existing permissible exposure limit among certain locations or job sites. Creating a stricter PEL will only punish those companies already adhering to the OSHA and MSHA regulations and will not address the current problem of non-compliance.\textsuperscript{135} "What is needed is not more regulation, but education for uninformed blasters and employers – especially the small shops rarely inspected by OSHA or reached by trade publications."\textsuperscript{136}

The cited violations of crystalline silica exposure decreased only slightly from 1979 to 1991.\textsuperscript{137} In 1996, OSHA launched a Special Emphasis Program (SEP) for industries where dust exposure is prevalent.\textsuperscript{138} During the SEP, OSHA inspectors visited 332 workplaces to monitor the extent workers were exposed to crystalline silica.\textsuperscript{139} Through April 1, 1997, thirty percent of the

\textsuperscript{133} See id. at 211.

\textsuperscript{134} See Beckett, supra note 9, at 761 (noting that among four states participating in a surveillance project, there were 447 confirmed cases of silicosis from 1980 to 1982).

\textsuperscript{135} "Most areas of the foundry that are now in compliance will be in violation of a lower PEL that is 50% of the current value." Alfred Spada, Educating Capitol Hill on the Issues, 89 Modern Casting 50 (1999). In order to comply with a new PEL, the foundry will have to make several revisions to its operations that may not be economically or technologically feasible. See id. at 54.


\textsuperscript{137} See Jim Morris, Of Dust and Dying, Houston Chron., Aug. 9, 1992, at 22A.


\textsuperscript{139} See id.
samples OSHA collected were in excess of the PEL. One investigation, of an abrasive blasting operation, found that a worker was exposed to respirable crystalline silica eighty-one times the OSHA PEL. In another investigation, initiated by a worker’s complaint, OSHA found exposure to crystalline silica 2.52 times the OSHA PEL. In compliance with the OSHA regulation, the employer subsequently initiated engineering controls to prevent continued overexposure. After the employer’s controls were completed, the measured exposure levels were reduced below OSHA’s PEL. Thus, if controls are taken, it is possible to eliminate excessive employee exposures through engineering.

The prevalence of the disease among those who do not speak English further establishes that non-compliance is the culprit in the current cases of silicosis. Mexican immigrants, with little English and few skills, work sixty to seventy hours per week sandblasting and drilling to earn money. They are unaware of the dangers of silicosis, and it is easy for an unscrupulous employer to take advantage of the language barrier by failing to supply the proper equipment or by failing to warn the workers of the potential dangers of exposure. Despite the advances in respiratory protection, many workers continue to wear improper disposable masks or bandanas as respirators. Thus, to reduce the risk of silicosis, it is necessary for OSHA and MSHA to improve compliance with current standards through enforcement, rather than through alteration of the current regulations.

140. See id.
141. See id. “A worker was exposed to silica sand in a small room with inadequate ventilation and inadequate respiratory protection.” Id.
144. See Fairfax, supra note 142, at 508.
145. See id.
146. See id.
147. See Jim Morris, Dusty Trades Victimize Mexican Immigrants, Houston Chronicle, Aug. 10, 1992, at 1A. See also U.S. Envtl. Protection Agency, supra note 11, at 5-3 (noting that from 1990 to 1993, a cluster of 100 acute and accelerated cases of silicosis were documented among Mexican sandblasters in Odessa, TX).
148. See Morris, Dusty Trades Victimize Mexican Immigrants, supra note 147, at 1A.
149. See id.
151. See Morris, Of Dust and Dying, supra note 137, at 21A.
IV. The Ambient Environment and State Regulations

