Before the
Occupational Safety and Health Administration
U.S. Department of Labor

Occupational Exposure to Crystalline Silica; Notice of Proposed Rulemaking, 78 Fed. Reg. 56274 (September 12, 2013); 78 Fed. Reg. 65242 (October 31, 2013); 79 Fed. Reg. 4641 (January 29, 2014)	)	Docket No. OSHA-2010-0034

Comments of the

American Chemistry Council

Crystalline Silica Panel

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Introduction and Executive Summary

The American Chemistry Council's Crystalline Silica Panel ("Panel") is pleased to submit these Comments on OSHA's proposed standard for Occupational Exposure to Crystalline Silica ("Proposed Standard"). 78 Fed. Reg. 56274 (September 12, 2013). The Panel consists of trade associations and individual companies that produce or use silica and silica-containing products or that perform operations (such as construction activity and mining) on natural materials that contain crystalline silica. As such, Panel members and/or companies that belong to the Panel's trade association members are subject to OSHA's existing Permissible Exposure Limits ("PELs") for crystalline silica (in the form of quartz, cristobalite, and tridymite) and would be affected by any change in those PELs that OSHA may make in this rulemaking as well as by any ancillary requirements it may adopt.[1]

In this rulemaking, OSHA is proposing to slash the current 8-hour time-weighted average PEL for respirable crystalline silica ("RCS") in general industry by 50 percent – from a formulaic equivalent of 100 $\mu g/m^3$ RCS to 50 $\mu g/m^3$.[2] (For construction and maritime

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[1] The Panel members are the American Foundry Society, American Petroleum Institute, Badger Mining Corporation, Concrete and Masonry Silica Coalition, ExxonMobil Corporation, Fairmont Minerals Ltd., International Diatomite Producers Association, Lafarge North America Aggregates and Concrete, Lehigh Hanson, National Industrial Sand Association, National Stone, Sand & Gravel Association, Specialty Granules, Inc., North American Insulation Manufacturers Association, The Refractories Institute, Unimin Corporation, U.S. Silica Company and Vulcan Materials Company. The Concrete and Masonry Silica Coalition includes the following members: American Concrete Pipe Association; Architectural Precast Association; Brick Industry Association; Cast Stone Institute; Interlocking Concrete Pavement Institute; National Concrete Masonry Association; National Precast Concrete Association; National Ready-Mixed Concrete Association, Prestressed/Post-Tensioned Concrete Institute; and Portland Cement Association. Various members of the Panel are filing their own comments as well as joining in these Comments.

[2] OSHA's proposed change in the definition of respirable dust has the effect of reducing the PEL by an additional 20 percent on average.

industries, the reduction is even more drastic, falling from the gravimetric equivalent of 250-500 $\mu g/m^3$ to 50 $\mu g/m^3$, a five to ten-fold reduction.) The Proposed Standard also would establish an "action level" of 25 $\mu g/m^3$ and would contain a host of ancillary provisions. These requirements would apply to a material that is the second most abundant mineral in the Earth's crust (12%), that is ubiquitous in rocks, gravel, sand, and soils, and that plays a crucial role in manufacturing, transportation, and everyday life. Accordingly, whatever regulatory action OSHA takes with regard to RCS will reverberate widely throughout the U.S. economy – affecting more than 2 million American jobs, none of which we can afford to lose.

Against that background, our position regarding OSHA's rulemaking proposal is, in brief, as follows:

1. **The best available science shows that the current OSHA PEL for general industry is appropriate to protect against silica-related disease** – as indicated by the drastic reduction in silicosis mortality that has occurred in the four decades following its adoption. While instances of silica-related disease can still be found in American workplaces, the numbers are dramatically lower than what was seen in the years before the current PEL was adopted in 1971. Moreover, the cases of silica-related disease that persist today undoubtedly are attributable to the fact that there have been (and continue to be) widespread exceedances of the existing PEL – with OSHA's own data indicating that the PEL is exceeded in about 30 percent of the samples taken by its compliance officers year after year. In many cases, these exceedances are by large margins of two to three times the PEL or even more. Indeed, according to OSHA, more than 500,000 workers currently are exposed to crystalline silica at levels that exceed 100 $\mu g/m^3$, and 265,000 are exposed above

250 μg/m^3. See 78 Fed. Reg. at 56347, 56349-56352, Table VIII-5. The large numbers of workers who are overexposed to silica as measured against the current limits almost certainly are the ones who are at risk of silica-related disease. If universal compliance with the existing PEL for general industry of 100 μg/m^3 were achieved in all workplaces where silica exposures occur, silica-related disease would begin to vanish altogether from American workplaces, as workers who were exposed to higher levels of silica in past years leave the workforce.

2. **OSHA has not established with reliable scientific evidence that reducing the PEL to 50 μg/m^3 would cause any change in mortality or morbidity in silica-exposed workers.** OSHA contends that at the current PEL, workers face a significant risk of material health impairment – primarily in the form of mortality from lung cancer, non-malignant respiratory disease, and renal disease and from silicosis morbidity. But the claimed association between silica exposure *per se* and lung cancer has been and remains controversial among researchers. Moreover, the literature indicates that if silica exposure increases lung cancer risk at all, it does so through an inflammation-mediated mechanism having a threshold that is comparable to the threshold for the relation between silica exposure and the risk of non-malignant lung pathologies such as chronic inflammation, fibrosis and silicosis. The best evidence indicates that this threshold is such that the risk of lung cancer and silicosis at the current PEL is negligible, if it exists at all. The same is true of deaths from non-malignant respiratory disease generally. At the same time, a causal association between silica exposure and renal disease mortality is questionable, and, in any event, the studies OSHA relies on do not provide a reliable basis for assessing potential risks of renal disease mortality. Furthermore, because of exposure uncertainties likely misclassification,

and because of biases and uncertainties in its exposure-response modeling, OSHA has not shown that reducing the PEL from 100 μg/m^3 to 50 μg/m^3 would result in a substantial reduction in risk even if a significant risk did exist at the current PEL for general industry.

3. **OSHA has not established that it would be technologically feasible to achieve and maintain compliance with the proposed PEL.** As noted, OSHA sampling results indicate widespread exceedances of the current 100 μg/m^3 PEL in general industry and the 250 μg/m^3 PEL in construction and maritime, and many of those exceedances are by a factor of 2, 3, or even more. Clearly, then, massive new control technology would be needed to attempt to comply with a PEL of 50 μg/m^3 let alone to achieve an action level of 25 μg/m^3. Engineering controls capable of reducing exposures to 50 μg/m^3 and maintaining them below that level are not available for various industry sectors, particularly since OSHA takes the position that a PEL may *never* be exceeded – which means that long-term average exposures must be maintained at a level that is significantly below the PEL in order to remain in compliance at all times.

4. **The proposed PEL is not economically feasible across multiple sectors of general industry – and should be withdrawn on that basis alone.** Apart from the daunting technological challenges involved, attempting to comply with a PEL of 50 μg/m^3 through the use of engineering controls would cost far more than OSHA estimates, resulting in a standard that is economically infeasible in many industry sectors. Once fundamental flaws in OSHA's cost estimates are corrected, the Panel estimates that it would cost general industry more than *$6 billion* annually to comply with the proposed rule, more than 40 times higher than OSHA estimates. The financial impact of a 50 μg/m^3 PEL combined with the costs of complying with the ancillary mandates contained in OSHA's proposal would have a

devastating impact on the revenues and profits of many employers, imperiling the long-term profitability and competitive structure of various industry sectors and likely resulting in the widespread loss of jobs in the American economy.

5. **The proposed PEL of 50 μg/m^3 is not technologically feasible because RCS exposures at that level and below cannot be measured reliably.** The available data indicate that AIHA-accredited commercial laboratories (and, indeed, OSHA itself) will be unable to reliably measure crystalline silica exposures with an acceptable degree of accuracy and precision at concentrations of 50 μg/m^3 and below – particularly when real world sampling conditions and mineral matrices are considered. OSHA implicitly acknowledges this very real concern by providing laboratories with a two-year period – beyond the effective compliance date for affected employers – to achieve the desired degree of analytical competence. However, the evidence suggests they will not be able to do so, which will make it virtually impossible for employers to reliably determine whether they are in compliance with the proposed PEL and whether actions triggered by exposures above the proposed action level need to be taken. For the two-year period during which laboratories gear up to meet their new requirements, OSHA's "cart-before-the-horse" approach will make matters even worse. The proposed standard is technologically infeasible for these reasons alone.

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In the balance of these Comments, we elaborate on and explain (with detailed references) the points summarized above, which show that the PEL for respirable crystalline silica should not be set at a level lower than 100 μg/m^3. Section I provides background information on silicosis rates in the United States over the last four decades and on the extent to which OSHA's PELs for crystalline silica have been exceeded over the years. In Section

II, we show that OSHA has not carried its burden of establishing that there is a significant risk of material health impairment at the existing general industry PEL of 100 $\mu g/m^3$ or that any such risk would be reduced substantially if the PEL were lowered to 50 $\mu g/m^3$. Accordingly, Section II addresses various issues that OSHA raises in Questions 1 and 2 in the NPRM under the heading *Health Effects*, in Questions 3-7 under the heading *Risk Assessment*, and in Question 38 under the heading *PEL and Action Level*. In Section III, we show that OSHA has not carried its burden of demonstrating that the Proposed Standard with a PEL of 50 $\mu g/m^3$ would be technologically and economically feasible in the affected industry sectors. Accordingly, Section III addresses various issues that OSHA raises in Question 8 under the heading *Profile of Affected Industries*, in Questions 9-17 under the heading *Technological and Economic Feasibility of the Proposed PEL*, in Question 18 under the heading *Compliance Costs*, and in Questions 20-22 under the heading *Economic Impacts*. The question whether crystalline silica exposures at a level of 50 $\mu g/m^3$ and below can be measured accurately and reliably by commercial laboratories goes to the issue of technological feasibility. Accordingly, Section III also addresses various issues that OSHA raises in Questions 46 and 47 under the heading *Exposure Assessment*.

The provision of the Proposed Standard being addressed throughout these Comments is the proposed PEL of 50 $\mu g/m^3$ as set forth in subsection (c) of the Proposed Standard. In elaborating on the points addressed in Section III of the Comments, we necessarily make reference to various ancillary provisions of the Proposed Standard that help determine whether a PEL of 50 $\mu g/m^3$ is technologically and economically feasible. These include direct or implied references to subsection (d) on exposure assessment, subsection (e) on regulated areas, subsection (f) on methods of compliance, subsection (g) on respiratory

protection, and subsection (h) on medical surveillance. However, these Comments (in contrast to the separate comments of various Panel members) do not address the specifics of those provisions from a legal or policy perspective apart from indicating their impact on the Proposed Standard's overall cost.

Because of the time constraints under which we labored, we have not been able to address fully all of the issues regarding the economic impact of the proposed Standard in these Comments. We anticipate submitting additional information and analyses on those issues before and during the Public Hearing.

I. Background

Crystalline silica, a compound consisting of the first and second most abundant elements in the Earth's crust (oxygen and silicon), is the second most abundant mineral in the Earth's crust, making up about 12% by weight of the crustal mass of the Earth.[3] It has been described as one of the building blocks of our planet and is considered to be to the mineral world what carbon is to the organic world. In short, silica is ubiquitous and, fortunately, is very useful as well.

As OSHA appears to recognize (see 78 Fed. Reg. at 56296), crystalline silica is perhaps the most common construction and manufacturing material in the world. It is a major component of most building products – including concrete, brick, mortar, ceramic tile, ceramic sanitary ware (*e.g.*, toilets), shingles and other items that are used in the construction of all homes and most commercial buildings. Silica is a constituent of the asphaltic-cement concrete and Portland-cement concrete used to construct roads, sidewalks, airport

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[3] See 78 Fed. Reg. at 56295; OSHA, Controlling Silica Exposures in Construction. OSHA 3362-04 (2009).

taxiways/runways, parking lots, driveways and other large hard-surfaced areas. It also is found in the crushed stone upon which railroad ties and track are placed and in the other stone, gravel, and concrete components of our transportation infrastructure. Silica is the primary raw material for manufacturing glass and for making the molds and cores used to produce metal shapes in foundries, which in turn are used extensively in cars, trucks, rail cars and many other items essential to everyday life. It also is a key component of many abrasives, paints, high tech equipment, and thousands of consumer products. In addition, silica sand plays an important role in the production of natural gas and oil through the process of hydraulic fracturing where it is used as a proppant to hold open cracks and fissures created by hydraulic pressure.

Because of crystalline silica's ubiquity and multitude of uses, it is to be expected that large numbers of workers would be exposed to crystalline silica in occupational settings – and, indeed, they are. By OSHA's estimate, 2.1 million workers employed by 533,000 entities at 534,000 establishments in general industry and maritime and in construction would be affected by the proposed Silica Standard.[4]

Given its widespread presence in rocks, sand, and soils, occupational exposure to crystalline silica is nothing new. Silicosis, the form of pneumoconiosis associated with prolonged exposure to high levels of crystalline silica, is an ancient occupational disease that has come under significant control in developed countries only in the last half century or so. Thus, the Centers for Disease Control and Prevention's analysis of silicosis mortality trends in the U.S. (based on data from the NIOSH National Occupational Respiratory Mortality

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[4] See 78 Fed. Reg. at 56347; OSHA Fact Sheet: OSHA's Proposed Crystalline Silica Rule: Overview (2.2 million exposed workers).

System or "NORMS" database) shows a decline of more than 90 percent in the overall silicosis mortality rate from 1968-2010, as the number of annual deaths with silicosis listed as either the underlying or a contributing cause decreased from 1,065 in 1968 to 101 in 2010.[5] Over that same period, there was a similar decline of approximately 90 percent in the annual Years of Potential Life Lost ("YPLL") attributed to silicosis as either the underlying or a contributing cause of death.[6] And, at the same time, the age-adjusted death rate for silicosis (as either underlying or contributing cause) declined by 95 percent – falling from 8.21 per million population in 1968 to 0.39 per million population in 2010.[7] OSHA says the incidence of silicosis mortality may be understated by a factor of 2.5-5 due to under-reporting. See 78 Fed. Reg. at 56298. But even if that speculation is correct, the percentage

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[5] See Centers for Disease Control and Prevention (Dep't of HHS) ("CDC"), National Occupational Respiratory Mortality System (NORMS): National Database Query Results on May 17, 2013. Available at http://webappa.cdc.gov/ords/norms.html. See also Silicosis Mortality, Prevention, and Control – United States, 1968-2002. Morbidity and Mortality Weekly Report 2005; 54:401-405; see also same in JAMA. 2005; 293(21) 585-586 (DOI: 10.1001/JAMA.293.21.2585). Silicosis was listed as the underlying cause in about 50 percent of these deaths and as a contributing cause in the balance. See *id.* Similarly, NIOSH researchers reported an 89 percent reduction in the annual number of silicosis deaths from 1968 – 2006 (1,135 deaths in 1968 and 125 deaths in 2006). See Nasrullah, M, et al., Silicosis Mortality with Respiratory Tuberculosis in the United States, 1968-2006. American Journal of Epidemiology. 2011; 174(7): 839-848. They also found that based on 5-year averages, silicosis mortality declined 85 percent during this period (from an average of 1,034 per year during 1968-1972 to an average of 156 per year during 2002-2006). See *id.*

[6] See Centers for Disease Control and Prevention (Dep't of HHS) ("CDC"), National Occupational Respiratory Mortality System (NORMS): National Database Query Results on May 17, 2013. Available at http://webappa.cdc.gov/ords/norms.html. See also CDC, Morbidity and Mortality Weekly Report / July 18, 2008 / 57(28); 771-775.

[7] See Centers for Disease Control and Prevention (Dep't of HHS) ("CDC"), National Occupational Respiratory Mortality System (NORMS): National Database Query Results on May 17, 2013. Available at http://webappa.cdc.gov/ords/norms.html. See also NIOSH Work-Related Lung Disease (WoRLD) Surveillance System: http://www2a.cdc.gov/drds/WorldReportData/, Table 3-2, accessed on-line on 2/28/2011.

declines of greater than 90 percent from 1968 to 2010 would remain accurate (or perhaps understated) – because, if anything, under-reporting was likely to be higher in the early years than in recent periods.

The same trend can be seen – even more starkly – for mortality from silicosis-respiratory tuberculosis (*i.e.*, deaths for which both silicosis and respiratory TB were mentioned as underlying or contributing causes on the death certificate). Thus, NIOSH researchers found that the number of silicosis-respiratory TB deaths declined from 326 in 1968 to zero in 2006.[8] By the same token, the annual average of silicosis-respiratory TB deaths declined 99.5 percent during this period (falling from an average of 239.8 per year during the five-year period 1968-1972 to an average of 1.2 per year during the five-year period 2002-2006).[9]

One important factor responsible for the steeply declining trend in silicosis mortality is that deaths in the early years of the period 1968-2006 involved individuals whose exposures to crystalline silica occurred before the introduction of the current OSHA and MSHA exposure limits and corresponding improvements in industrial hygiene practices.[10] Thus, according to the CDC, one of the main factors responsible for the declining trend in silicosis mortality from 1968-2002 was that "many of the deaths in the early part of the study

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[8] See Nasrullah, M, et al., Silicosis Mortality with Respiratory Tuberculosis in the United States, 1968-2006. American Journal of Epidemiology. 2011; 174(7): 839-848.