“Ambient crystalline silica is emitted into the environment as a fractional component of many types of particulate emissions.” Because of the IARC classification, states have become increasingly concerned with the non-occupational exposure of crystalline silica dust in the ambient air. States may wish to monitor the fractional components of crystalline silica in addition to other air pollutants. Some states, therefore, have considered creating regulations to control industry’s emission of crystalline silica in ambient air. State laws often trigger this consideration when a material is listed as a potential or known carcinogen. Texas is one such state that has established “Effects Screening Level” (ESL’s) for crystalline silica (quartz) of 1 ug/m$^3$ for a one hour average and 0.1ug/m$^3$ for an annual average. When a facility applies for a new construction or modification permit in Texas, the facility completes a checklist to determine if modeling and effects review is required. Thus, an excess level of crystalline silica above the ESL’s triggers the modeling and review of the effects of excessive emission on the community. An excess level neither excludes a facility from receiving a permit nor implies that public health will be adversely affected. Depending on site specific factors, an excess emission, even several times higher than the ESL, may be shown to be non-detrimental to the public health and approved by the agency. By satisfying the guidelines, the facility

152. U.S. ENVTL. PROTECTION AGENCY, supra note 11, at 3-1.
153. See id.
154. See, e.g., Wisconsin Air Toxics Rule, WISC. ADMIN. CODE § 445.06(3) (1997), which states:

The department shall monitor changes in the classification of hazardous air contaminants as reported by the American conference of government-mental industrial hygienists, the United States Environmental Protection Agency, the international agency for research on cancer, and the national toxicology program and shall prepare rule modifications to the tables to incorporate these changes. The department shall presume that any hazardous air contaminant which is included on a list of known or suspected carcinogens by both the international agency for research on cancer and the national toxicology program is a hazardous air contaminant which should be listed in table 3. This presumption may be overcome if the greater weight of the evidence demonstrates the presumption is incorrect.

156. See id. at 12.
157. See id. at 2.
is in compliance with the Texas Clean Air Act\textsuperscript{158} and with the protection of public health and welfare.\textsuperscript{159}

Oklahoma's Air Toxic regulations\textsuperscript{160} have also set an environmental standard for industry to meet with respect to crystalline silica.\textsuperscript{161} The "maximum acceptable ambient concentration" or MAAC for a twenty-four hour period is 0.5 \text{ug/m}^3.\textsuperscript{162} If the MAAC is being exceeded outside the property line, the facility must obtain permission from the Executive Director of the Air Quality Division to continue operation.\textsuperscript{163} As in Texas, an excess emission does not necessarily shut down the facility.

Based on the initial IARC classification in 1988, crystalline silica is listed under Proposition 65\textsuperscript{164} as an agent known to the State of California to cause cancer.\textsuperscript{165} Under Section 44321(b),\textsuperscript{166} carcinogens listed under Proposition 65 are to be included in the Air Toxics Hot Spots Program.\textsuperscript{167} Thus, respirable crystalline silica was included in the program.\textsuperscript{168} The Program evaluates the potential adverse health effects to determine the need for a quantitative risk analysis. The risk analysis establishes a regulatory threshold that industry must subsequently comply with in California.\textsuperscript{169} The listing under Proposition 65 also requires that manufacturers label each material containing crystalline silica with a warning that it is known to cause cancer in California.\textsuperscript{170}

\begin{flushleft}

\textsuperscript{159} See Texas Natural Resource Comm'n et al., supra note 155, at 1.


\textsuperscript{161} See id.


\textsuperscript{165} See id.

\textsuperscript{166} Cal. Health & Safety Code § 44321(b) (West 1998).


\textsuperscript{168} See id.

\textsuperscript{169} See id.

\textsuperscript{170} See Cal. Health & Safety Code § 108510 (West 1998). To reduce the risk of liability for failure to warn, California's Office of Environmental Health Hazard Assessment ("OEHHA") may receive requests for safe-use determinations. See 22 Cal. Code Regs. tit. 22 §12104. Once a product is given a safe-use determination, the warning requirement under Proposition 65 is not triggered, despite the fact that the product contains a substance known to cause cancer. The Sorptive Minerals Institute, concerned about liability from their kitty litter because it contains crystalline silica, requested, and was granted, a safe-use determination from the OEHHA. See State Reg. Alert, Issuance of a Safe-Use Determination for Crystalline Silica in Sorptive Mineral Based Pet Litter (1999).
\end{flushleft}
The California Environmental Protection Agency, upon completing its review of the potential health threats, concluded in 1995 that neither the human nor animal data provided a clear dose response relationship between respirable crystalline silica inhalation and formation of tumors.171 It found sufficient evidence that the risk of lung cancer is increased only among persons with silicosis.172 Thus, there was insufficient evidence to conduct a quantitative risk assessment and to impose environmental standards on California industries.