[9] See *id.*

[10] See CDC, Morbidity and Mortality Weekly Report / July 18, 2008 / 57(28); 771-775 ("The decline in annual silicosis-attributable YPLL is mostly attributed to the decrease in deaths from silicosis among persons aged 45-64 years, indicating the effects of implementation of exposure standards and regulations, changes in industrial activity, and other factors.").

period occurred among persons whose main exposure to crystalline silica dust probably occurred before introduction of national compliance standards for silica dust exposure" by OSHA and MSHA in the early 1970s.[11] This explains the decline in silicosis morbidity as well. Thus, approximately 88 percent of silicosis cases confirmed from 1993-2006 in the states of Michigan and New Jersey were first exposed to silica in the six decades before OSHA was created in 1970.[12]

Despite the dramatic reductions in silicosis mortality that have occurred in the last half century, some deaths from silicosis continue to occur, and cases of radiological silicosis continue to be reported (though at greatly reduced rates[13]). The reasons for this are not hard to find. Silicosis continues to be a public health issue for two reasons: First, as OSHA

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[11] See CDC, Silicosis Mortality, Prevention, and Control – United States, 1968-2002. Morbidity and Mortality Weekly Report 2005; 54:401-405; see also same in JAMA. 2005; 293(21) 585-586 (DOI: 10.1001/JAMA.293.21.2585).

[12] See NIOSH, Work-Related Lung Disease Surveillance Report (March 2012), http://www2a.cdc.gov/drds/WorldReportData/FigureTableDetails.asp?FigureTableID=2599&GroupRefNumber=F03-05, Figure 03-05, accessed on-line on 2/1/14; see also 2001 Annual Report on Silicosis in Michigan, July 10, 2002 (Docket Item # OSHA-2010-0034-0807) at 1 ("Silicosis continues to occur mainly among men born before 1940 who began working in a Michigan ferrous foundry in the 1930s, 1940s or 1950s who worked in silica for over 25 years.").

[13] See NIOSH Work-Related Lung Disease (WoRLD) Surveillance System: http://www2a.cdc.gov/drds/WorldReportData/, Table 3-11, accessed on-line on 3/10/2011 (showing the annual rate of silicosis-related discharges from short-stay non-federal hospitals declining from 6,000 in 1970 to 1,000 in 2004); Occupational Exposure to Respirable Crystalline Silica – Review of Health Effects Literature and Preliminary Quantitative Risk Assessment ("Health Effects Review"), Docket item OSHA-2010-0034-1711, at 44, Table I-7 (same); see also 2001 Annual Report on Silicosis in Michigan, July 10, 2002 (Docket Item # OSHA-2010-0034-0807) at 1 (noting that 32 cases of silicosis were reported in Michigan in 1999 compared to an average of 60-70 reports per year in earlier years). And the number of reported silicosis cases in Michigan has continued to decline – to an average of 29 per year from 1998 through 2008, with provisional data showing only 13 cases in 2009 and 16 cases in 2010. See 2010 2001 Annual Report on Silicosis in Michigan, February 17, 2012, at 1 & Figure 1.

recognizes, because of its long latency period, silicosis cases today are attributable largely to overexposures that occurred decades ago.[14] Second, exposures to crystalline silica in excess of the existing PEL continue to be widespread even today. To quote the CDC, "*intense overexposures* to respirable crystalline silica continue to occur despite the existence of legally enforceable limits."[15] These "intense overexposures" explain why silicosis mortality continues to exist and why the "decline [in YPLL attributed to silicosis] among young adults

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[14] See Health Effects Review at 39 ("Because of the long latency period of chronic silicosis (i.e., the interval between beginning of exposure to silica and the onset of disease), the deaths that have occurred in the recent past may be due to exposures that occurred decades ago."). Similarly, Table 3-13a of the NIOSH Work-Related Lung Disease (WoRLD) Surveillance System Report (updated as of June 2008) shows that only 23.8% of the silicosis cases diagnosed in Michigan, New Jersey and Ohio for the years 1993-2002 had fewer than 20 years of occupational exposure to silica, and the latency period would have been even longer since many of the cases may not have become manifest until after the worker had retired. See http://www2a.cdc.gov/drds/WorldReportData/FigureTableDetails.asp?FigureTableID=550&GroupRefNumber=T03-13a, accessed on-line on 5/31/2011. The first silica exposures of almost 90 percent of these cases had occurred in the decades before 1970, the largest number having first been exposed in the decade from 1940-1949. See *id.*, Figure 3-4 at http://www2a.cdc.gov/drds/WorldReportData/FigureTableDetails.asp?FigureTableID=552&GroupRefNumber=F03-05, accessed on-line on 5/31/2011. See also 2001 Annual Report on Silicosis in Michigan, July 10, 2002 (Docket Item # OSHA-2010-0034-0807) at 1 ("Silicosis continues to occur mainly among men born before 1940 who began working in a Michigan ferrous foundry in the 1930s, 1940s or 1950s who worked in silica for over 25 years."). Similarly, of the 104 silicosis deaths in Ohio from 1999 through December 17, 2013, 72% of the decedents were 70 years or older at time of death, implying that they had entered the workforce between 1930 and 1960. Personal communication from John Paulson of the Ohio Department of Health, Office of Vital Statistics, providing a listing of deaths with silicosis as the underlying cause).

[15] CDC, Morbidity and Mortality Weekly Report / July 18, 2008 / 57(28); 771-775 (emphasis added); see also Nasrullah, M, et al., Silicosis Mortality with Respiratory Tuberculosis in the United States, 1968-2006. American Journal of Epidemiology. 2011; 174(7): 839-848 (noting that "[s]ubstantial overexposures [as measured against currently enforceable workplace exposure limits] continue to occur, particularly in construction, manufacturing, and mining industries….").

aged 15-44 years is less marked" than among older workers.[16] These young workers likely are being slotted into jobs where there are "intense overexposures" to silica, and those very high exposures can cause accelerated or acute silicosis even in younger workers whose exposure durations are limited.

Confirmation of this assessment is provided by K. Linch and his colleagues at NIOSH, who used OSHA's IMIS data to estimate the number and percentage of workers in various SIC industry sectors exposed to at least 1, 2, 5 and 10 times the NIOSH REL in 1993. They then attempted to correlate the industry exposure data with silicosis mortality data for 1985-1993. Not surprisingly, they found a high agreement between SIC codes listed on silicosis death certificates and the industry sectors having the largest number of workers whose exposures were more than 5 - 10 times the REL.[17] This suggests that workers whose exposures were many times higher than OSHA's current PEL (particularly during the decades before Linch and his colleagues made their assessment in 1993) are the ones who may be susceptible to developing fatal cases of silicosis.

But the problem of overexposure to crystalline silica is not limited to a few job categories where the overexposures are so "intense" that they may result in acute or accelerated silicosis and early death. Rather, as OSHA's own data and analyses demonstrate, exposures in excess of the existing PEL are widespread in both general industry and

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[16] CDC, Morbidity and Mortality Weekly Report / July 18, 2008 / 57(28); 771-775; Leung, C, et al. Silicosis. Lancet. 2012; 379: 2008–18, published on-line, April 24, 2012, at http://dx.doi.org/10.1016/S0140-6736(12)60235-9.

[17] See Linch, KD, et al., Surveillance of Respirable Crystalline Silica Dust Using OSHA Compliance Data (1979-1995). Am J. Indust. Med. 1998; 34:547-558.

construction, and the severity of these overexposures – *i.e.*, the extent to which OSHA's samples exceed the PEL– often is startlingly high.

Thus, a NIOSH analysis of OSHA sampling results shows that for the years 1993 through 2003, the percent of OSHA samples exceeding the *OSHA PEL* in *Construction* ranged from a low of 24.1 percent to a high of 51.7 percent, with the most recent year (2003) having an exceedance rate of 35.4 percent.[18] In *Manufacturing*, the *PEL* exceedance rates measured by OSHA for 1993 through 2003 ranged from a low of 18.1 percent to a high of 36.2 percent, with the most recent year (2003) having an exceedance rate of 30.5 percent.[19] The NIOSH analysis also shows the percent of OSHA silica samples exceeding the OSHA PEL and NIOSH REL, respectively, during three separate periods stretching from 1979 to 2003:

- For 1979-1988, 30.7 percent of total OSHA silica samples exceeded the PEL, while 46.0 percent exceeded the REL.
- For 1989-1992, 18.4 percent of total OSHA silica samples exceeded the PEL, while 34.5 percent exceeded the REL.
- For 1993-2003, 27.4 percent of total OSHA silica samples exceeded the PEL, while 39.6 percent exceeded the REL.[20]

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[18] See NIOSH Work-Related Lung Disease (WoRLD) Surveillance System: at http://www2a.cdc.gov/drds/WorldReportData/FigureTableDetails.asp?FigureTableID=560&GroupRefNumber=T03-16b http://www2a.cdc.gov/drds/WorldReportData/, accessed on-line on 12/09/2012. The percent of Construction samples exceeding the *NIOSH REL* in those years ranged from a low of 27.8 percent to a high of 62.9 percent, with the most recent year (2003) having an exceedance rate of 43.8 percent. See *id.*

[19] See *id.* The *REL* exceedance rates in *Manufacturing* during those years ranged from a low of 26.0 percent to a high of 54.7 percent, with the most recent year (2003) having an exceedance rate of 41.2 percent. See *id.*

[20] See *id.*, Table 3-20.

OSHA's own analyses of the data show the same basic picture of widespread overexposures. Thus, in a paper published in 1995, two OSHA researchers analyzed inspection data from OSHA's Integrated Management Information System ("IMIS") for the years 1980-1992. They found that of the 255 industries inspected for silica during those years, 48% had average silica exposures exceeding the PEL.[21] In 12 of the 15 most frequently inspected industries, the mean severity value of the OSHA samples (*i.e.*, the ratio of the measured exposure to the PEL) exceeded 1 – meaning that the average OSHA-sampled exposure in those 12 industries exceeded the PEL. *Id.* And in 13 of those 15 industries, the maximum exposure levels were more than 10 times the PEL – ranging up to 153 times the PEL in gray and ductile iron foundries. *Id.* Moreover, in the 10 industries subject to 10 or more inspections which had the most severe exposures to respirable quartz, the average exposure value of the OSHA samples ranged from 2.84 times the PEL to 33.11 times the PEL – with 8 of the 10 industries having average exposures at least 8 times greater than the PEL. *Id.*

Similarly, in an analysis of 7,209 OSHA inspection data points from the IMIS database for 1988-2003, A. Yassin and colleagues at OSHA found the prevalence of silica exposure levels ≥ 0.1 mg/m^3 to be 29.9 percent, while the prevalence of silica exposure levels ≥ 0.05 mg/m^3 was 85 percent.[22] And a summary of IMIS data for the period January 1, 1992 through December 31, 2002 shows the following:

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[21] Freeman, C. & E. Grossman, Silica exposures in workplaces in the United States between 1980 and 1992. Scandinavian Journal of Work, Environment & Health. 1995; 21: 47-49.

[22] Yassin, A. et al., Occupational Exposure to Crystalline Silica Dust in the United States, 1988-2003. Environ. Health Perspectives. 2005; 113(3):255-260.

- In *Construction*, 44 percent of OSHA samples exceeded the PEL – with 13 percent being between 1 and 2 times the PEL; 7 percent being between 2 and 3 times the PEL; and 24 percent being more than 3 times the PEL.

- In *General Industry*, 31 percent of OSHA samples exceeded the PEL – with 14 percent being between 1 and 2 times the PEL; 6 percent being between 2 and 3 times the PEL; and 11 percent being more than 3 times the PEL.[23]

For the period 1997 through 2002, OSHA "identified high rates of noncompliance" with the crystalline silica PEL in both construction and general industry – with the OSHA samples showing an exceedance rate of 42% in construction and 34% in general industry. See 78 Fed. Reg. at 56293-94 & Table III-1. Moreover, "24 percent of silica samples from the construction industry and 13 percent from general industry were at least three times the OSHA PEL." *Id.* at 56293.

OSHA's National Sampling Activity results for Construction and Non-Construction sectors from January 1, 2003 through December 31, 2009 were much the same. In *Construction*, 7 percent of the OSHA samples were between 1 and 2 times the PEL; 4 percent were between 2 and 3 times the PEL; and 14 percent were more than 3 times the PEL. In *Non-Construction*, 8 percent of the OSHA samples were between 1 and 2 times the PEL; 3 percent were between 2 and 3 times the PEL; and 19 percent were more than 3 times the PEL.[24] For the period January 1, 1997 through December 31, 2009, 33 percent of OSHA's IMIS samples in construction and general industry were above the PEL – with 19 percent being more than 3 times the PEL in construction and 15 percent being more than 3 times the PEL in general industry.[25] And in considering these widespread exceedances of the

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[23] See Attachment 1 hereto (produced by OSHA's in response to FOIA Request).

[24] See 78 Fed. Reg. at 56294 & Table III-2.

[25] See Health Effects Review at 39 & Table I-5.

PEL, it is important to bear in mind that, by OSHA's reckoning, the current PEL in construction is equivalent to 250 μg/m^3 (or perhaps even higher).[26]

Most recently, NIOSH investigators reported on exposure monitoring results for oil and gas workers involved in hydraulic fracturing. They found that of 111 personal breathing zone samples of respirable silica taken at 11 different sites, 51.4 percent exceeded the current OSHA PEL, and 68.5 percent exceeded the NIOSH REL.[27] Moreover, 9 percent of the samples showed silica exposures 10 or more times the PEL, while 31 percent were greater than 10 times the NIOSH REL (or 5 times the OSHA PEL); indeed, some individual samples were 20 to 30 times the current PEL.[28] When OSHA focused on the fracturing sand workers at these sites, it found "a full-shift mean exposure of 464 μg/m^3, a median of 330 μg/m^3, and range of 10 to 2,570 μg/m^3 for this group of workers. Seventy-five percent of the sample results in this job category exceed the current PEL of 100 μg/m^3 and more than half (27 of 51 samples) exceed 250 μg/m^3."[29]

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[26] See 78 Fed. Reg. at 56328.

[27] See Esswein, E.J. *et al.*, Occupational Exposures to Respirable Crystalline Silica During Hydraulic Fracturing. Journal of Occupational and Environmental Hygiene. 2013; 10: 347–356, available on-line at http://dx.doi.org/10.1080/15459624.2013.788352. See also Esswein EJ, Breitenstein M & Snawder J. NIOSH Field Effort To Assess Chemical Exposures in Oil and Gas Workers: Health Hazards in Hydraulic Fracturing. Slides presented at Workshop On The Health Impact Assessment Of New Energy Sources: Shale Gas Extraction Sponsored by The Roundtable on Environmental Health Sciences, Research, and Medicine (Washington, DC, April 30- May 1, 2012); NIOSH Science Blog, at http://blogs.cdc.gov/niosh-science-blog/2012/05/silica-fracking/ (May 23, 2012); OSHA-NIOSH Hazard Alert: Worker Exposure to Silica during Hydraulic Fracturing, available on-line at http://www.osha.gov/dts/hazardalerts/hydraulic_frac_hazard_alert.html, last visited December 9, 2012.

[28] See *id.*

[29] OSHA, Preliminary Economic Analysis and Initial Regulatory Flexibility Analysis ("PEA") Appendix A, p. A-24 (Docket Item # OSHA-2010-0034-1720).

In sum, despite improvements in controlling silica exposures over the last half century, the data show that crystalline silica exposures in excess of OSHA's existing PEL are widespread – with the exceedance rate in OSHA samples averaging in the neighborhood of 30 percent or more in most years.[30] Indeed, by OSHA's estimate, 501,000 workers (420,000 in construction; 81,000 in general industry and maritime) currently have silica exposures that exceed 100 $\mu g/m^3$, with more than half of those (264,959) being exposed above 250 $\mu g/m^3$. See 78 Fed. Reg. at 56347, 56349-56352, Table VIII-5. And these numbers of overexposed workers would be even higher if the measurements had been made using the new ISO/CEN respirable dust model that OSHA proposes to adopt in this rulemaking.[31]

Furthermore, in a significant percentage of cases, exceedances of the 100 $\mu g/m^3$ exposure level are not just marginal. Rather, the OSHA samples show that in a large number of cases, the PEL is being exceeded by a factor of two, three, or even more. In fact, according to an evaluation performed by OSHA's Directorate of Enforcement Programs, the Average Severity per silica inspection was 9.4 in 1996, declined for several years afterwards (when the Silica Special Emphasis Program was instituted), and then began climbing again – reaching an Average Severity level of 4.0 in 2003.[32] Simply put, despite the Silica Special

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[30] See Pannell, M.A., Senior Industrial Hygienist, OSHA Office of Health Enforcement, *Impediments to Developing a Viable SiO2 Exposure Assessment Program*: Slide Presentation at the 2013 American Industrial Hygiene Conference & Exposition, May 18-23, Montreal, Canada. These national overexposure figures are mirrored at the state level in Michigan, where "[i]ndustrial hygiene inspections reveal violations of the exposure standard for silica in 36.1% of the facilities where sampling was done." 2010 Annual Report on Siliosis in Michigan, February 17, 2012, at 8.

[31] See pp. 21-22, *infra*.

[32] See Memorandum for Frank Strasheim from Richard E. Fairfax, Director Directorate of Enforcement Programs, on the Silica National Emphasis Program, February 27, 2007.

Emphasis Program, respirable crystalline silica – in the words of OSHA's enforcement chief – "continues to have one of the highest rates of employee over exposures of all chemicals for which OSHA samples."[33] As Dr. Peter Morfeld explains, the existence of these widespread and often extreme overexposures – not some alleged inadequacy of OSHA's existing PEL for general industry – is very likely "the driving cause of cases of silicosis, lung cancer, and other silica-related diseases reported in registries, screening programs and epidemiological studies" and presumably is the reason why silicosis has not yet been eliminated from American workplaces.[34] The fact of such widespread noncompliance with the existing PELs "also calls into question why OSHA fails to explain how improved enforcement of the existing rule is not superior to the proposed regulation's more stringent PEL."[35] OSHA should answer that question before it drastically reduces the current general industry PEL of 100 $\mu g/m^3$.

II. OSHA Has Not Shown that a Significant Risk of Material Health Impairment Exists at the Current PEL or that Any Such Risk Would Be Reduced Substantially if the PEL Were Lowered to 50 $\mu g/m^3$.