However, the local air control districts in California have since requested that the Air Resource Board (ARB) and the Office of Environmental Health Hazard Assessment develop health values for crystalline silica.173 As a result, the Air Resource Board issued an initial draft update in January 1999 that named crystalline silica as one of four substances for which the ARB will do a risk assessment.174 If the risk assessment concludes that crystalline silica is a Toxic Air Contaminant, the ARB will develop Air Toxic Control Measures to reduce the risk of the public's exposure.175

Michigan has considered regulations for crystalline silica in the ambient environment and has rejected them. The Michigan Air Quality Division (AQD) requested the Scientific Advisory Panel (SAP) to make recommendations regarding the regulation of crystalline silica.176 The SAP must be an independent panel of at least three members.177 One member must be from industry and no members can be from the AQD or from other employment within the state government.178 The SAP concluded that, based on the studies indicating the presence of tumors in rats, crystalline silica satisfies the definition of a carcinogen.179 The SAP noted that the conclusion receives little support in human epide-

172. See id.
173. See ARB To Develop Risk Values For MTBE and Three Other Substances, 12 No. 17 CAL. ENVTL. INSIDER (Feb.16, 1999).
174. See id.
175. See id.
176. See MICH. ADMIN. CODE r. 336.1230 (1997). The SAP may be convened to establish initial threshold screening levels and initial risk screening levels.
177. See id.
178. See id.
miological studies.\textsuperscript{180} Despite their conclusion on the definition of crystalline silica as a carcinogen, the SAP recommended that the NAAQS for PM\textsubscript{10}\textsuperscript{181} would adequately protect the general population from crystalline silica exposure.\textsuperscript{182} The SAP also noted in their recommendation that, based on the epidemiological studies, silica exposure at the current OSHA permissible exposure limit indicated no adverse health effects.\textsuperscript{183}

V. Analysis of Regulating Crystalline Silica in Ambient Air

A. The Abundance of Crystalline Silica

Crystalline silica comprises more than one quarter of the earth's crust and is the major component in ninety-five percent of the earth's rocks.\textsuperscript{184} Literally every time the wind blows, silica is emitted into the air. Regulation of this ubiquitous material in ambient air is futile.

Because of crystalline silica's natural occurrence and its variety of sources, it is impossible to regulate. Imposing regulations on industry's emission outside of the workplace will have little effect in controlling crystalline silica in ambient air. In fact, manufacturing\textsuperscript{185} and mining account for only six percent of ambient emission.\textsuperscript{186} Unpaved roadways, a common sight in rural areas, are the major source of emission.\textsuperscript{187} Paved roadways become a source of increased emission as the road naturally deteriorates.\textsuperscript{188} Emissions increase as winter de-icing agents on roads grind down

\begin{itemize}
\item \textsuperscript{180} See id.
\item \textsuperscript{182} See MICHIGAN SCIENCE ADVISORY PANEL, supra note 179, at 4 (1995).
\item \textsuperscript{183} See id.
\item \textsuperscript{184} See Jean Kiel, Silica Scare: Part II, AGGREGATES MANAGER, May 1996, at 1.
\item \textsuperscript{185} See U.S. ENVTL. PROTECTION AGENCY, supra note 11, at 3-9 (indicating that ceramic, brick, clay industries emissions are estimated to be 0.368 Tg/yr and the metallurgical industry's emissions are estimated to be 0.265 Tg/yr).
\item \textsuperscript{186} See id. at 3-5 (estimating the emission from mining and quarrying is 0.4 Tm/yr).
\item \textsuperscript{187} See id. (estimating that the emission from unpaved roads is measured to be 13.8 Tg/yr). The quantity of emission on unpaved roads varies linearly with several factors: the volume of traffic, the speed of traffic, and the weight and wheel number of the vehicles. See R.J. Dyck & J.J. Stubel, Fugitive Dust Emissions for Trucks on Unpaved Roads, 10(10) ENVTL. SCIENCE & TECH. 1046 (1976).
\item \textsuperscript{188} See U.S. ENVTL. PROTECTION AGENCY, supra note 11, at 3-7. Emissions from paved roads are estimated to be 7.0 Tg/yr. The largest contributor to emission on paved roadways results from vehicle carry out from unpaved areas. See id. Other contributing factors include surrounding soils and frequency of cleanings. See id.
\end{itemize}
with increased traffic. A state cannot regulate these major and mostly natural sources of emission.