Before reducing the PEL for crystalline silica, OSHA first must carry its threshold burden of showing that employees are exposed to a "significant risk" of "material impairment of health or functional capacity" at the existing PEL and that the proposed

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[33] *Id.* (statement of Richard Fairfax, head of OSHA's Directorate of Enforcement Programs).

[34] Comment of Dr. Peter Morfeld on Epidemiological Issues Related to OSHA's Proposal of an Occupational Health Standard for Crystalline Silica ("Morfeld Comment"), Attachment 2 hereto, at 36.

[35] Comment of Michael L. Marlow on behalf of the Mercatus Center at George Mason University, OSHA-2010-0034-1819, at 9.

reduction in the PEL will substantially reduce that risk.[36] To meet this burden, OSHA initially must provide substantial evidence as to the level of increased risk that exists at the current PEL. And, while OSHA need not state with certainty or precision the exact point at which a harm becomes "material impairment,"[37] it can hardly claim that something that does not affect health or functional capacity during a person's lifetime and can be found only by post-mortem examination constitutes "material impairment of health or functional capacity."

In addition, as OSHA points out, for a standard to meet the "reasonably necessary or appropriate" criterion of Section 3(8) of the OSH Act, the agency must show that the standard "is economically feasible; is technologically feasible; is cost effective; is consistent with prior Agency action or is a justified departure; [and] adequately responds to any contrary evidence and argument in the rulemaking record. . . ." 76 Fed. Reg. 24576, 24579 (May 2, 2011). And, as part of the determination of technological feasibility, OSHA must show that exposures at the proposed PEL and action level can be reliably measured with an acceptable degree of accuracy and precision. Section III of these Comments will address the issues of technological and economic feasibility and measurability. In this section, we show that OSHA has failed to carry its burden of demonstrating that a workplace where crystalline silica exposures are maintained at a level consistent with a PEL of 100 $\mu g/m^3$ presents a

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[36] See Industrial Union Dep't, AFL-CIO v. American Petroleum Inst., 448 U.S. 607 (1980) ("*Benzene*"); American Textile Mfrs. Inst. v. OSHA, 452 U.S. 490 (1981) ("*Cotton Dust*"); Public Citizen Health Research Group v. United States Department of Labor, 557 F.3d 165, 176 (3d Cir. 2009) ("*Hexavalent Chromium*"); 76 Fed. Reg. 33590, 33591 (June 8, 2011) ("A standard is reasonably necessary or appropriate within the meaning of Section 652(8) if it substantially reduces or eliminates significant risk.").

[37] See AFL-CIO v. OSHA, 965 F.2d 962, 975 (11th Cir. (1992).

significant risk of material health impairment to workers or that reducing the PEL to a level of 50 $\mu g/m^3$ would substantially reduce any such risk.

OSHA's significant risk finding in the present rulemaking is based on the agency's assessment of the purported risks of mortality from lung cancer, non-malignant respiratory disease, and renal disease, and the risk of silicosis morbidity as reflected radiologically. As shown below, OSHA's assessment of these risks is flawed, and its conclusions that the risks are significant at a PEL of 100 $\mu g/m^3$ and would be substantially reduced by lowering the PEL to 50 $\mu g/m^3$ are unsupported. In considering these issues, it is worth bearing in mind four points.

First, the existing PEL was based on a Threshold Limit Value (TLV) that was designed to limit silica exposures to a particle count-based level that had been found to be adequate to prevent cases of silicosis in the Vermont granite industry. But in converting the Vermont particle-count standard of 10 million particles per cubic foot to a gravimetric standard expressed as respirable mass per cubic meter, errors were made that rendered the respirable mass standard approximately twice as stringent as the Vermont particle-count standard that it supposedly was replicating.[38] Thus, the current PEL for crystalline silica is about half the level that was actually intended by those who established the TLV on which the current PEL was based.

Second, under the proposed standard, OSHA will be using a new model as the criterion to define respirable dust. It will be shifting from the respirable dust model adopted by ACGIH in 1968 to the new ISO/CEN model that has been adopted by ACGIH and others

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[38] See Ayer, H.E., Origin of the U.S. Respirable Mass Silica Standard. Appl. Occup. Environ. Hyg. 1995; 10(12): 1027-1030. The errors involved use of an incorrect conversion factor and an effective sampling pump flow rate that was about 40% too high. See *id.*

over the last two decades.[39] The ISO/CEN model has a higher sampler collection efficiency at most particle sizes in the respirable range than the model that has long been used for evaluating compliance with OSHA's current general industry PEL.[40] Thus, applying the new model as the respirable dust criterion will result in the collection of more respirable silica – approximately 20-25% more in most workplaces – than would be collected under the 1968 ACGIH model that OSHA has been applying heretofore.[41] In effect, then, by switching to the new ISO/CEN model, OSHA will be reducing the general industry PEL from the current value of approximately 100 $\mu g/m^3$ to a level of approximately 80 $\mu g/m^3$ – even if the nominal exposure limit itself is left unchanged. (This, of course, has implications for OSHA's technological and economic feasibility analyses, which are based on an exposure profile that reflects sampling results obtained using the 1968 ACGIH respirable dust model. Under the new ISO/CEN model, the exposures of a larger number of workers would be found to exceed 50 $\mu g/m^3$ and 100 $\mu g/m^3$ (as well as 250 $\mu g/m^3$), thereby requiring more controls, more exposure monitoring, more medical exams, more regulated areas, and greater compliance costs than OSHA's analysis assumes.)

Third, by introducing a set of new ancillary requirements, OSHA will ensure that employers achieve reductions in risk even without changing the exposure limit that applies under the current PEL for general industry. See 78 Fed. Reg. at 56446 ("OSHA anticipates that the ancillary provisions in the proposed standard, including requirements for regulated

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[39] Preliminary Economic Analysis and Initial Regulatory Flexibility Analysis ("PEA") (Docket Item # OSHA-2010-0034-1720) at IV-16 – IV-19.

[40] See *id.* at IV-19.

[41] See *id.* at IV-19 – IV-20.

areas and medical surveillance, will further reduce the risk beyond the reduction that would be achieved by the proposed PEL alone."); see also *id.* at 56428.

Fourth, OSHA treats its standards as never-to-be-exceeded values – which means that an employer is deemed to be out of compliance if an employee's exposure exceeds the PEL on any single day when exposure sampling is performed. Because of sampling, analytical, and particularly day-to-day environmental variability, an employer who wishes to remain in compliance with a never-to-be-exceeded PEL must maintain long-term average exposures at a level significantly below the PEL. While the precise level will vary depending on the statistical distribution of sampling results in any given workplace, in many cases, long-term average exposures will have to be maintained at a level substantially below 50 percent of the PEL in order to meet a never-to-be-exceeded standard with a high level of confidence.[42] Hence, as OSHA recognizes, to be confident that an employee's exposure will not exceed a PEL of 100 $\mu g/m^3$ on virtually any day on which exposure monitoring might be conducted, many employers will have to control workplace exposures to a long-term average level

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[42] See Leidel, N.A., *et al.*, Exposure Measurement Action Level and Occupational Environmental Variability. HEW Publication No. (NIOSH) 76-131 (1975) (Docket Item # OSHA-2010-0034-1501); 78 Fed. Reg. at 56281, 56443. See also Buchanan, D. *et al.*, Quantitative Relationship between exposure to respirable quartz and risk of silicosis at one Scottish colliery. Institute of Occupational Medicine Research Report *TM/01/03* at 30 ("Depending on the patterns of variation of concentrations in a workplace, frequency of monitoring and effectiveness of the control measures taken in response to high concentrations," the setting of a never-to-be-exceeded maximum concentration means that "average concentrations can be between one third and one tenth of the maximum exposure limit."); Greim, H (1998). Derivation of MAK values for dusts from long-term threshold values. Occupational Toxicants. MAK Collection for Occupational Health and Safety. Wiley-VCH, Weinheim. Vol 11: 281-301; Morfeld Comment at 35 ("Calculations that assume a log-normal distribution of exposure concentrations at the workplace and apply empirically determined values for the geometric standard deviations, estimate that long-term average exposures are probably less than half of the shift limit values even when accepting an overexposure in 5% of all shifts (Greim 1998, Bochmann and Morfeld 2011).").

below 50 μg/m^3.[43] That is to say, the *effective PEL* viewed in terms of an employee's average exposure over time will be significantly less than the *nominal PEL* – and the degree of protection afforded to workers will be increased correspondingly.

Thus, if silica-related disease is a function of long-term average or cumulative exposure (as OSHA contends), OSHA's never-to-be-exceeded PEL of 100 μg/m^3 – if fully complied with – already provides protection associated with an average exposure that is significantly below the PEL and in many cases will be less than 50 μg/m^3, which is equivalent to a 45-year cumulative exposure of 2.25 mg/m^3-years. Therefore, to estimate risks associated with a PEL of 100 μg/m^3, OSHA should assume that workers will be exposed to a *long-term average* silica concentration substantially below 100 μg/m^3 (and very likely less than 50 μg/m^3), which would yield a 45-year cumulative exposure that could be less than 2.25 mg/m^3-years. And to estimate risks associated with the proposed PEL of 50 μg/m^3, OSHA should assume that workers will be exposed to a *long-term average* silica concentration substantially below 50 μg/m^3 (and very likely less than 25 μg/m^3), which would yield a 45-year cumulative exposure that could be less than 1.125 mg/m^3-years. The estimated reduction in risk associated with lowering the PEL from 100 μg/m^3 to 50 μg/m^3 would be the difference between the two values calculated on that basis. (Of course, if there is an exposure threshold for the relevant health effect at or above the higher of the two exposure values, lowering the PEL would not produce any reduction in risk.) Similarly, in estimating the compliance costs of a standard having a PEL of 50 μg/m^3, OSHA should

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[43] See 78 Fed. Reg. at 56443. ("In brief, OSHA previously determined (based in part on research conducted by Leidel et al.) that where exposure measurements are above one-half the PEL, the employer cannot be reasonably confident that the employee is not exposed above the PEL on days when no measurements are taken (Leidel, et al., 1975).").

assume that many employers will have to reduce long-term average exposures to a level of less than 25 $\mu g/m^3$ – with all the additional engineering control costs that would entail.

In sum, the combination of OSHA's never-to-be-exceeded approach to determining compliance, its proposed change in the criterion for respirable dust to the new ISO/CEN model, and its proposed requirement for exposure assessment means that long-term average exposures that comply with a standard having a nominal PEL of 100 $\mu g/m^3$ would, in many workplaces, be equivalent to about 40 $\mu g/m^3$ as exposures are measured under OSHA's current respirable dust criterion. And the imposition of other new ancillary requirements to provide medical surveillance, establish regulated areas, etc. would reduce any potential risk to silica-exposed workers even further.

A. There Are Fundamental Shortcomings and Limitations in the Risk Assessments for All of the Health Endpoints on Which OSHA's Finding of Significant Risk Is Based.

In sections II.B.-II.F below, we address OSHA's specific risk assessments for lung cancer, non-malignant respiratory disease mortality, renal disease mortality, and silicosis morbidity. Before doing so, however, we want to point out some fundamental shortcomings and limitations that characterize OSHA's risk assessments for all these endpoints and that make the resulting projections of risk incorrect and unreliable. These fundamental problems are well described in the attached Comments of Dr. Louis Anthony Cox, Jr., who identifies the overarching problems with OSHA's risk assessment (which he refers to as the "Preliminary QRA") in the numbered paragraphs below. (In the event OSHA is not familiar with Dr. Cox's work, we are submitting a one-page summary of his credentials as Attachment 3 to these Comments. His full 65-page Curriculum Vitae is attached to his Written Testimony in this proceeding.)

1. ***No non-random exposure-response association has been demonstrated at relevant exposure levels.*** The Preliminary QRA and the published articles that it relies on do not correct for well-known biases in modeling statistical associations between exposures and response. (These include study, data, and model selection biases; model form specification and model over-fitting biases; biases due to residual confounding, e.g., because age is positively correlated with both cumulative exposure and risk of lung diseases within each age category (typically 5 or more years long); and biases due to the effects of errors in exposure estimates on shifting apparent thresholds to lower concentrations). As a result, *OSHA has not demonstrated that there is any non-random association between crystalline silica exposure and adverse health responses* (e.g., lung cancer, non-malignant respiratory disease, renal disease) at exposure levels at or below 100 $\mu g/m^3$. The reported findings of such an association, e.g., based on significantly elevated relative risks or statistically significant positive regression coefficients for exposed compared to unexposed workers, are based on unverified modeling assumptions and on ignoring uncertainty about those assumptions.

2. ***OSHA has not shown that reducing exposures below currently permitted exposure levels would create any additional health benefits for workers.*** OSHA's analysis and the studies on which it relies have not demonstrated the absence of an exposure threshold above 100 $\mu g/m^3$ for the various adverse health effects considered in the QRA. In particular, the uncertainty and sensitivity analyses on which OSHA has relied do not correctly quantify the effects of exposure estimation errors on estimated thresholds (namely, to shift apparent thresholds to lower exposure levels).

3. ***Incorrect quantification of association.*** Even if a non-random association did exist, the methods used in the Preliminary QRA would not quantify it correctly. Model selection and over-fitting biases and ignored uncertainties about the correctness of assumed models, as well as errors in exposure

estimates (which OSHA and its subcontractors tried but failed to correct for) lead to overly narrow confidence intervals and artificially inflated lower bounds on exposure-associated risks.

4. ***Inadequate uncertainty characterization***. OSHA's Preliminary QRA presents confidence intervals and ranges of values for various health risks before and after a reduction in the current general industry PEL for crystalline silica of 100 $\mu g/m^3$. None of these intervals addresses the probability that further reducing the PEL will not create positive health benefits. None of them reflects any uncertainty at all about whether the underlying modeling assumptions used are correct, even though they do not fit or explain many aspects of available data, such as non-increasing exposure-response relations over substantial ranges in several studies. As a result, the most important sources and implications of uncertainty in the Preliminary QRA have not been addressed in OSHA's uncertainty analysis – *viz.*, are the premises and conclusions correct, and how probable is it that they are wrong in important ways?

5. ***No causal analysis***. Apart from the foregoing problems with OSHA's attempt to determine quantitative exposure-response associations, the Preliminary QRA is devoid of any causal analyses. It asserts causal conclusions based on non-causal studies, data, and analyses; this is technically unsound. Throughout, OSHA has conflated *association* and *causation*, ignoring the fact that modeling choices can create findings of statistical associations that do not predict correctly the changes in health effects (if any) that would be caused by changes in exposures. This lapse all by itself invalidates the Preliminary QRA's predictions and conclusions. As a result, there is no logical basis for

OSHA's predictions about how reducing exposures would reduce risk (an explicit causal prediction).[44]

Not only does OSHA's risk assessment fail to distinguish between *association* and *causation* or to apply appropriate tests to assess whether a true causal relation exists; it lacks even the most rudimentary framework for evaluating causality. OSHA does not explicitly set forth or purport to apply any specific set of criteria in a standardized manner to determine whether silica exposure can properly be said to have caused a particular adverse health effect and, if so, what the risks would be at alternative levels of exposure. The most basic criteria for determining causality – such as those listed in Table 7-1 of the National Research Council's Formaldehyde Report[45] – do not appear anywhere in OSHA's 483-page Health Effects Review and Preliminary Quantitative Risk Assessment document. Nor does OSHA's approach comport with the guidelines EPA has adopted for cancer risk assessment.[46] "Instead, OSHA simply announces its own beliefs *ex cathedra*, or repeats the judgments of others with whom it agrees, without providing a rational, independently verifiable derivation for its conclusions" and without testing them against formalized standards or criteria of causality.[47]

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[44] Comments of Louis Anthony Cox, Jr., Ph.D. on OSHA's Preliminary Quantitative Risk Assessment for Crystalline Silica, February 7, 2014 ("Cox Comments") at 1-3. The Cox Comments are submitted herewith as Attachment 4.

[45] See Committee to Review EPA's Draft IRIS Assessment of Formaldehyde; National Research Council of the National Academies, Review of the Environmental Protection Agency's Draft IRIS Assessment of Formaldehyde. The National Academies Press (Washington, D.C.) 2011 at 157. Available on-line at http://www.nap.edu/catalog/13142.html.

[46] See Cox Comments at 12-15.

[47] See *id.* at 13.

Given the foregoing problems, Dr. Cox concludes: "The evidence, discussions, and conclusions presented in the Preliminary QRA do not show that occupational exposures at the current general industry PEL of 100 μg/m^3 cause any real increase in health risks. Nor do they properly quantify the extent of any such causal impact (if there is one) or yield correct or credible predictions for how further reductions in exposure would affect risks."[48] In the balance of Section II, we will show, among other things, how the fundamental problems with OSHA's risk assessment that Dr. Cox has identified apply to the various health endpoints on which OSHA's significant risk finding is predicated.

B. Workers Do Not Confront a Significant Risk of Lung Cancer at a PEL of 100 μg/m^3.

In the NPRM and associated Health Effects Review document, OSHA presents estimates that 45 years of occupational exposure to crystalline silica at a level of 100 μg/m^3 will increase the risk of lung cancer by an amount falling within the range of 13/1,000 on the low end to 60/1,000 on the high end. See 78 Fed. Reg. at 56333. As discussed below, those projections are unfounded and unreliable.

1. Silica Exposures Have Not Been Shown To Increase the Risk of Lung Cancer in the Absence of Silicosis.

Late in 1996, an IARC Working Group recommended that crystalline silica be classified as a Group 1 carcinogen, a classification that was published in Volume 68 of the IARC Monographs in 1997. That recommendation was controversial when the assessment was made in late 1996 – with a spirited debate ending "in a narrow vote, reflecting the majority view of the experts present at that particular time."[49] The reasons for the

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[48] *Id.* at 3.

[49] McDonald, C., Editorial. Ann. Occup. Hyg. 2000; 44:3-14. See also Soutar, C.A. *et al.*, Epidemiological Evidence on the Carcinogenicity of Silica: Factors in Scientific

controversy include conflicting findings in epidemiological studies (a phenomenon that has continued in studies published after the IARC Working Group made its recommendation)[50]; the absence of clear exposure-response relationships in many of the studies that nominally were viewed as positive; difficulties in controlling for the effects of possible confounders – like smoking, radon, arsenic, asbestos, and PAHs; data suggesting that lung cancer risk is increased only among silicotics (so that a PEL protective against silicosis would prevent lung cancer risk as well); and the failure to find increased lung cancer risks in animal species other than rats (where a particle overload phenomenon may very likely be the causal factor).[51] As the British Health and Safety Executive explains:

Judgement. Ann. Occup. Hyg. 2000; 44:3-14. See also Testimony of Professor Kyle Steenland, January 24, 2014, (Docket Item No. OSHA-2010-0034-2162) at 5 (noting that the IARC decision "remained controversial").

[50] *See* Brown, T.P. & L. Rushton, Mortality in the UK industrial silica sand industry: 2. A retrospective cohort study. Occup. Environ. Med. 2005; 62: 446-452 (noting that of the nine studies identified as least confounded by IARC, four showed a clear excess cancer risk while five showed a negative or equivocal risk).

[51] Cancer bioassays performed with mice, guinea pigs, and Syrian hamsters have all been negative, even though some tested animals, such as the A-strain mouse, are notably susceptible to the induction of lung tumors. *See* Holland, L., Animal Studies of Crystalline Silica: Results and Uncertainties. Appl. Occup. Environ. Hyg. 1995; 10(12): 1099-1103; Saffiotti, U., *et al*., Carcinogenesis by Crystalline Silica: Animal, Cellular, and Molecular Studies. In: V. Castranova, *et al*., Eds., Silica and Silica-Induced Lung Diseases. CRC Press 1996, pp. 345-381. The rat, as has been noted by many investigators, is not a good model for evaluating potential human lung carcinogenicity – because a particle overload effect is a likely causative factor for lung tumorigenesis in rats. Results of experimental animal studies indicate that the rat lung is particularly susceptible to tumorigenesis following exposure to nonfibrous durable particles, and the response appears to be non-specific – with a wide variety of nonfibrous particles (including carbon black, coal dust, oil shale dust, talc, titanium dioxide, and volcanic ash) causing intrapulmonary lung tumors in the rat. *See* Mauderly, J. Relevance of Particle-induced Rat Lung Tumors for Assessing Lung Carcinogenic Hazard and Human Lung Cancer Risk. Environ. Health Perspectives. 1997; 105 (Supp. 5):1337-1346 at 1338, Table 2. In effect, the rat epithelium may be "primed" for a tumorigenic response to non-specific particulate exposure, making the rat an inappropriate model for extrapolating lung cancer risk to humans. See Cox Comments at 83 ("Rats are known to be uniquely

> The findings with crystalline silica of tumours in rats and not in mice and hamsters, and more prominent tumour response in females than males, is consistent with findings from studies with other dusts, such as carbon black and titanium dioxide. The relevance of lung tumours in rats to human health is uncertain. Experimental evidence suggests that the rat may be the most sensitive species (of those commonly used in experimental studies) to the effects of dust accumulation in the lungs. Rats generally show a more aggressive inflammatory response to lung overload (ie impairment of dust clearance rates) than do mice and hamsters, and develop tumours under these conditions when other species do not.[52]

The foregoing points were explored at length in a journal article published in 2000, taking issue with the IARC Working Group Report.[53]

In a report prepared in May 2005, Dr. Patrick A. Hessel reviewed epidemiological studies on silica and lung cancer completed after IARC Monograph 68 was published and concluded that the silica-lung cancer hypothesis remained questionable at that time.[54] In Dr. Hessel's words:

> Viewed as a whole, and considering the many factors that impact lung cancer risk, the literature published since 2000 (like the literature published earlier) does not suggest that silica exposure is a risk factor for lung cancer or that individuals with radiographic silicosis are at increased risk of lung cancer. Although some of the studies before and after 2000 have found increased rates

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sensitive to particulate pollution, for species-specific reasons that do not generalize to other rodents or mammals, including humans (Mauderly et al., 1997; Oberdorster, 1996; Nikula et al., 1997).").

[52] British Health and Safety Executive, *Respirable crystalline silica – Phase 2: Carcinogenicity* (2003) ("British HSE Phase 2 Report") at 75. OSHA Docket item OSHA-2010-0034-1057. This report is available on-line at http://www.centredoc.csst.qc.ca/pdf/Publications_Internet/HSE/2000-2005/179621HSE.pdf.

[53] Hessel, P., *et al.*, Silica, Silicosis, and Lung Cancer: A Response to a Recent Working Group Report. JOEM. 2000; 42:704-720. An updated analysis of the epidemiological literature taking account of developments up to 2011 can be found in Gamble, J., Crystalline silica and Lung cancer: A critical review of the occupational epidemiology literature of exposure-response studies testing this hypothesis. Critical Reviews in Toxicology. 2011; 41(5): 404-465.

[54] A copy of Dr. Hessel's May 2005 Report is submitted as Attachment 5 hereto.

> of lung cancer among working populations exposed to silica and among groups of workers compensated for silicosis, others have not, and exposure-response relationships have rarely been seen. Overall, the data suggest that where increased cancer risks have been seen, they can best be explained by other characteristics of the populations that have been studied (e.g., smoking, lifestyle factors).[55]

Other investigators also have noted the continuing uncertainty regarding an association between silica exposure and increased risk of lung cancer. Thus, in a Report from an International Workshop on Silica and Lung Cancer, L. Rushton and T. Brown observed that the "epidemiological literature [on silica, silicosis, and lung cancer] is indeed inconsistent."[56] Based on a meta-analysis of what they characterized as the 30 best studies on silica, silicosis, and lung cancer published between 1966 and 2001, N. Kurihara and O. Wada found that while silicosis appears to be a risk factor for lung cancer (particularly among smokers), the studies do not support the view that "'silica itself' increases lung cancer risk in humans."[57] Similarly, after reviewing 28 cohort, 15 case-control, and two proportionate mortality ratio studies evaluating the association between silica exposure (or silicosis) and lung cancer published between 1996 and 2005, C. Pelucchi *et al.* concluded that an association between silicosis and lung cancer existed but that the "issue as to whether silica *per se* materially increases lung cancer risk in the absence of silicosis" remains open.[58]

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55 *Id.* at 4-5.

56 L. Rushton and T. Brown, Epidemiological Perspectives on Silica and Health - Report from an International Workshop (2005), Electronic letter published in Occup. Environ. Med. 62:430-432.

57 Kurihara, N. & Wada, O., Silicosis and Smoking Strongly Increase Lung Cancer Risk in Silica-Exposed Workers. Industrial Health. 2004; 42: 303-314.

58 Pelucchi, C. *et al.*, Occupational silica exposure and lung cancer risk: a review of epidemiological studies 1996-2005. Annals of Oncology. 2006; 17(7): 1039-1050.

In 2007, I.T.S. Yu *et al.*, reported on a study of lung cancer mortality among silicotic workers in Hong Kong. They found no consistent exposure-response relationship between silica dust (measured as duration of exposure, cumulative dust exposure, and mean dust exposure) and lung cancer death, or between severity of silicosis (profusion of small opacities) and lung cancer death.[59] Concluding that their study "did not offer positive support to a link between silica or silicosis and lung cancer," the authors opined that the "classification of silica dust as a human carcinogen might need to be reviewed."[60]

In a 2007 update and further analysis of the mortality studies of Chinese tungsten miners, tin miners, iron-copper miners, and pottery workers, W. Chen, F. Bochmann and Y. Sun observed no relationship between silica exposure and lung cancer after adjusting for occupational confounders (notably arsenic in tin mines and PAHs in potteries).[61] In particular, increased lung cancer risk was not found in the tungsten miners, who had the highest silica exposures but no significant confounding exposures to arsenic or PAHs. The authors state that their analysis provides no evidence indicating that exposure to crystalline silica causes lung cancer in the absence of confounding factors, and it does not support the

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[59] Yu, I.T.S. *et al.*, Lung cancer mortality among silicotic workers in Hong Kong – no evidence for a link. Annals of Oncology. 2007; 18: 1056-1063. The authors explain why other studies (including Pelucchi *et al.*) have been more likely to find a relationship between silicosis and lung cancer than their study – namely, confounding by other occupational exposures, inadequate adjustment for smoking, selection bias, and low socioeconomic status of silicotic workers.

[60] *Id.*

[61] Chen, W., Bochmann, F. & Sun, Y. Effects of work related confounders on the association between silica exposure and lung cancer: a nested case-control study among Chinese miners and pottery workers. Int. Arch Occup Environ Health. 2007; 80:320-326.

hypothesis that crystalline silica exposure is causally associated with increased risk of lung cancer.[62]

A recent mortality study of 17,644 medical surveillance participants in the German porcelain industry by T. Birk *et al.* reaches a similar conclusion. The authors found that death from lung and renal cancers and from non-malignant renal disease was not associated with employment or silica-exposure surrogates in this large cohort (when the analysis used either the German population or the Bavarian population as referents).[63] Among other things, the SMR for lung cancer was not elevated in the subgroup of men who had work experience in the "preparation area" where silica exposures were highest (averaging in excess of 0.15 mg/m^3). Putting their study in context, the authors noted that research reports and reviews published since the 1997 IARC classification have continued to generate divergent evidence and conclusions as to the human carcinogenicity of crystalline silica in the absence of silicosis and/or at low to moderate levels of exposure. In a further analysis of the German porcelain industry cohort, the authors found that exposure to respirable silica was not associated with mortality from lung cancer, kidney cancer, or any other cause of death (except silicosis), even when cumulative silica exposures exceeded 4 mg/m^3-years.[64]

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[62] *Id.* In addition, like Yu *et al.* (2007), the authors point to methodological limitations in studies of the relationship between silicosis and lung cancer and an additional possible bias resulting from a positive association between silicosis and smoking.

[63/] Birk, T. *et al.*, Mortality in the German Porcelain Industry 1985-2005: First Results of an Epidemiological Cohort Study. JOEM. 2009; 51, No. 3: 373-385.

[64] Mundt, K. *et al.*, Respirable Crystalline Silica Exposure-Response Evaluation of Silicosis Morbidity and Lung Cancer Mortality in the German Porcelain Industry Cohort. JOEM 2011; 53(3): 282-289.

In a recent study of Swedish foundry workers, H. Westberg, *et al.* made a similar finding with respect to lung cancer morbidity (*i.e.*, incidence values). Although the overall incidence of lung cancer was elevated in the cohort (which had exposures to phenol, formaldehyde, PAHs, isocyanates, and asbestos in addition to silica), there was no association with silica exposure. Indeed, the authors found a non-significant *negative* exposure-response with silica exposure levels using both external and internal comparison groups.[65]

Recently, T. Erren *et al.* searched the PubMed data base from 1966 through January 2007 for reports of lung cancer in silica-exposed persons with and without silicosis. They then applied meta-analytical techniques to see whether they could determine if silica exposure in the absence of silicosis is associated with an increased risk of lung cancer. While they found a significant link between silicosis and lung cancer, their analysis of the studies left open the question whether exposure to silica increases the risk of lung cancer in the absence of silicosis.[66] In a further update of this meta-analysis, the authors reached the same conclusion – *i.e.*, the question whether silica causes lung cancer in non-silicotics remains open.[67]

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[65] Westberg, H., *et al.*, Cancer morbidity and quartz exposure in Swedish iron foundries. Int Arch Occup Environ Health. May 22, 2012 [Epub ahead of print]. Available on-line at http://dx.doi.org/10.1007/s00420-012-0782-4.

[66] Erren, T.C. *et al.*, Is exposure to silica associated with lung cancer in the absence of silicosis? A meta-analytical approach to an important public health question. Int. Arch. Occup. Environ. Health. 2009; 82(8): 997-1004 (Published online: Dec. 6, 2008 at http://dx.doi.org/10.1007/s00420-008-0387-0).

[67] Erren, T.C. *et al.*, Meta-analysis of published epidemiological studies, 1979-2006, point to open causal questions in silica-silicosis-lung cancer research. Med Lav. 2011; July-Aug 102(4): 321-335. A recent update of the Stoke-on-Trent pottery workers cohort also failed to find any clear relationship between mean or cumulative silica exposure and

Perhaps of most interest and relevance for present purposes – because the cohort has been studied so extensively in the past and because the present PEL is based indirectly on experience in the Vermont granite industry – is the mortality study of Vermont granite workers published in 2011.[68] While the Vermont granite workers cohort has been studied on a number of previous occasions, this is the most comprehensive mortality study of Vermont granite workers conducted to date. It includes more workers (7,052), has a longer follow-up (average of 38 years), and reflects more complete mortality ascertainment than previous studies. In addition, work histories and exposure estimates were based on multiple sources of information, some of which had not been used in previous studies. The investigators performed a nested case-control analysis, using conditional logistic regression to model the relationship between mortality and each of three different exposure variables – cumulative exposure, exposure duration, and average exposure intensity. Cumulative exposure was analyzed both as a continuous variable and as a categorical variable. No significant associations were observed between respirable silica exposure (measured both by excluding exposures occurring within 10 years of death and, alternatively, by including them) and mortality from lung cancer. This was true of all three of the exposure metrics (cumulative exposure, average exposure, and duration of exposure), whether expressed as a continuous variable or a categorical variable divided into quintiles of the combined exposure distribution

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increased risk of lung cancer. Cherry, N. *et al.*, Mortality in a cohort of Staffordshire pottery workers: follow-up to December 2008. Occup Environ Med, published online October 26, 2012. Available on-line at http://oem.bmj.com/content/early/2012/10/25/oemed-2012-100782.full.html.

[68] Vacek, P., Verma, D., Graham, W. & Gibbs, G., Mortality in Vermont granite workers and its association with silica exposure. Occup Environ Med. 2011; 68: 312-318, available on-line at http://dx.doi.org/ 10.1136/oem.2009.054452.

for cases and controls.[69] These findings are consistent with an earlier study of the Vermont granite worker cohort where the investigators found that despite large differences in quartz exposure between the pre-1940 and post-1940 hire groups, the SMRs for lung cancer were elevated to about the same degree when tenure and latency for the workers are the same – *i.e.*, there was essentially no difference in lung cancer mortality despite large differences in quartz exposure.[70] As noted by Dr. Cox, an "intervention"-type analysis of this sort is one way to help determine whether a possible association between exposure and adverse health outcome is causal or not;[71] in this case, the answer was no.

OSHA rejects the findings of the Vacek *et al.* (2011) study, preferring to rely instead on an earlier study of the Vermont granite worker cohort by Attfield and Costello (2004),[72] which OSHA used to estimate an increased lung cancer risk for silica-exposed workers that is higher (by a factor of 2-4 or more) than the increased risk of lung cancer that it estimated on the basis of any other study of any cohort in any industry.[73] That fact alone should have given the Agency pause – and it does not stand alone. There are a host of other reasons as

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[69] Although the SMR for lung cancer was elevated for the cohort as a whole, the prevalence of smoking among cohort members was higher than in the comparison populations, a factor that the authors noted could account for the elevated SMR observed in their study. In addition, because there were considerable gaps in Vermont granite work among a significant proportion of the cohort, many of the workers may have been exposed occupationally to other lung carcinogens (*e.g.*, asbestos) outside the granite industry.

[70] W. Graham, *et al.*, Vermont Granite Mortality Study: An Update With an Emphasis on Lung Cancer. JOEM. 2004; 46(5): 459-466.

[71] See Cox Comments at 11, Table 1, 50 & 51.

[72] Attfield, M.D. & Costello, J. (2004). Quantitative exposure-response for silica dust and lung cancer in Vermont granite workers. Am J Ind Med 45:129–138. OSHA-2010-0034-0543.

[73] See Health Effects Review at 351.

well why Vacek *et al.* (2011) provides a more accurate and better supported picture of potential silica-related lung cancer risk among Vermont granite workers than does Attfield and Costello (2004). When the two studies are compared, we find the following:

- Attfield and Costello had 5,414 workers in the cohort; Vacek *et al.* had 7,052 (about 1,700 more), and Vacek *et al.* covered a wider range of years.

- Vacek *et al.* had 356 lung cancer cases in their cohort, compared to Attfield and Costello's 201.

- Follow-up in Vacek *et al.* extended through 2004, whereas follow-up in Attfield and Costello terminated in 1994 – so Vacek *et al.* had ten more years of follow-up. (OSHA mistakenly says there were only four additional years of follow-up in the Vacek *et al.* study compared to Attfield and Costello,[74] when in fact there were ten).

- Vacek *et al.* had more complete mortality ascertainment than Attfield and Costello. In addition, "although both studies used employment information collected as part of the DIH [Vermont Department of Industrial Health] surveillance program, [Vacek *et al.*] . . . re-examined this data and augmented it with information from other sources. This revealed that the DIH information [on which Attfield and Costello relied] was incomplete for many workers."[75] In fact, Vacek *et al.* found that "162 workers, whom Attfield assumed were alive in 1994, had died before that time and some died decades earlier."[76]

- The exposure data in Attfield and Costello was summary information from particle count measurements taken at various times from 1924-1977. Vacek *et al.* used 5,204 exposure measurements made in the Vermont granite industry between 1924 and 2004, including a large number of additional measurements and raw data not used by Attfield and Costello.

- Attfield and Costello used a conversion factor of 10 mppcf = 0.075 mg/m^3 respirable crystalline silica (RCS). Vacek *et al.* used a conversion factor of 10

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[74] See Supplemental Literature Review of Epidemiological Studies on Lung Cancer Associated with Exposure to Respirable Crystalline Silica ("Supplemental Health Effects Review") at 4.