Assuming a state was capable of controlling these varied sources, where would a state draw the line on its regulations? Biking on trails would need to be regulated. The sand traps of golf courses would become a concern. Gardening would be considered a hazardous hobby. Beaches would be fenced off. The Great Basin states would be made into vast hazardous areas.

The slippery slope of regulation is inevitable. It is obvious that regulations to control crystalline silica exposure during these activities are preposterous. So too are regulations requiring industry to control its emissions of crystalline silica in ambient air. In short, the amount of crystalline silica in the ambient air is too great, and scientific evidence of its allegedly adverse health effects is too scanty to warrant regulation.

B. Ambient/Occupational Differences

Not only is it impossible to regulate crystalline silica in ambient air, it is also unnecessary to regulate it. It is important for states to distinguish between the risk factors to workers in the workplace and the risk factors to the general population. IARC based its classification on occupational studies and classified crystalline silica as a known carcinogen in the occupational setting.

Occupational exposure is usually measured within a closed building. This measurement cannot accurately represent the crystalline silica fraction of outdoor fugitive emissions. Further, the fraction of fugitive dust "found to be silica in the occupational setting may not be representative of the ambient

189. See id. at 3-8 (stating that de-icing agents contribute to four to five percent the total emissions on paved roads).
190. See Kiel, supra note 184, at 1.
191. See IARC, supra note 2, at 210.
192. See U.S. ENVTL. PROTECTION AGENCY, supra note 11, at 3-6.
193. See id. Pollution sources are either process stream or fugitive emissions: Process-stream emissions occur when dust releases are inherent to the primary function of an activity. Fugitive emissions are ancillary to the primary activity and are not confined to the process stream. Examples of fugitive crystalline silica emissions would be soil particles containing crystalline silica entrained to the atmosphere by vehicles from unpaved roads.

Id. at 3-2.
environment." The crystalline silica may settle out faster or slower as the dust is dispersed from the site.

Information on the measurement of crystalline silica in ambient air, which is at much lower levels than found in the workplace, is sparse. One such study, however, was conducted in Idaho, a dusty state that has a large agricultural community. A database was developed to monitor PM\textsubscript{10} levels in the state. The database also recorded all of the deaths attributable to respirable disease from 1969 to 1994. The study found that the PM\textsubscript{10} samples consisted of ten percent quartz. However, Idaho residents, including farmers, had below-average lung cancer rates compared to the rest of the U.S. population.

There are no known cases of silicosis resulting from ambient air exposure to crystalline silica. Since silicosis is most likely a precursor to the risk of lung cancer (if there is a relationship between crystalline silica and lung cancer at all), there is also little risk of cancer from crystalline silica in ambient air. Thus, regulations of crystalline silica in the ambient air are unnecessary to protect the general population from the adverse health effects that may be present in the occupational setting.

Other important factors relating to silicosis that may be present in the workplace, but not in the ambient air, are particle size, the duration of dust exposure, and the structure of the crystalline silica particles. Fine silica, less than 2.5 \(\mu\)m, is present in many

\begin{itemize}
  \item 194. Id. at 3-29.
  \item 195. Id.
  \item 196. See id. at 1-5. Estimates of major construction emission, for example, are difficult because the rate of emission is affected by applying water to control dust, by the time of year, and by soil geology. See id. at 3-6. Smaller construction activities will also result in crystalline silica emissions, but there has been no attempt to quantify these emissions. See id.
  \item 198. See id.
  \item 199. See id.
  \item 200. See id.
  \item 201. See id.
  \item 202. See Beckett, supra note 9, at 761 (noting that there is no evidence of adverse health effects from brief or casual exposure to crystalline silica dust blown from industrial sites).
  \item 203. See Holland, supra note 110, at 1102. See also Weill & McDonald, supra note 98, at 101; U.S. Envtl. Protection Agency, supra note 11, at 1-5 (noting an increased risk of cancer in those with silicosis has been shown, but the risk of cancer is unclear in healthy individuals).
  \item 204. See U.S. Envtl. Protection Agency, supra note 11, at 8-5.
\end{itemize}
occupational settings. Silica in the ambient environment represents less than ten percent of the particles smaller than fifteen μm in size. Most crystalline silica particles released into the environment, therefore, are not within the respirable range that can cause serious health problems.

Duration and period of exposure are important because the risk of developing the signs of silicosis increases with age. Individuals who participated in household activities such as vacuuming and dusting had higher silica exposure even though the levels were lower than outdoors. This indicates that "peak exposures from indoor activities contribute significantly to an individual's overall cumulative exposure." Thus, to truly protect the general population from crystalline silica exposure in the ambient environment, states must also consider the activities performed inside of the home. This is further evidence of the impracticality of regulating crystalline silica.

Additionally, the general population, unlike a worker in the dusty trades, is not exposed to freshly ground crystalline silica particles. Freshly ground particles have been found to create a greater health risk than aged particles. Mining environments, because of the fresh, finely fractured dust, and the peak exposures, are more hazardous than ambient exposure. But even if it were assumed that the ambient exposure was comparable to the mining exposure, the risk of silicosis to a healthy population continuously exposed for seventy years to the highest anticipated silica levels under the NAAQS would be less than one percent. Thus, adherence to the NAAQS for PM₁₀ is adequate to protect against silicosis from ambient crystalline silica exposure. Separate regulations for crystalline silica are unnecessary.
C. Economic Analysis of Regulation

Some states may argue that it is better to err on the side of caution and allow over-regulation of crystalline silica, in the chance that over-regulation will save lives. However, "over-regulation, contrary to the prevailing perception, may actually cause a net loss of life." Although states like Texas and Oklahoma do not necessarily prohibit a facility from operating if it exceeds the state's standards, the states have still placed an undue economic burden on the facility to perform costly modeling. These costs will be borne primarily by two groups, the workers in the affected industries and the consumers of the products. Workers may lose their jobs or may receive lower pay, while consumers may have to pay more for the products because the regulations influence the prices.

Over-regulation can be just as dangerous to lives as under-regulation, because the cost of regulation affects mortality and morbidity negatively. This is true even if it were found that crystalline silica causes cancer. States can save more lives by focusing regulations on more certain causes of cancer. Thus, to require companies to bear an economic burden to unnecessarily control crystalline silica will ultimately result in a burden for all of society. The economic costs created for society in attempting to control crystalline silica depletes funds that could be used to deter more serious, more established causes of cancer.

States that require environmental crystalline silica standards will also put industry unjustifiably at risk for liability in citizens' lawsuits. The risk of liability might occur only if a facility does

217. Id. at 3.
218. See id. at 18.
219. See id.
220. See id. at 24.
221. See Martin & Tilton, supra note 216, at 26.
222. See id. at 17 (noting that air pollutants account for only 0.2% of cancer deaths and crystalline silica, if it was found to cause cancer, would account for a very small portion of the 0.2%).
223. See Orchard Lane Rd. Ass'n v. Pete Lien & Sons, Inc., 16 F.3d 416 (10th Cir. 1994). The plaintiffs filed suit for a declaratory judgment against the Defendant, a silica mining and processing company, for violations of open dumping and imminent hazard provisions. See id. Although the first claim was settled and plaintiffs therefore dropped the entire suit, the imminent hazard claim was to be based on the crystalline silica emission from the processing operation and the open dumps. See id. at n. 3.
not adhere to the regulations, thus some might not consider the risk unfair or unjustifiable. The environmental standards imposed on industry, however, treat crystalline silica as if it is an evil, synthetic material manufactured by the industry rather than as small sand particles that are carried naturally by the wind. Unlike industrial spills or dumping, crystalline silica is a natural substance. It is wrong to require corporations to adhere to such strict regulations for such a natural, universal substance.