[75] Vacek, P., Verma, D., Graham, W. & Gibbs, G., Mortality in Vermont granite workers and its association with silica exposure. Occup Environ Med. 2011; 68: 312-318, available online at http://dx.doi.org/ 10.1136/oem.2009.054452.

[76] Letter of November 18, 2013 from Pamela M. Vacek, Ph.D. and Peter W. Callas, Ph.D. to William Perry. Docket Item # OSHA-2010-0034-1804.

mppcf = 0.1 mg/m^3 RCS because a majority of reported research supports that value. In addition, both NIOSH and OSHA recommend use of 10 mppcf = 0.1 mg/m^3 as a conversion factor in silica studies.[77] Indeed, that is the conversion factor OSHA has used in this rulemaking.[78]

- In Attfield and Costello, work histories (and associated exposures) were truncated at the date of the worker's last medical exam, so exposures after that date are not included.[79] This results in exposures being underestimated and the Exposure-Response (E-R) coefficients being given an upwards bias of unknown magnitude. The difference between Vacek *et al.* and Attfield and Costello in this respect is explained by Dr. Vacek as follows:

> Although both studies used data abstracted from the DIH surveillance program records to reconstruct work histories, we used additional data sources (pension records and interviews from other studies). The work histories collected by the DIH were obtained by self-report when a worker had a chest radiograph. In previous studies it was assumed that a person remained in the same job until the next x-ray or until retirement. For workers with only one or two x-rays this could be very inaccurate. Evidence from pension records indicates that some workers only reported the work performed for their current employer. The pension records also indicated that workers frequently left and rejoined the Vermont granite industry, and these gaps in employment were often not reported on the DIH work history.[80]

- Some of the exposure assignments used by Attfield and Costello appear suspect on their face. For example, they show silica exposures of sandblasters to be 0.06 mg/m^3 prior to 1940, 0.05 mg/m^3 from 1940-1950, and 0.04 mg/m^3 after 1950.[81] These values are simply not credible. By OSHA's own estimate, more than 57% of abrasive blasters in the cut stone industry are

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77 See pp. 126-127 & nn. 357 & 358, *infra.*

78 See Health Effects Review at 268.

79 See Cox Comments at 79.

80 Letter of November 18, 2013 from Pamela M. Vacek, Ph.D. and Peter W. Callas, Ph.D. to William Perry. Docket Item # OSHA-2010-0034-1804.

81 See Attfield, M.D. & Costello, J. (2004). Quantitative exposure-response for silica dust and lung cancer in Vermont granite workers. Am J Ind Med 45:129–138 at 131, Table I.

exposed above 0.05 mg/m^3 today, while 43% are exposed above 0.10 mg/m^3, and 28.6% are exposed above 0.25 mg/m^3.[82]

- Attfield and Costello claim to have formed their 8 categorical cumulative exposure groups by subdividing the range of cumulative exposure so as to have "roughly equal" numbers of deaths from lung cancer in each exposure range grouping. However, the number of respiratory cancer deaths in the various groups varies by a factor of two – from a low of 15 in the second group to a high of 30 in the eighth, which suggests there may have been something wrong in the way they set up the categories. Moreover, there is an inconsistency in the reported study as to the exposure cut-off value for the eighth exposure group. Table III in Attfield and Costello indicates that it begins at 5 mg/m^3-years, while Tables IV, V and VI show it beginning at 6 mg/m^3-years.

- Vacek *et al.* used all their data in evaluating potential E-R trends with increasing exposure. Attfield and Costello did not. Instead, on a *post hoc* basis, they excluded the highest exposure category from their analysis when they discovered that the E-R trend for lung cancer was not significant if that group was included (even though the trends for non-malignant respiratory diseases were significant when all the data were used). This is an example of both data selection bias and confirmation bias.[83] Moreover, as Dr. Cox points out: "It is a problem of Attfield and Costello's statistical modeling that inferences about lower-exposure effects depend on decisions about whether to include high-exposure group data. Logically, what is true at low exposures should not depend on modeling choices for high exposures."[84]

- Finally, Vacek *et al.*'s finding of no association between silica exposure and lung cancer risk in Vermont granite workers is consistent with the findings of the "intervention-style" study by Graham *et al.* (2004) and the proportional mortality study by Davis *et al.* (1983) on which Attfield and Costello relied for their exposure information.[85] By contrast, Attfield and Costello's purported findings are at odds with the results of all three of these studies.

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82 See PEA at III-51, Table III-5.

83 See Cox Comments at 22, 25, 55.

84 *Id.* at 81.

85 Davis, L. *et al.*, Mortality Experience of Vermont Granite Workers. American Journal of Industrial Medicine. 1983; 4:705-723. As the British Health and Safety Executive notes: "Overall, the analyses [in Davis *et al.* (1983)] showed no association between lung cancer risk and cumulative exposures to quartz, even in subjects with the highest cumulative dust exposures." British HSE Phase 2 Report at 27.

In sum, when judged without a result-oriented confirmation bias, the larger, more recent, more comprehensive, and more detailed study by Vacek *et al.* (2011) must be deemed to supersede Attfield and Costello (2004) as the basis for evaluating potential silica-related lung cancer risks in the Vermont granite industry. OSHA, however, rejects Vacek *et al.* (2011) on grounds that are confusing and unfounded.

For example, OSHA states that "the quintiles used in the Vacek et al. analysis were higher than typical values of cumulative exposure to silica used in many studies upon which OSHA based its risk assessment."[86] And it goes on to note that in the ten cohort pooled analysis by Steenland et al. (2001), the median cumulative exposures ranged from 0.13 to 11.37 mg/m^3-years.[87] But OSHA does not explain why there is anything wrong or unusual about conducting a categorical analysis based on a proportional grouping of exposures into quintiles of the combined exposure distribution of cases and controls for each mortality endpoint. In fact, Steenland *et al.* (2001) used quintiles as well. And, although the median cumulative exposure value for the Vacek *et al.* cohort is not given in the paper, the categorical data shows that it surely fell within the range of 0.13 to 11.37 mg/m^3-years.

In fact, the quintiles of exposure used by Vacek *et al.* are *not* higher than typical values. OSHA's Table 1 on page 4 of the Supplemental Health Effects Review purports to show that they are higher. But the Table is grossly misleading. It compares the *silicosis* exposure categories of Vacek *et al.* with the *lung cancer* exposure categories of Attfield and Costello, an "apples-and-oranges" comparison of the first order. The *lung cancer* exposure quintiles in Vacek *et al.* ranged from <0.26 mg/m^3-years to >4.1 mg/m^3-years – so they were

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86 Supplemental Health Effects Review at 3.

87 *Id.*

quite similar to Attfield's exposure categories, which ranged from <0.25 mg/m^3-years to >5 (or 6) mg/m^3-years (depending on whether Table III or Tables IV, V and VI in Attfield and Costello show the correct exposure categories). Moreover, Vacek's cumulative exposure groups were all within the range of cumulative exposures that would be accumulated in 45 years at today's general industry PEL. The first four quintiles go up to 4.1 mg/m^3-years, and the last one is 4.1 mg/m^3-years or higher. At a PEL of 0.1 mg/m^3, 45 years exposure would create 4.5 mg/m^3-years of cumulative exposure. By contrast, Attfield and Costello's 7th exposure category goes up to 6 mg/m^3-years, and their eighth (which they discarded *ex post facto* after seeing the results) is >6 mg/m^3-years. So there is no basis for claiming that Vacek *et al*. used atypically high exposure quintiles when compared to Attfield and Costello or to Steenland *et al*. (2001), or that they are out of line with exposures at the current PEL.[88] In any event, as Dr. Vacek points out, the "primary exposure-response analyses" in her study "were based on continuous measures of exposure," which OSHA puzzlingly ignores.[89]

OSHA's next objection to Vacek *et al.* is as follows:

> The regression models used in the Vacek study also exhibited signs of uncontrolled confounding. For instance, for every outcome (except silicosis), workers in the second lowest exposure stratum in the models exhibited a lower risk than those in the lowest stratum of cumulative silica exposure [though in none of these cases were the odds ratios significantly lower]. In the highest exposure (fifth) stratum, all outcomes except non-malignant respiratory disease showed a decline in the likelihood of the outcome (calculated as odds ratio) compared to the next lower stratum. These two

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[88] OSHA's Table 1 also is misleading in comparing the number of workers in the Vacek *et al.* and Attfield and Costello studies. OSHA presents the number of cases + controls for *silicosis* in Vacek *et al*., but the number of cases + controls for *lung cancer* (which is the relevant endpoint for comparison to Attfield and Costello) was much higher.

[89] Letter of November 18, 2013 from Pamela M. Vacek, Ph.D. and Peter W. Callas, Ph.D. to William Perry. Docket Item # OSHA-2010-0034-1804.

> problems at the high and low ends of the quintile divisions would be more than sufficient to suppress a linear trend from being observed.[90]

As Dr. Cox observes:

> It is revealing that OSHA interprets as "problems," rather than as neutral empirical facts, data that do not support the hypothesis of a positive linear ER relation . . . . OSHA's argument that the real patterns observed in the data are "more than sufficient to suppress a linear trend from being observed" suggests a conviction that OSHA's hypothesis of a linear trend should be preferred to real data that tend to refute it, and that a study yielding such data should be excluded precisely because it does not support the hypothesis. This perfectly illustrates study selection bias.[91]

Why does OSHA assume that the results of the Vacek *et al.* study amount to evidence of uncontrolled confounding? The Vermont granite worker cohort, after all, supposedly is free of confounding exposures.[92] In the words of Attfield and Costello, it consists of "workers exposed almost exclusively to rock dust containing silica and no other major occupational confounding exposures."[93] So if there is uncontrolled confounding, what is the confounding factor? OSHA suggests none. Once again, to quote Dr. Cox:

> there is no clear basis for interpreting these patterns in the data as "signs of uncontrolled confounding," and OSHA does not suggest what any such confounder might be. No list of criteria for including or excluding data was published in advance stating that deviations from the hypothesis of a linear ER association would be rejected as "signs of uncontrolled confounding." This appears to be an *ad hoc, ex post* reason to reject data that do not confirm OSHA's prior beliefs.[94]

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[90] Supplemental Health Effects Review at 3.

[91] Cox Comments at 22.

[92] See Health Effects Review at 76.

[93] Attfield, M.D. & Costello, J. (2004). Quantitative exposure-response for silica dust and lung cancer in Vermont granite workers. Am J Ind Med 45:129–138 at 137. OSHA-2010-0034-0543.

[94] Cox Comments at 23.

Rather than speculating that these findings may be attributable to uncontrolled confounding by an unidentified agent, it is far more reasonable to conclude, as the study's authors do, that there is no evidence of an exposure-response trend for silica exposure and lung cancer in this cohort. Vacek *et al*. reported what the data showed without any *post hoc* exclusions. Since OSHA apparently does not like the results, it says there are "problems" with the data and suggests there must be something that the authors missed (though OSHA does not say what that might be). This is a prime example of "confirmation bias."[95] At the same time – presumably because it likes the results – OSHA embraces the Attfield and Costello study, where the authors discarded some of the data by excluding the highest exposure group from the analysis after they found there was no significant E-R trend for lung cancer when that group was included. This does not appear to be an objective assessment. To the contrary, OSHA's treatment of the two Vermont granite studies by Attfield and Costello (2004) and by Vacek *et al.* (2011) perfectly illustrates what Dr. Cox refers to as "study selection bias," "data selection bias," and "confirmation bias."[96] Biases of this sort "should be formally assessed and quantified,"[97] but OSHA has made no attempt to do so.

OSHA then says (or at least implies) that it was right for Attfield and Costello to exclude the highest exposure group (with 30 deaths) in their study and wrong for Vacek *et al*. to include the highest exposure group (with 51 lung cancer deaths) in their study. But why is it right to exclude data when it produces a result the authors don't like and wrong to include all the data without regard to what the result will be? This is counter-intuitive. The burden

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95 Cox Comments at 55.

96 See *id.* at 22-23, 25-26, 55, 79, 80.

97 *Id.* at 58.

should be on those authors who exclude data to justify their action – and not simply by speculating on why the excluded data do not produce the result that they expected.[98] "At a minimum," as Dr. Cox notes, "the reported finding of a "significant" association in the Attfield and Costello study should have been adjusted for bias arising from their willingness to drop high-exposure data to create such an association. Regardless of whether their speculations are correct about why there might be a relation when high-exposure data are excluded, the fact that the same procedure can also create the appearance of a positive association where none exists requires modification of the usual statistical rules for calling a reported association 'significant' to reflect possible biases arising from subset selection."[99]

In short, OSHA's discussion of the exclusion (in Attfield and Costello) and the non-exclusion (in Vacek *et al.*) of the highest exposure group "illustrates confirmation bias in action. Authors should not reject data based on personal opinions or beliefs about whether it supports their preconceptions"; that, however, appears to be precisely what was done in Attfield and Costello (2004).[100] Furthermore, inclusion of the high exposure group in Vacek *et al.*'s analyses did not mask a significant E-R trend for lung cancer. As Dr. Vacek explained:

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[98] Moreover, the speculation that competing causes of death may have clouded the exposure-response relation at high cumulative exposure levels (see Supplemental Health Effects Review at 3) seems inconsistent with the fact that Attfield and Costello's calculation of excess risks was adjusted for competing causes. Cf. Peer review comment of Kenny Crump in External Peer Review of OSHA's Draft "OSHA Preliminary Health Effects Section for Silica" and "Preliminary Quantitative Risk Assessment for Silica" Peer Review Comments, Silica Docket Item OSHA-2010-0034-1716 (hereinafter "Peer Review Comments") at 171.

[99] Cox Comments at 27.

[100] *Id.* at 80, 89-90.

> Attfield excluded workers with exposures at or above 6.0 mg-yr/m^3, based on the assumption that exposure data for that group is weakest. (It appears that Attfield may have excluded person-years, not workers, which would be inappropriate because workers with "weak" exposure data contributed person-years to lower exposure categories before their cumulative exposure reached the cut-point for the highest category). We used all available data because post hoc data selection inflates type II error. However, we performed secondary analyses to explore potential non-linear exposure-response relationships. These included logarithmic transformation of cumulative exposure, as well as fitting polynomial regression models and spline functions. The logarithmic transformation yielded an even stronger relationship with silicosis, but we did not observe significant non-linear relationships with cumulative exposure for any of the other diseases. If the absence of a significant exposure-response relationship for lung cancer were due to lower risk among men with high exposures, this would have been evident in the polynomial regression and spline analyses. We also performed sensitivity analysis to examine the impact of potential errors in the exposure estimates for some jobs prior to 1940, which could have been overestimated. Modifications to the estimates had very little effect on the results.[101]

OSHA goes on to contend that Vacek *et al.*'s SMR for lung cancer is understated because they did not adjust it upward to account for a healthy worker effect (HWE). But SMRs typically are not adjusted to account for a potential HWE, and OSHA does not criticize other studies for failing to adjust for a potential HWE. In any event, the possibility of a potential HWE in this cohort could not have affected the E-R analyses, which were not based on an external reference population. Instead, they were based on an internal case-control analysis – where there could be no HWE, and where no E-R trend for lung cancer and silica exposure was found.

Finally, OSHA suggests that the lack of complete smoking data for the cohort is a problem and contends that smoking could not explain the elevated SMR for lung cancer. This criticism, as Dr. Vacek explains, is overstated, and, in any event, does not detract from

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[101] Letter of November 18, 2013 from Pamela M. Vacek, Ph.D. and Peter W. Callas, Ph.D. to William Perry. Docket Item # OSHA-2010-0034-1804 (footnote omitted).

the study's findings regarding the absence of an association between silica exposure and lung cancer. As Dr. Vacek points out, the lack of smoking data:

> is not unique to our study. Adequate smoking data was also lacking in all previous mortality studies of the Vermont granite industry, including Attfield's, as well as many of the other epidemiologic studies that were considered useful for quantitative risk assessment. In the discussion section of our paper, we mentioned that smoking prevalence was 50% among the 1457 workers in our cohort who were part of a pulmonary function study conducted between 1979 and 1985. The review stated that this may be an overestimate because of selection bias and that comparison to men in the general U.S. population (smoking prevalence of 36% in 1980) should have been adjusted for age because of the "aging granite cohort". Neither of these comments is valid because the smoking data from the pulmonary function study included almost all workers employed at the time, not just those who agreed to have a pulmonary function test, so there is no basis for presuming a selection bias. Also, the men (average age 45 years) were unlikely to be older than the U.S. adult male population of the time because the latter included retirees, and the smoking prevalence we reported is consistent with NHIS results for similar occupations in 1978-1980. In any case, this was only a discussion point in the paper and has nothing to do with the integrity of our study.[102]

As shown above, OSHA's criticisms of the Vacek *et al.* (2011) study are unfounded. Its rejection of that study in favor of Attfield and Costello (2004), which the Vacek *et al.* study clearly supersedes, is unfounded and raises serious questions about the Agency's objectivity. In Dr. Cox's words, "using Attfield and Costello (2004) in preference to Vacek et al. (2011) . . . appear[s] to reflect OSHA's own study selection, data selection, and confirmation biases."[103] The Attfield and Costello study should not be used for risk assessment either on an independent basis or as part of a pooled analysis.

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[102] Letter of November 18, 2013 from Pamela M. Vacek, Ph.D. and Peter W. Callas, Ph.D. to William Perry. Docket Item # OSHA-2010-0034-1804 (footnote references omitted).

[103] See Cox Comments at 55.