D. Labeling Requirements

Companies distributing a product containing 0.1% silica must comply with OSHA's hazard communication requirements. The product must include a label that warns of cancer and must be included in a Material Safety Data Sheet. Because of the abundance of crystalline silica, labels are potentially required on numerous, everyday objects and another slippery slope is created. A label may be necessary on a supply of sandbox sand or on a package of kitty litter. It seems only a natural progression that soon roadways and beaches would need to be posted with warning signs. However, because of the widespread presence of silica-containing materials, labeling silica everywhere it occurs is impracticable. Moreover, the common occurrence of warning labels will lessen the effect of labels that really should be heeded—those that appear on truly hazardous materials.

Yet without the label, manufacturers face liability. Courts have held there may be no duty to warn of the danger of silica with a sophisticated user. Most persons, however, using sand for non-occupational purposes (e.g., to fill a sandbox) would proba-

225. See id. § 1910.1200(f).
226. See id. § 1910.1200(g).
227. However, in California, the Sorptive Minerals Institute was granted a safe use determination on kitty litter. See Cal. Health & Safety Code § 108510 (West 1998).
228. See Beckett, supra note 9, at 764 (noting that high-risk materials used in industry, however, should always be labeled).
229. In California v. Ace Hardware, numerous businesses were sued for failure to warn of the risks of crystalline silica. See Mark Harrison & Thomas Henry, California Proposition 65 Creates New Reporting Requirements for Aggregates Industry, Aggregates Manager 43 (Aug. 1999). Businesses that sold grinders and power tools which create dust when they are used, were included in the suit. See id.
230. See, e.g., Damond v. Avondale Indus., Inc., 718 So. 2d 551 (La. Ct. App. 4th Cir. 1998) (holding that there is a duty to warn only if it is not obvious that airborne sand is dangerous, and for a sophisticated user, this is obvious); Phillips v. A-Best Prods. Co., 542 Pa. 124, 665 A.2d 1167 (1995) (holding that appellant, supplied with
bly not be considered sophisticated users. A consumer may then attribute a development of lung cancer to filling the sandbox rather than to years of smoking, and subsequently hold the manufacturer or distributor liable for the lung cancer. There would be no differentiation to the consumer between the risk of cancer from a bag of sand and the risk of cancer from years of smoking. Manufactures will risk liability for adverse health effects that will be attributed to crystalline silica instead of to their true sources.

Problems remain, however, even if a manufacturer provides warning labels on products containing crystalline silica. The bold, simple warning labels that California requires will obviously cause alarm in many people. The alarm is unfounded because the label fails to provide further information about the amount or conditions of exposure that actually cause the risk.

E. Comparison to Asbestos

When firefighters fighting a blaze at a pottery plant saw the warning labels on some empty bags of sand, they cordoned the area a “hazardous materials hot zone” and residents were told to keep their windows closed to avoid toxic fumes. When a father noticed the warning label while filling his daughter's sandbox, he took his daughter out of the sandbox and returned the sand to the store.

These incidents demonstrate the potential for crystalline silica to become the equivalent of the asbestos scare that occurred in the 1980s. Asbestos in buildings was thought to cause cancer until the early 1990s, when it was found that only long, heavy exposure, coupled with smoking, caused lung cancer. Billions of dollars have been spent to control asbestos, yet most forms of as-

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education on the dangers of silica sand and dust masks, knew of dangers without the warning label).