In sum, both at the time of the IARC Working Group's narrowly divided vote in late 1996 and in subsequent years, the hypothesis that crystalline silica exposure is causally associated with increased risk of lung cancer was – and has remained – controversial and unsettled. Epidemiological studies have been negative as often as they have been positive; exposure-response trends have generally been absent even in the studies that appeared to be positive; and the effects of confounding factors such as smoking or other occupational exposures and/or the necessity of a mediating silicotic response cannot be ruled out where increased risks have been found. The result, as M. Sogl and colleagues recently noted, is that "the carcinogenic role of silica in the absence of silicosis is still debated" – because studies restricted to non-silicotics or those with 'unknown silicotic status' mainly show no increased risk of lung cancer."[104]

A recent study of Chinese pottery workers and miners by Liu *et al.* (2013) purports to show that silica exposures cause lung cancer in the absence of silicosis,[105] but – for a number of reasons – it is premature to draw that conclusion from that study. Liu *et al.* investigated a cohort of 34,018 Chinese pottery workers and miners who the authors state were without exposure to carcinogenic confounders – notably, radon, polycyclic aromatic hydrocarbons, and arsenic. Based on that belief, Liu *et al.* (2013) did not adjust for any occupational confounders in their analyses. Their cohort included 6 tungsten mines, 1 iron mine and 4 potteries. This was a sub-cohort of a larger cohort of 29 Chinese mines and potteries that had

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[104] Sogl, M. *et al.*, Quantitative relationship between silica exposure and lung cancer mortality in German uranium miners, 1946 - 2003. British Journal of Cancer. 2012; 107, 1188–1194.

[105] Liu, Y., Steenland, K., Rong, Y., 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. Am J Epidemiol. 2013;178:1424-1433.

been studied on several earlier occasions, including by Chen *et al.* (2007).[106] The particular mines and potteries chosen by Liu *et al.* were selected because smoking information was available for workers at these locations. While that may well have been the basis for narrowing the cohort, it is far from clear that there were no confounding exposures at the selected locations.

In particular, Chen *et al.* (2007) stated that the Chinese pottery workers were exposed to high levels of polycyclic aromatic hydrocarbons (PAHs) and that some of the iron-copper miners had relatively high exposure to PAHs and radon daughters. Without adjusting for PAH exposures, Chen *et al.* (2007) found an association between respirable silica and lung cancer mortality, as Liu *et al.* did in their study. After adjusting for PAH exposures, however, this association no longer was observed. Instead, they found a strong association between lung cancer mortality and cumulative exposure to carcinogenic PAHs. Liu *et al.* (2013) claim there were no confounding exposures in their cohort, so they made no adjustment for PAH exposures in the 4 potteries they studied or for PAH exposures and radon in the iron mine. As Dr. Peter Morfeld observes: "It is unclear whether restricting the analysis to the 4 potteries and one iron mine considered by Liu et al 2013 makes a difference in terms of PAH (and possible radon) exposures, and Liu et al do not explain the point." [107] While they assert that they "minimized possible carcinogenic confounders by excluding

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[106] Chen, W., F. Bochmann & Y. Sun (2007). Effects of work related confounders on the association between silica exposure and lung cancer: a nested case-control study among Chinese miners and pottery workers. Int. Arch Occup Environ Health. 80:320-326.

[107] Morfeld Comment at 15. See also Cox Comments at 34-35 ("Why they believe that workers in metal mines and pottery factories were not exposed to any other important carcinogenic confounders is not documented – and, indeed, it runs counter to the findings of an earlier study of the full cohort, where PAH exposures were found to be a major confounder Chen et al. (2007))").

those who worked in tin or copper mines," that hardly rules out possible confounding by PAHs in the potteries, which Chen *et al.* (2007) found made all the difference as far as an association between silica exposure and lung cancer was concerned. Moreover, Chen *et al.* (2007) found no increased lung cancer risk among tungsten miners, while Liu *et al.* apparently did. It is not clear what accounts for the discrepancy. In Dr. Morfeld's words: "Unless and until these issues are resolved, Liu et al (2013) should not be used to draw conclusions regarding exposure-response relationships between RCS, silicosis and lung cancer risk" [108] – and, in particular, should not be cited as having established that silica exposure causes lung cancer in the absence of silicosis.[109]

There are modeling shortcomings in the Liu *et al.* (2013) study as well. Thus, as Dr. Cox observes:

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[108] See Morfeld Comment at 15. An additional concern is that "the exposure estimates used [by Liu *et al.* (2013)] seem to rely on the 'NIOSH approach' (Chen et al 2001, Zhuang et al 2001) although this approach has been shown to be unreliable (Dahmann et al 2008b) and was already replaced by an updated assessment (Yang et al 2012) before the Liu paper was published." *Id.* at 16.

[109] Even if the questions discussed in text regarding Liu *et al.* (2013) did not exist, it is doubtful that the study would establish a causal association between silica exposure and lung cancer in the absence of silicosis. As explained by B. Miller and L. MacCalman: "In the first place, all pneumoconioses, including silicosis, are gradual damage processes characterised by the formation of fibrotic nodules within the lung tissue. These are usually established by examination of a chest radiograph, when classification of a radiograph as showing 'silicosis' (or acceptance to a register of silicotics) implies that the film shows at least a certain profusion of opacities. Failure to reach this level does not necessarily imply that no silicotic-type opacities are present, simply that they are not sufficiently numerous to justify a classification. And none of this excludes the possibility that the exposed lung may show fibrotic changes at autopsy that were not visible on a radiograph. Apart from these difficulties in defining 'silicosis', there are the added time-related problems that radiographs are not taken regularly through life, and that it is not possible to be certain about when the carcinogenic process begins that will ultimately lead to a diagnosis of or death from lung cancer." Miller, B.G. & MacCalman. L. (2010). Cause-specific mortality in British coal workers and exposure to respirable dust and quartz. Occup Environ Med. 67:270-276 at 275.

> the authors "use a Cox proportional hazards model, but do not show that its assumptions are met. For example, the model assumes that the relative risk of cancer is independent of age – an assumption not assessed for realism or shown to be valid. Nor do the authors correct for uncertainties in estimated exposures using appropriate statistical techniques (such as SIMEX, regression calibration, multiple imputation etc., see Bang et al., 2013). Thus, their model is not appropriate for describing the data, which contains important uncertainties about individual exposures.[110]

Similarly, Liu *et al.* (2013) ignored exposure uncertainty. They "select[ed] a single exposure-response model . . . and then ignore[d] all uncertainty about it, including the fact that even the best-fitting model (by any criterion) is almost certainly wrong (Viallefont et al., 2001)."[111] And, in order to produce the monotonically increasing results, they made "an arbitrary logarithmic transformation of the estimated exposures," which "changes the qualitative nature of the estimated relation" from non-monotonic to monotonic.[112] But "picking a transformation [that] allows any desired result to be produced . . . does not provide a sound, objective basis for risk conclusions that are dictated by external reality rather than by the modeler's choices."[113]

In short, as pointed out by Environment Canada and Health Canada in a recent assessment of potential health risks associated with exposure to quartz and cristobalite: "At this time, within the epidemiology literature there is debate on whether human workplace exposure to silica which does not cause silicosis can be associated with lung cancer. . . . Thus, the question of whether silica exposure, in the absence of silicotic response, results in

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110 Cox Comments at 34.

111 See Cox Comments at 35.

112 See *id.* at 36-37.

113 *Id.* at 37.

lung tumours remains unanswered."[114] But, while the question is still open, "a silica-related lung cancer risk," as Dr. Peter Morfeld explains, "appears to be restricted to subjects who contracted silicosis,"[115] a view which the British Health and Safety Executive has expressed as well: "Where evidence is available concerning the relationship between lung cancer and silicosis, it tends to show that excess lung cancer mortality in RCS-exposed workers is restricted to those with silicosis…."[116]

Furthermore, even if high level silica exposures can increase lung cancer risk in the absence of silicosis (which has yet to be shown), it is highly unlikely, as discussed in section 2. below, that low level silica exposures (in the neighborhood of 100 $\mu g/m^3$ and below) cause a significantly increased risk of lung cancer.

2. Like Other Silica-Related Respiratory Effects, Any Risk of Silica-Related Lung Cancer that May Exist Most Likely Has a Threshold Above 100 $\mu g/m^3$.

If it exists at all, silica-related carcinogenicity most likely arises through a silicosis pathway or some other inflammation-mediated mechanism, rather than by means of a direct genotoxic effect.[117] As the British Health and Safety Executive observes, "silicosis and lung cancer are both likely to stem from a common background of chronic inflammatory lung damage"[118] That, in turn, implies that there is "a threshold for any causal association

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114 Environment Canada and Health Canada, Screening Assessment for the Challenge: Quartz and Cristobalite, June 2013 at 52.

115 Morfeld Comment at 7.

116 British HSE Phase 2 Report at 5.

117 See Morfeld Comment at 5-6.

118 British HSE Phase 2 Report at 16.

between silica exposure and risk of lung cancer."[119] As noted above, crystalline silica has been found to cause lung cancer in only one animal species, the rat (which is the most sensitive species for increased lung cancer risk from inhaled particles), and exposure thresholds for increased lung cancer risk have been described in rats for multiple types of particles for over a decade. Crystalline silica is typical in this regard. Mechanistic studies and *in vitro* as well as *in vivo* data exhibit strong concordance in demonstrating that even the earliest changes, such as lung inflammation, exhibit dose-response thresholds for low-toxicity, low-solubility particles.[120]

Mechanistic studies are most consistent with the existence of an inflammation-mediated pathway in which the production of reactive oxygen species (ROS) and the release of TNF-α by alveolar macrophages participate in causing sustained lung injury – although other factors (such as unmodeled exposure misclassification and estimation errors) also must be considered to fully explain the conflicting findings from different epidemiological investigations.[121] These points are elucidated at greater length from both biochemical and mathematical perspectives in a paper by Dr. Cox entitled *An Exposure-Response Threshold*

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[119] Morfeld Comment at 6.

[120] See Donaldson, K, Borm, PJ, Oberdorster, G, Pinkerton, KE, Stone, V, Tran, CL. Concordance between in vitro and in vivo dosimetry in the proinflammatory effects of low-toxicity, low-solubility particles: the key role of the proximal alveolar region. Inhal Toxicol. 2008; 20(1): 53-62; Borm, P., *et al.*, The carcinogenic action of crystalline silica: A review of the evidence supporting secondary inflammation-driven genotoxicity as a principal mechanism. Critical Reviews in Toxicology. 2011; 41(9): 756-770.

[121] Cocco, P, Dosemeci, M, Rice, C. Lung cancer among silica-exposed workers: the quest for truth between chance and necessity. Med Lav. 2007; 98(1):3-17.

for Lung Diseases and Lung Cancer Caused by Crystalline Silica.[122] In this paper, Dr. Cox describes an inflammatory mode of action, having substantial empirical support, in which exposure increases alveolar macrophages and neutrophils in the alveolar epithelium, leading to increased reactive oxygen species (ROS) and reactive nitrogen species (RNS), pro-inflammatory mediators such as TNF-alpha, and eventual damage to lung tissue and epithelial hyperplasia, resulting in fibrosis and increased lung cancer risk among silicotics. This view of the likely mechanism for silica-related lung cancer is widely accepted in the scientific community,[123] including by OSHA's primary source of silica-related health risk estimates, Dr. Kyle Steenland.[124] OSHA appears to share this view as well.[125] A recent study showing that treatment with suppressive oligonucleotides can inhibit pulmonary

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[122] Cox, L.A. Jr., An Exposure-Response Threshold for Lung Diseases and Lung Cancer Caused by Crystalline Silica. Risk Analysis. 2011; 31(10):1543-1560. Available on-line at http://dx.doi.org/10.1111/j.1539-6924.2011.01610.x. A more detailed mathematical treatment of this subject can be found in Cox, L.A. Jr., Dose-Response Thresholds For Progressive Diseases. Dose-Response. 2012; 10(2): 233-250. Available on-line at http://dx.doi.org/10.2203/dose-response.11-039.Cox.

[123] See Morfeld Comments at 6. See also British HSE Phase 2 Report at 78. ("Recent research has shown that the process of inflammation may cause genotoxicity as a result of increased production of oxidant species leading to oxidative DNA damage. It has been demonstrated that the extent of genotoxicity, in terms of gene mutation at the HPRT locus or production of 8-OHdG, is directly related to the severity of inflammation, particularly the number of neutrophils, present in the lung. It therefore seems most likely that RCS is not a direct-acting genotoxicant. However, in some circumstances it could lead indirectly to genotoxicity as a secondary consequence of inflammation.").

[124] See Steenland, K. & Ward, E. Silica: A Lung Carcinogen. CA CANCER J CLIN 2013;00:00–00. Available on-line at http://dx.doi.org/10.3322/caac.21214 (first published on-line December 10, 2013) ("Both silicosis and lung cancer are believed to result from the strong inflammatory response that silica evokes in the lung.").

[125] See 78 Fed. Reg. at 56310.

fibrosis and other inflammatory manifestations of chronic silicosis and reduce the incidence and multiplicity of lung tumors in silicotic mice further buttresses this view.[126]

This mode of action, Dr. Cox explains, involves several positive feedback loops. Exposures that increase the gain factors around such loops can create a disease state with elevated levels of ROS, TNF-alpha, TGF-beta, alveolar macrophages, and neutrophils. This mechanism implies a "tipping point" threshold for the relation between crystalline silica exposure and the risk of lung pathologies such as chronic inflammation, silicosis, fibrosis, and (to the extent it is silica-related) lung cancer.[127] And that threshold, Dr. Cox explains, appears to be above 0.1 mg/m^3; indeed, it may well be considerably higher.[128] OSHA contends that this conclusion "is not supported by the evidence presented."[129] But, as discussed immediately below and in the discussion of a threshold for silicosis at pages 90-102, *infra*, there is considerable evidence of a threshold above 100 $\mu g/m^3$ for the respiratory effects of silica exposure.

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126 See Bode, C. *et al.*, Suppressive Oligodeoxynucleotides Reduce Lung Cancer Susceptibility in Mice with Silicosis. Carcinogenesis (2014). Available on-line at http://carcin.oxfordjournals.org/content/early/2014/01/07/carcin.bgu005. First published online: January 8, 2014.

127 See Cox, L.A. Jr., An Exposure-Response Threshold for Lung Diseases and Lung Cancer Caused by Crystalline Silica. Risk Analysis. 2011; 31(10):1543-1560. Available on-line at http://dx.doi.org/10.1111/j.1539-6924.2011.01610.x. The British Health and Safety Executive has expressed a similar view, stating: "The pattern of evidence [of inflammation-mediated genotoxicity] is consistent with the concept of a threshold related to the severity of inflammation." British HSE Phase 2 Report at 78.

128 See Cox, L.A. Jr., An Exposure-Response Threshold for Lung Diseases and Lung Cancer Caused by Crystalline Silica. Risk Analysis. 2011; 31(10):1543-1560. Available on-line at http://dx.doi.org/10.1111/j.1539-6924.2011.01610.x.

129 Supplemental Health Effects Review at 37.

In addition to mechanistic understandings of silica-related carcinogenicity, epidemiological studies suggest the existence of a threshold for any increased risk of silica-related lung cancer. Thus, in a detailed exposure-response study of silica and lung cancer in a cohort of 58,677 German uranium miners followed up from 1946-2003 (providing almost 2 million person-years of follow-up and employing extensive side-by-side measurements using original historic equipment to assess exposures), M. Sogl and co-workers found no increased risk of lung cancer among workers whose cumulative silica exposure was less than 10 mg/m^3-years.[130] That is equivalent to 40 years' exposure to a silica concentration of 0.25 mg/m^3 or 2½ times the current general industry PEL for quartz and 5 times the proposed PEL. Similarly, in an analysis of the German porcelain industry cohort, K. Mundt *et al.* found that exposure to respirable silica was not associated with increased mortality from lung cancer, kidney cancer, or any other cause of death (except silicosis), even when cumulative silica exposures exceeded 4 mg/m^3-years, thereby suggesting the existence of an exposure threshold at or above that level.[131] And in an investigation of alternative exposure metrics

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[130] Sogl, M. *et al.*, Quantitative relationship between silica exposure and lung cancer mortality in German uranium miners, 1946 - 2003. British Journal of Cancer. 2012; 107, 1188–1194. In this study, the authors were able to develop individual information on occupational exposure to crystalline silica in mg/m^3-years and the potential confounders radon and arsenic based on a detailed job-exposure matrix. See Dahmann, D. *et al.*, Retrospective exposure assessment for respirable and inhalable dust, crystalline silica and arsenic in the former German uranium mines of SAG/SDAG Wismut.. Int Arch Occup Environ Health. 2008:81(8):949-958. The average follow-up period in this study was 34 years, and the mean duration of employment was 14 years. While the authors did find a statistically significant exposure-response relationship for lung cancer in miners whose cumulative exposures exceeded 10 mg/m^3-years, they possessed only limited data on silicotics in the cohort – so they could not rule out the possibility that the increased risk at these very high exposure levels may have been limited to silicotics.

[131] Mundt, K. *et al.*, Respirable Crystalline Silica Exposure-Response Evaluation of Silicosis Morbidity and Lung Cancer Mortality in the German Porcelain Industry Cohort. JOEM 2011; 53(3): 282-289. OSHA questions this suggestion of a threshold, arguing that

and analytical methods used in studies applying job-exposure matrices to assess the association between crystalline silica and cancer, E. Pukkala and co-workers found that the excess risk of lung cancer was mainly attributable to workers in occupations with an estimated cumulative exposure exceeding 10 mg/m^3-years or a threshold exposure of at least 0.2 mg/m^3.[132]

Results suggesting the existence of a threshold for silica-related lung cancer also were found in a NIOSH-sponsored case-control study by G. Calvert, *et al.* (2003) in which cases were subjects whose death certificate mentioned a postulated silica-related disease, and controls were subjects whose death certificate did not mention the disease.[133] Subjects were assigned to a qualitative silica exposure category (low, medium, high, or super high) based on the industry/occupation pairing shown on the death certificate. While there was a significantly increased risk of lung cancer among those postulated to have had the highest silica exposure (judged to be 5 times greater than the current PEL), there was no increased lung cancer risk when the combined results for the medium, high, and super high estimated exposure categories were compared to the low/no exposure category. And those judged to have silica exposures below the PEL showed a mortality odds ratio for lung cancer of only

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"no formal threshold analysis was conducted in this study." See Supplemental Health Effects Review at 11. As discussed below, a formal threshold analysis of the German porcelain worker cohort subsequently was conducted and found distinct evidence of a threshold above 100 μg/m^3. See pp. 98-99, *infra.*

[132] Pukkala, E. *et al.*, National job-exposure matrix in analyses of census-based estimates of occupational cancer risk. Scand J Work Environ Health. 2005; 31(2): 97–107. Available on-line at http://dx.doi.org/10.5271/sjweh.856.