231. A sophisticated user is one presumed to know the dangers and hazards of a product because of his familiarity with that product. See Natural Gas Odorizing, Inc. v. Downs, 685 N.E.2d 155, 163 (Ind. App. 1997).

232. See Kiel, supra note 184, at 4.

233. See Cal. Health & Safety Code § 108510(b)(1) (1997), which provides: If the product contains a human carcinogen, the warning shall contain the statement: "CANCER HAZARD! Overexposure may create cancer risk." Id.

234. See Kiel, supra note 184, at 2.

235. See id. at 4.


237. See id. at A1.

bestos are now known to be relatively harmless.\textsuperscript{239} Needless fear was generated about the health risks of asbestos.\textsuperscript{240} Similarly, crystalline silica regulations can create excessive costs and unnecessary fear in a state's citizens. It is important for states to consider and learn from the asbestos scare before regulating crystalline silica in ambient air.\textsuperscript{241}

States must spend money to monitor and enforce their regulations of crystalline silica. Industry must spend money to comply with the regulations, even if it is only for detailed testing to determine whether it already complies. To spend this money, time, and effort on the crystalline silica regulations for ambient air exposure is not only unnecessary and without benefit, it is also detrimental. It deters funds from proven causes of cancer. It deters enforcement and compliance of crystalline silica where it is needed most—within the occupational setting. It creates unjustified liability for industry. The only successful result of regulations on the abundant substance of crystalline silica in the ambient environment is the instillation of another cancer fear in society.

VI. Conclusion

The most common mineral in the earth's crust has been classified by an over-ambitious agency as a known carcinogen.\textsuperscript{242} OSHA now plans to propose a more stringent Permissible Exposure Limit for crystalline silica, and many states are considering regulations of crystalline silica in the ambient air. However, for OSHA to revise its current PEL of crystalline silica, there must be substantial evidence to support the change.\textsuperscript{243} This evidence does not exist.

First, the dose response studies support the current exposure level. The data that seem to represent a need for a more stringent exposure limit are marred with uncertainties and bias. Second, despite IARC's reclassification, a relationship between exposure to crystalline silica and the development of lung cancer is lacking. The human data that IARC examined were severely confounded, especially by smoking, and only one of numerous studies showed

\textsuperscript{239} See id.
\textsuperscript{240} See id.
\textsuperscript{241} See Hardy & Weill, supra note 122, at 154 (noting that pausing to consider the low ambient levels is wise given the mistakes made with asbestos).
\textsuperscript{242} See id.
\textsuperscript{243} See Alabama Power Co. v. Occupational Safety & Health Admin. 89 F.3d 740, 743 (11th Cir. 1996).
an excess risk of cancer. The animal data, concluding that crystalline silica caused tumors, were unique to high exposures in rats and are far removed from establishing the risk in humans. Thus, OSHA does not have the required evidence to revise the current standard.

Because crystalline silica is such a common mineral in the earth's crust and because dust is a natural occurrence, attempts to control crystalline silica dust in the ambient environment are futile and unnecessary. There is neither a risk of silicosis nor a risk of lung cancer at the low, infrequent levels of exposure within the ambient air. Outside of the workplace, any attempt to regulate silica will create a slippery slope of costly over-regulation, as well as unsubstantiated fear that the public's health is at risk.

It is undisputed that silicosis is a serious occupational disease. However, revising the existing permissible exposure limit is not the answer to the problem of silicosis. Workers are still at risk of silicosis today because some employers are not adhering to the current standards. OSHA and MSHA must protect workers in the dusty trades by strictly enforcing the established exposure limit. A more stringent regulation will only punish those companies that are successfully adhering to the current regulations.

Agencies should focus on enforcing the existing limit in uncontrolled workplaces, rather than using their resources to attempt to find the ideal exposure limit. State regulators should also shift their attention away from the attempts to regulate this natural, ubiquitous substance in ambient air and focus on the hazardous conditions some workers still encounter in the dusty trades. Perhaps a concentrated effort to enforce the existing regulations would put an end to both the ancient disease of silicosis and to the newly acquired fear of sand.