[133] Calvert, G.M., *et al.*, Occupational silica exposure and risk of various diseases: an analysis using death certificates from 27 states of the United States. Occup. Environ. Med. 2003; 60:122-129.

0.88 (C.L. 0.87-0.90). Considered as a whole, this study suggests a threshold for silica-related lung cancer above 100 μg/m^3. Similarly, in a study of lung cancer mortality in a cohort of diatomaceous earth workers, Checkoway *et al.* (1997) found that "[e]xcess risk was predominantly concentrated in the highest cumulative exposure stratum of either respirable dust or respirable crystalline silica [where cumulative exposure to RCS exceeded 5 mg/m^3-years]."[134] And, as the British Health and Safety Executive notes, in the studies of North American industrial sand workers and California diatomaceous earth workers, "[t]he relative risks were generally only statistically significantly increased in the highest exposure categories, which is consistent with other studies showing that relative heavy and prolonged exposure is required for RCS to cause lung cancer."[135]

The pooled analysis of 10 studies by Steenland *et al.* (2001) also suggests the existence of a threshold above 100 μg/m^3 for increased risk of silica-related lung cancer. Thus, the best-fitting model considered in this analysis was a spline model (shown in Figure 1 of Steenland *et al.*, 2001), which indicates a flat or declining exposure-response relation at levels of cumulative silica exposure below about 4-5 mg/m^3-years.[136] And, when

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[134] Checkoway, H., *et al.* (1997). Dose-response Associations of Silica with Nonmalignant Respiratory Disease and Lung Cancer Mortality in the Diatomaceous Earth Industry. Am J Epidemiol 145:680–688 at 687. OSHA-2010-0034-0326; see also *id.* at 686, Table 6 (showing that the relative risk for lung cancer was significant only in the highest cumulative exposure category). Moreover, the trend for relative risk with increasing exposure was only of borderline significance whether analyzed on an unlagged or a 15-year lagged basis. See British HSE Phase 2 Report at 35.

[135] British HSE Phase 2 Report at 13.

[136] Steenland, K. *et al.*, Pooled exposure-response analyses and risk assessment for lung cancer in 10 cohorts of silica-exposed workers: an IARC multicentre study. Cancer Causes and Control. 2001; 12: 773-784. The authors mistakenly describe this model as showing "a reasonably monotonic increase in risk with increasing cumulative exposure." In fact, however, even a cursory inspection of Figure 1 in Steenland *et al.*, 2001 shows that it

heterogeneity between above-ground and underground exposures is considered, lung cancer excess risks were demonstrated in this pooled analysis only for workers having high (≥ 6 mg/m^3-years) cumulative exposures to RCS.[137] Moreover, the risk estimates in Steenland *et al.*, 2001 "did not take into account the potential upward bias due to smoking" and did not account for "the role of silicosis as a potential intermediate confounder."[138] Thus, as Dr. Peter Morfeld observes, "lung cancer excess risks [in Steenland *et al.* (2001)] were demonstrated only under rather high occupational exposures to RCS dust, and, even then, an upward bias due to smoking and a necessary intermediate role for silicosis could not be ruled out."[139]

In short, as the British Health and Safety Executive notes, "the studies that provide the most convincing evidence of carcinogenicity indicate that increased risks of lung cancer are restricted to those groups with the highest cumulative [silica] exposures, suggesting the

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exhibits a clear threshold (at a cumulative exposure level of about 4-5 mg/m^3-years) below which risk is not increased. Moreover, both the Steenland *et al.* paper and other epidemiological studies have failed to adjust for the effects of uncertainties and errors in exposure estimates in the context of an exposure-response threshold (or threshold-like nonlinearity, such as the one shown in Figure 1 of Steenland *et al.*, 2001). This is a significant failure – because if the *true* exposure-response relation has a threshold but the *estimated* exposure-response relation is fit to data in which some above-threshold exposures are misclassified or misestimated as below-threshold values, then the net effect will be to smear out the true (threshold) relation, giving an estimated exposure-response relation that incorrectly appears to be monotonically increasing even below the true threshold. See Cox, L.A. Jr., An Exposure-Response Threshold for Lung Diseases and Lung Cancer Caused by Crystalline Silica. Risk Analysis. 2011; 31(10):1543-1560. Available on-line at http://dx.doi.org/10.1111/j.1539-6924.2011.01610.x. See also Pukkala, E. *et al.*, *supra,* (noting that in Steenland *et al.* 2001, the relative risk of lung cancer was observed to increase when cumulative exposure exceeded 9 mg/m^3-years).

137 See Morfeld Comment at 9-10.

138 *Id.* at 10.

139 *Id.*

existence of a threshold. The groups with the highest cumulative exposures tend to be the early hire workers who commenced employment before the introduction of adequate dust controls."[140]

These results are consistent with the broader observation noted above that studies supporting an association between *silicosis* and increased lung cancer risk (while not conclusive) are far more compelling than the mixed and inconclusive results of studies evaluating the association of *silica exposure* and lung cancer risk in the absence of silicosis or where silicosis status was unknown. This suggests that the exposure threshold for silicosis may be a threshold for any increased risk of silica-related lung cancer as well. The British Health and Safety Executive put the point this way: "Overall, where evidence is available concerning the relationship between lung cancer and silicosis, it tends to show that excess lung cancer mortality in RCS-exposed workers is restricted to those with silicosis, and the more severe the category of silicosis, the higher the risk of lung cancer. The implication of this is that exposures to RCS insufficient to cause silicosis would be unlikely to lead to a significant increase in the risk of lung cancer over and above background levels."[141] The Minnesota Department of Health recently made a similar observation: "There is . . . a large body of evidence that indicates that lung cancer attributed to silica occurs only after repeated insult leads to silicosis. While some controversy remains, MDH has determined that if

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[140] British HSE Phase 2 Report at 5. See also *id.* at 15.

[141] British HSE Phase 2 Report at 16.

exposure to silica is maintained at levels below those that result in silicosis the likelihood of increased risk of developing lung cancer is minimal."[142]

In their recent assessment of quartz- and cristobalite-related health risks, Environment Canada and Health Canada reached a similar conclusion regarding the appropriateness of applying a threshold approach to assess any potential lung cancer risk associated with exposure to crystalline silica. As the agencies explained:

> Although the mechanism of induction for the lung tumours has not been fully elucidated, there is sufficient supportive mode of action evidence from the data presented to demonstrate that a threshold approach to risk assessment is appropriate based on an understanding of the key events in the pathogenesis of crystalline silica induced lung tumours.
>
> The lines of evidence include the following:
>
> • In experimental studies, all rats that developed tumours also showed fibrosis.
>
> • Adenocarcinomas, the most common type of tumour identified in rats, are commonly associated with fibrosis and deeply scarred lung tissue.
>
> • Experimental rat studies showed a clear progression of the effects from initially mild inflammation, followed by fibrosis over-time, leading eventually to lung tumours.
>
> • Tumours are not present in all treated species dosed in the same way.
>
> • The tumours, both in rats and humans, are concentrated in the lungs only, although other organs are indirectly exposed.
>
> • In human studies, cancer risk is often more significant in workers exposed over a 20-year period or to higher cumulative exposure levels; however a consistent finding is that the onset of silicosis, requires a smaller lag period than that for the appearance of tumours.
>
> • Similarly, cancer risk is often more significantly associated at higher quintiles of exposure compared to the lower quintiles.

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[142] Minnesota Department of Health, Health Risk Assessment Unit, Environmental Health Division, 2013 Health Based Value for Ambient Air for Silica, Crystalline (July 2013).

• Lung cancer rates are higher in workers confirmed to have silicosis versus similarly exposed workers that do not have silicosis.

• The vast majority of the positive genotoxicity assay results can be explained by the generation of reactive oxygen species, as demonstrated experimentally, where ROS scavenging prevents the genotoxicity.

• In vivo, macrophage deficient mice (macrophages produce ROS in response to crystalline silica) do not develop silicosis nor do they develop tumors and the Nalp3 inflammazome, a key factor in the macrophage initiated inflammatory response, is required for the development of pulmonary fibrosis after inhalation of silica.

• Though inhalation exposure to crystalline silica in multiple occupational settings is clear, the increase in risk, based on the several recent meta-analyses of the multiple human epidemiological studies, remains low.[143]

In sum, there is strong evidence for the existence of an exposure threshold for all silica-related respiratory pathologies, including lung cancer, and that threshold appears to be in excess of the current general industry PEL of 100 $\mu g/m^3$. Yet, as Dr. Cox notes, the models used by OSHA and in the studies on which it relies

> assume no thresholds or J-shaped relations, independent of what the data show, and in conflict with biologically-based evidence and the findings of Health Canada and others (Cox, 2011) that a threshold approach to risk assessment is appropriate for crystalline silica because lung cancer (and other inflammation-mediated lung diseases) have an etiology in which positive feedback loops play a prominent role. Such loops (with saturation at high exposures and filtering of noise at low exposures) create a bistable response, with a threshold separating healthy and pathological responses (e.g., Pomerening, 2008).[144]

OSHA itself recognizes that there likely is a threshold for silica-related lung cancer. Thus, in explaining the rationale for the proposed medical surveillance provision, OSHA states: "The proposed requirement that a medical examination be offered at the time of initial assignment is intended to determine if an individual will be able to work in the job involving

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143 Environment Canada and Health Canada, Screening Assessment for the Challenge: Quartz and Cristobalite, June 2013 at 49-50.

144 Cox Comments at 91.

respirable crystalline silica exposure without adverse effects." 78 Fed. Reg. at 56468. This presumes there is a threshold for silica-related health risks, including potential lung cancer; otherwise, one could not work in a silica-exposed job "without adverse effects." Indeed, OSHA explicitly acknowledges that there may very well be a threshold for silica-related lung cancer, but it contends – based on an analysis by Steenland and Deddens (2002) and Kuempel *et al.* (2001) – that any such threshold is likely to be below 0.01 mg/m^3 or 0.036 mg/m^3.[145] As Dr. Cox points out, however, this conclusion ignores the facts "that errors in exposure estimates tend to smooth out, and hence conceal or obscure, J-shaped or threshold ER relations; and that they make any apparent thresholds that survive this smoothing tendency appear to occur at lower concentrations than the true thresholds"[146] – *i.e.*, they "*shift estimated thresholds leftward* (i.e., to lower exposure levels) compared to real thresholds."[147] And, in the studies on which OSHA relies, "exposure uncertainty and the likelihood of significant exposure estimation error and misclassification are very large,"[148] a point that OSHA's own peer reviewers emphasized. Thus, Bruce Allen observed:

> If anything, the weaknesses of all the studies with respect to reconstruction of exposure histories (both with respect to the atmospheric concentrations and the job-specific features that lead to worker exposures to those concentrations) may not have been presented with enough emphasis to convey just how limiting and problematic that process can be. There is some discussion of the

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145 See Health Effects Review at 275, 284.

146 Cox Comments at 46.

147 *Id.* at 42. As Dr. Cox notes, "exposure estimation errors can also shift leftward the apparent point at which an S-shaped, non-threshold dose-response curve hits the x axis, i.e., the point below which risk is not elevated. This remains true even if exposure estimation errors are asymmetric, e.g., larger for older, relatively large exposures, and small or zero for more recent, lower exposures." *Id.* at 42.

148 *Id.* at 40.

> reasonableness of the exposure assessment" on p. 10, where silicosis mortality odds ratios are compared across exposure categories from the pooled cohorts of Steenland et al. The values presented do not give me a very strong sense that exposure misclassification was negligible, since the odds ratios presented hardly differ (the highest three are almost identical) and I am not sure that a formal test would reject the hypothesis that the odds ratios were the same, i.e., would not reject the hypothesis that would hold if all individuals were randomly allocated to the four groups regardless of their cumulative exposure level.[149]

Similarly, peer reviewer Kenny Crump stated:

> A major source of error that apparently was not accounted for is in assuming that the average measure of exposure assigned to a job is the true average. But it is not always clear how representative the underlying measurements were. . . . There is possibly considerable error in such estimates. Another source of uncertainty in the averages stems from the need to convert from one measurement method to another (e.g., from particle counts to gravimetric measurements).[150]

In short, as the British Health and Safety Executive observes, "when trying to quantify the relation between cumulative [silica] exposure and increased risk, it has to be borne in mind that all of the available epidemiological studies suffer from various uncertainties and weaknesses in their exposure assessments. In particular, the early hire workers who have the highest risk of lung cancer were employed in times when dust levels were highest, but for which no reliable exposure data were available."[151] In these circumstances, Dr. Cox points out: "Although the error variance for exposure estimates has not been reliably quantified, it is clearly substantial, thus making OSHA's estimates of

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149 Peer Review Comments, Silica Docket Item OSHA-2010-0034-1716, pp. 151-152.

150 Peer Review Comments, Silica Docket Item OSHA-2010-0034-1716, p. 162.

151 British HSE Phase 2 Report at 15. See also *id.* at 12 (noting that "the underlying exposure assessments are fraught with uncertainty and may only be rough estimates of the true exposures").

thresholds in the ER relation lower than the corresponding true thresholds."[152] In particular, a recent analysis by Morfeld *et al.* (2013) identifies a concentration threshold of 0.25 mg/m^3 (95% CI: 0.15 – 0.30 mg/m^3) as an exposure threshold for silicosis and (given the role of inflammation as the mediating mechanism in both silica-related lung cancer and silicosis) for lung cancer as well.[153] Yet none of the models utilized by OSHA or by the authors of the studies on which it relies allowed for the existence of an exposure threshold for silica-related lung cancer. As Dr. Cox observes: "The biological plausibility of a threshold [which OSHA itself acknowledges] suggests that all of the non-threshold models specified and used in the analyses of Steenland et al. (2001a), Attfield and Costello (2004), and others should be re-done using models that allow a threshold."[154] In the absence of such re-analyses, OSHA's projections of lung cancer risk are not credible.

3. Even If Silica Exposures *per se* Could Increase Lung Cancer Risk at Levels of 100 μg/m^3 and Below, the Data on which OSHA Relies and the Methodology It Has Employed To Assess Exposure-Response Relations Would Produce Unreliable Estimates of Risk.

By OSHA's reckoning, 45 years of occupational exposure to crystalline silica at a level of 100 μg/m^3 will increase the risk of lung cancer by an amount falling within the range

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[152] Cox Comments at 43. In addition, Dr. Cox notes, "OSHA's discussion of Kuempel et al. focuses on thresholds for early, pre-disease responses, such as reduced macrophage clearance and neutrophil infiltration and inflammation, rather than the potentially much higher thresholds for induction of the diseases that it attributes to crystalline silica exposure. The ratio of the exposure concentration needed to cause such normal, healthy responses to inhaled particulate matter to the concentration needed to induce silicosis or lung cancer provides a factor by which the Kuempel estimated threshold of 0.036 mg/m^3 should be increased to make it relevant for the chronic lung diseases that OSHA attributes to crystalline silica." *Id.*

[153] See pp. 98-99, *infra*.

[154] Cox Comments at 82.

of 13/1,000 on the low end (based on the study of British coal miners by Miller and MacCalman (2010)) to 60/1,000 on the high end (based on the study of Vermont granite workers by Attfield and Costello (2004)). See 78 Fed. Reg. at 56333. The study yielding intermediate risk values (22-29/1,000) to which OSHA devotes most attention is the pooled analysis of ten studies by Steenland *et al.* (2001). In fact, none of these studies (or the studies of diatomaceous earth workers and North American industrial sand workers that OSHA also references) shows that silica exposure causes lung cancer in the absence of silicosis. Nor do they contradict the evidence suggesting the existence of a concentration threshold above 100 $\mu g/m^3$ for any increased risk of silica-related lung cancer that may exist. More generally, as discussed below, because of problems with the methodological approach to exposure-response modeling followed in OSHA's Preliminary Quantitative Risk Assessment and in the studies on which it relies, "OSHA has not established that a non-random association exists between crystalline silica exposures at or below the current PEL and the adverse health effects [including lung cancer] on which it bases its determination of significant risk and calculates supposed health effect benefits."[155]

Among these methodological problems are *study selection bias* and *data selection bias*, which have been discussed above in the context of the Vermont granite worker studies. Another methodological problem is the failure to make appropriate adjustments for *model selection bias*. Dr. Cox explains this point as follows.

> The papers and reports relied on by OSHA tried many different combinations of modeling choices, including
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> - *alternative exposure metrics* (e.g., peak, cumulative, average, log-transformed, etc.)
> - *different lags* (e.g., 0 years to 15 years or more)

[155] Cox Comments at 30.

- *alternative model forms* (e.g., log-linear, two-piece linear spline, log square root, log quadratic, power, linear relative rate, shape, additive excess rate, etc.) (pp. 275 and 279); and
- *different subsets of data* to consider (e.g., selectively excluding high exposure data points until a positive relation was generated)

and assessed their effects on the ability to produce "significant"-looking regression coefficients before selecting a single final combination of modeling choices from which to assess ER associations and to predict risks. None of the many choices considered was known to be correct, or to give a close approximation to the true ER relation (if any). This type of multiple testing of hypotheses and multiple comparisons of alternative approaches, followed by selection of a final choice based the outcomes of these multiple attempts, completely invalidates the claimed significance levels and confidence intervals reported for the final ER associations. Trying in multiple ways to find a positive association, and then selecting a combination that succeeds in doing so and reporting it as "significant," while leaving the nominal (reported) statistical significance level of the final selection unchanged (typically at p = 0.05), is a well-known recipe for producing false-positive associations (e.g., Bender and Lange, 2001; Bender et al., 2008; Greenland, 2008). This problem can be avoided by applying appropriate methods of significance level reduction to compensate for multiple testing bias (Bender et al., 2001). However, neither OSHA's assessment nor the key studies that it relies on (e.g., Rice et al. (2001), Steenland et al. (2001a), and Attfield and Costello (2004)) performed the required corrections. Thus, the reported "significant" positive ER associations in these studies are based on invalid significance tests. They are biased toward false-positive results, i.e., declaring that "significant" associations exist even when they do not. It is unclear whether any significant association would exist in the absence of such uncorrected bias. The Preliminary QRA does not answer the question.[156]

A related problem involves *model uncertainty bias*, which Dr. Cox explains in these terms:

OSHA's methodological approach of examining many different models and then picking one (e.g., a "best-fitting" model within some class . . .) on which to base risk calculations and claims of significant positive ER relations, contains another fundamental flaw: it treats the finally selected model as if it were known to be correct, for purposes of calculating confidence intervals and significance levels. But, in reality, there remains great uncertainty about what the true causal relation between exposure and response looks like (if there is one). Ignoring this *model uncertainty* in making risk calculations and significance determinations, as both OSHA and the papers it relies on do,

[156] Cox Comments at 27-28.

> leads to artificially narrow confidence intervals. This biases conclusions toward false-positive findings (since confidence intervals that would include "no effect" if model uncertainty were accounted for are incorrectly narrowed) (e.g., Piegorsch, 2013; Swartz et al., 2001; Viallefont et al., 2001). Methods for overcoming this bias by including multiple possible models in the calculation of results are now widely available (e.g., Piegorsch, 2013 and references therein) but they have not been used in OSHA's Preliminary QRA.[157]

In addition to *study selection bias, data selection bias, model selection bias*, and *model uncertainty bias* (which remain uncontrolled and uncorrected in OSHA's risk assessment and its key supporting studies), it is likely that *model specification bias* has infected OSHA's Preliminary QRA as well. A misspecified statistical model, as Dr. Cox explains,

> can give a statistically significant positive regression coefficient for the estimated ER relation (with a 95% confidence interval lying entirely above zero for absolute excess risk and above 1 for relative risk) for the best-fitting model in the selected class of models, even if the raw data show no relation at all between exposure and risk. . . . OSHA's Preliminary QRA and the key papers it relies on present no regression diagnostics for model specification errors. They offer no biological or other rationales for why the selected models should be considered relevant or appropriate for crystalline silica. Most of the models considered simply assume a monotonic, non-threshold relation between exposure and risk (i.e., no threshold and no J-shaped relation), despite empirical evidence for a threshold relation (e.g., Steenland and Deddens, 2002 and Kuempel et al., 2001, both cited by OSHA and discussed later). Thus, model specification error is likely to have biased the reported findings, since even best-fitting or most-plausible models are highly likely to be misspecified (Maldonado and Greenland, 1993). (For example, if the true ER relation were that risks are positive at exposures much higher than the current PEL, but zero at exposures at and below the current PEL, then the models selected by OSHA would mistakenly estimate a significant positive ER slope even at low exposures.)[158]

In combination, uncontrolled *study selection bias, data selection bias, model selection bias*, *model uncertainty bias*, and *model misspecification bias*

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157 *Id.* at 28-29.

158 *Id.* at 38-39.

> can generate findings of statistically "significant" positive ER associations even in random data, or in data for which there is no true relation between exposure and risk of adverse health responses. Because OSHA's Preliminary QRA and the studies on which it relies did not apply appropriate technical methods . . . to diagnose, avoid, or correct for these sources of false-positive conclusions, the reported findings of "significantly" positive ER associations between crystalline silica exposures at and below the current PEL and adverse outcomes (lung cancer, non-malignant lung disease, renal disease) are not different from what might be expected in the absence of any true ER relations. They therefore provide no evidence for (or against) the hypothesis that a true ER relation exists.[159]

Furthermore, as Dr. Cox points out, OSHA and the authors of the studies on which it relies

> use available data sets both to estimate the parameters of selected statistical models for describing the ER relation, and also to assess the quality of the resulting fitted model (e.g., to estimate the values of goodness-of-fit statistics, widths of confidence intervals, and significance levels of coefficients). However, using the same data to fit a model and to assess the fit leads to biased results: estimated confidence intervals are too narrow (and hence lower confidence limits on estimated ER slopes are too high); estimated significance levels are too small (i.e., significance is exaggerated); and estimated measures of goodness-of-fit overstate how well the model fits the data. This is known in statistics as *over-fitting bias* (e.g., Babyak, 2004; Bilger and Manning, 2013). Appropriate statistical methods have been developed to overcome it (e.g., k-fold cross-validation), but they were not applied to de-bias the risk estimates and confidence intervals in OSHA's Preliminary QRA. Therefore, risk estimates and confidence intervals are likely to contain uncorrected over-fitting bias, and lower confidence limits for ER slopes and predicted risks are likely to be too high.[160]

Another problem, alluded to earlier, is the large "exposure uncertainty and the likelihood of significant exposure estimation error and misclassification" in the studies on which OSHA relies.[161] As Dr. Cox notes, "[t]he effect of exposure misclassification can be dramatic. . . Even . . . [if the biases discussed above have not come into play], the finding of a positive slope at . . . exposure levels [of 100 $\mu g/m^3$ and below] could be due simply to

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159 *Id.* at 29-30.

160 *Id.* at 39-40.

161 Cox Comments at 40.

exposure misclassification" in the neighborhood of a threshold even if there is an exposure threshold above 100 μg/m^3 and even if the true E-R relation is U-shaped or J-shaped.[162] OSHA recognized the importance of exposure estimation errors in quantitative risk assessments and attempted to address the issue in a report prepared by its consultants at ToxaChemica International who conducted a Monte Carlo simulation analysis of errors in individual exposure estimates. But, as Dr. Cox explains, the approach followed by ToxaChemica was "based on inappropriate methods (Monte Carlo simulation) and *ad hoc* models reflecting personal beliefs and unjustified assumptions."[163] As a result, "the likely effects of exposure estimation errors" in the studies relied on by OSHA have not been addressed "using relevant, validated, technically appropriate, or biologically plausible models and methods."[164]

Not only has OSHA failed to properly address the impact of exposure uncertainty and misclassification, it has, more generally, failed to take account of the most important uncertainties in its risk assessment. OSHA does present "uncertainty intervals around best estimates to characterize its uncertainty and confidence about the range of plausible values for estimated risks."[165] But these "do not reveal the major uncertainties in the analyses and results" because "the presented results are implicitly *conditioned* on many uncertain (and some almost certainly false) assumptions (Maldonado and Greenland, 1993), such as selection of specific models. Yet, uncertainties about these assumptions, which drive the

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162 Cox Comments at 41.

163 *Id.* at 46.

164 *Id.*

165 *Id.* at 53.

major uncertainties in results, are not included in OSHA's presentation of uncertainty intervals."[166]

As a result of the foregoing biases and exposure estimation errors, OSHA's risk assessment "has not established that a non-random ER association [between silica and lung cancer] exists at relevant exposure levels [of 100 $\mu g/m^3$ and below]; nor does it correctly quantify the association if it does exist."[167] But, even if OSHA's risk assessment had properly established and quantified a non-random association, it would not show that the association is causal – because, instead of utilizing "the relatively rigorous and objective methods of causal analysis," OSHA indulged in the "fallacy of conflating association and causation."[168] This, as Dr. Cox points out, is an error – because "a positive ER association can always be found . . ., even in the absence of any real causal relation, given the types of uncontrolled modeling biases prevalent in the studies that OSHA has relied on (e.g., study selection bias, data selection bias, model specification error, model selection bias, ignored model uncertainty and model over-fitting bias)."[169] "Formal causal models and methods," Dr. Cox notes, "can readily be applied to the types of data collected in the studies cited by OSHA, but this was not done in the Preliminary QRA, which offers causal conclusions unsupported by proper causal analyses."[170]

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166 *Id.* at 53-54.

167 See *id.* at 47.

168 *Id.*

169 *Id.* at 49.

170 *Id.* at 50.

Finally, to estimate lifetime risks at the various exposure levels for lung cancer and the other mortality endpoints, OSHA "implemented each of the risk models [taken from the various studies] in a life table analysis that accounted for competing causes of death due to background causes and cumulating risk through age 85."[171] But, as Dr. Cox points out:

> Such competing-risk modeling does not correctly quantify cause-specific risks from observational data unless implausible and unverifiable assumptions (such as that all competing risks operate independently of each other) hold (Tsiatis, 1975). It has been known for nearly four decades that "results of a customary method of analysis, based on the assumption that [cause-specific times to death] are independent, may have no resemblance to reality" (*ibid*). To obtain risk estimates (e.g., age-specific, cause-specific hazard rates) that have some resemblance to reality, and that overcome known biases in the naïve life table method used by OSHA (e.g., Andersen et al., 2013), OSHA could have applied modern methods that explicitly consider sub-distribution functions. Examples of such modern methods include Bayesian competing-risks analysis (e.g., Ge and Chen, 2012), expectation-maximization (EM) methods (e.g., Craiu and Reiser, 2006), and copula-based approaches (Escarela G, Carrière, 2003). However, such methods have not been used in OSHA's Preliminary QRA, and the fact that causal effects of interventions cannot be identified in competing-risk models without explicitly modeling dependencies among risk factors appears to be simply ignored.[172]

In sum, the Panel believes that the methodology OSHA used for modeling exposure-response relations and the uncertain data on which the modeling was based result in unreliable estimates of increased lung cancer risks at exposure levels of 100 μg/m^3 and below.

4. <u>Conclusion as to Lung Cancer Risk</u>

In sections II.B. 1-3 above, we showed that (1) the evidence does not support the hypothesis that silica exposure *per se* increases the risk of lung cancer in the absence of silicosis; (2) there very likely is an exposure concentration threshold above 100 μg/m^3 for

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171 Health Effects Review at 269.

172 Cox Comments at 61.

any risk of silica-related lung cancer that may exist; and (3) the data on which OSHA relies and the methodology it has employed to assess exposure-response relations would not produce reliable estimates of risk even if silica could cause lung cancer at exposure levels of 100 $\mu g/m^3$ and below. These points apply to all of the studies that OSHA has used to develop estimates of lung cancer risk.

For the reasons discussed at pages 37-47 above, risk estimates based on Attfield and Costello (2004) – which has been superseded by the updated study of Vermont granite workers by Vacek *et al.* (2011) – have no credibility.[173] So OSHA's high end estimate of risk based on Attfield and Costello – which, with values 2-4 times those of all other studies, is an outlier in any event – should be dismissed out of hand. Of the remaining estimates, that based on Miller and MacCalman (2010) is more credible than the others – because it involved a very large cohort and was of higher quality in terms of design, conduct, and detail of exposure measurements, and because risk "estimates [based on that study] are adjusted for individual smoking histories so any smoking-related lung cancer risk (or smoking – silica interaction) that might possibly be attributed to silica exposure in the other studies will not be reflected in the risk estimates derived from the study of these coalminers."[174] This last point, as Dr. Cox emphasizes, calls into question the claims of a significant silica-lung cancer association in the other studies on which OSHA relies: "The possibility of such misattribution [of smoking effects to silica exposures] undermines the interpretation of

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[173] Cf. *id.* at 89.

[174] Health Effects Review at 288; see also *id.* at 289. OSHA refers to this as Miller and MacCalman (2009), the year it was first published on-line. The paper was subsequently published as Miller, B.G. & MacCalman. L. (2010). Cause-specific mortality in British coal workers and exposure to respirable dust and quartz. Occup Environ Med. 67:270-276.

statistically 'significant' results in other studies. A statistically significant association between estimated silica exposure and lung cancer that is in fact based (in whole or in part) on misattribution of smoking effects to silica does not provide a sound basis for risk assessment of silica effects."[175] For these reasons, Miller and MacCalman (2009) provides the most credible basis for estimating the *associational* relation (though not necessarily a *causal* relation) between silica exposure and lung cancer risk.[176]

Even so, the risk estimates based on Miller and MacCalman are biased upward by the lack of exposure information for cohort members after the mines closed in the mid-1980s. As OSHA recognizes: "Since the lung cancer death ratio was higher during this last study period, 1990 – 2005, this period of time [during which silica exposures of cohort members were unknown and unaccounted for] contributed to the increased lung cancer risk. . . . Not accounting for this exposure, if there were any, would bias the risk estimates upwards."[177] Risk estimates based on Miller and MacCalman also could be biased upward by the unrestricted smoking of cohort members after the closure of the coal mines, which was reflected in a "sharp rise in the lung cancer SMR at the end of follow-up."[178] In addition, Miller and MacCalman (2009) did not adjust significance levels to account "for multiple comparisons bias (here, induced by selection of a lag to increase estimated risks [over what

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175 Cox Comments at 88.

176 See *id.* at 89.

177 Health Effects Review at 289.

178 Miller, B.G. & MacCalman. L. (2010). Cause-specific mortality in British coal workers and exposure to respirable dust and quartz. Occup Environ Med. 67:270-276 at 274. See also Morfeld Comment at 11-12.

was found in the absence of a lag period]).”[179] Furthermore, as pointed out by Dr. Cox, the claim of “significance” for the relative risk estimates in this study is problematic: “The reported confidence intervals are overly narrow because they ignore model uncertainty (Viallefont et al., 2001) and fail to perform multiple imputation of uncertain exposure values (Donders et al., 2006). The conclusion that these confidence intervals ‘showed statistically significant increased risks’ is therefore unjustified.”[180] This same criticism applies to the other studies OSHA uses for lung cancer risk assessment as well because the risk estimates based on those studies also result from multiple comparisons, ignore model uncertainty, and fail to perform multiple imputation of uncertain exposure values.[181] Finally, because of model uncertainty, model selection bias, model specification error, and the failure to look for concentration thresholds, the results of Miller and MacCalman (2009), like the other studies OSHA’s uses for risk assessment, “do not rule out the existence of an exposure threshold above 0.1 mg/m^3,” or j-shaped exposure-response relations that flatten out or exhibit a negative slope at low exposures, or the very real possibility that the increased risk of lung cancer at exposure levels of 100 $\mu g/m^3$ and below is effectively zero.[182]

Lung cancer risk estimates based on the ten cohort pooled analysis by Steenland *et al.* (2001) are problematic for a number of reasons, including the following:

- There is significant heterogeneity in the exposure-response coefficients derived for the individual studies in the pooled analysis. As OSHA notes,

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179 Cox Comments at 87.

180 *Id.*

181 See, *e.g.*, Cox Comments at 85-66 (Hughes *et al.* (2001), 96 (Steenland *et al.* (2002).

182 See *id.* at 88, 89. This is particularly true, since all of these studies are about *statistical associations*, not about *causation*. See *id.* at 87, 88.

"risk estimates based on the coefficients derived from the individual studies for untransformed cumulative exposure varied by almost two orders of magnitude."[183] This significant heterogeneity using either cumulative exposure or average exposure as the metric "suggests that these models [used by Steenland *et al.* (2001] are misspecified for the data, and that a mixture distribution model (or at least a zero-inflated model) (Chu and Nie, 2005) should have been used instead.[184] The difference of two orders of magnitude in the risk assessments "indicates a need for different models that describe and explain the heterogeneity" – because, "contrary to OSHA's modeling assumptions," the "estimated exposures to crystalline silica [clearly] do not account for these differences."[185]

- In all ten cohorts, uncertain exposure estimates were used to model exposure-response relations. Indeed, as Dr. Cox observes: "Uncertainty in the exposure estimates presumably is a major reason why the individual study ER coefficients calculated in Steenland et al. (2001a) vary by two orders of magnitude."[186] OSHA seems to agree: "It may also be that exposure estimates for some cohorts were subject to systematic misclassification errors resulting in under- or over-estimation of exposures due to the use of assumptions and conversion factors that were necessary to estimate mass respirable crystalline silica concentrations from exposure samples analyzed as particle counts or total and respirable dust mass."[187]

- Exposure misclassification in pooled data set of *estimated* exposures used by Steenland *et al.* (2001) could very well have created the appearance of a monotonic exposure-response relationship "even if the true response-vs.-exposure relation is not monotonic (e.g., is J-shaped, or increasing only above a threshold value), due to effects of exposure estimation error." [188] This is a particular problem here because, as Dr. Cox notes,

> there is a virtual certainty of exposure estimation error in the studies on which the Steenland et al. (2001a) pooled analysis was based. For example, the median average exposure to respirable silica was estimated to be more than ten times higher

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183 78 Fed. Reg. at 56330.

184 Cox Comments at 69.

185 *Id.* at 75.

186 See *id.* at 57.

187 78 Fed. Reg. at 56330.

188 See Cox Comments at 67.

> in the Finnish granite worker cohort than in the Vermont granite worker cohort, which seems unlikely to be true for the real (but unknown) exposures. Similarly, given the extensive conversions and extrapolations used to develop exposure estimates for the three Chinese cohorts, exposure estimation errors for those cohorts were unavoidable. And even OSHA and its contractor ToxaChemica International acknowledge the distinct possibility of "biased underestimation of exposure in the South African miner study." (Preliminary QRA p. 308.) OSHA also acknowledges the possibility "that some conversion factors [used in other studies included in the pooled analysis] may have been similarly over or under-estimated, resulting in systematic over or under-estimation of exposure, with the resulting systematic under or over-estimation of the exposure coefficient for a given study." (Preliminary QRA p. 308.)[189]

OSHA makes the same basic point in its Health Effects Review, describing the exposure data used by Steenland *et al.* (2001) as follows:

> The exposure information available from each of the 10 cohort studies varied and included dust measurements representing particle counts, mass of total dust, and respirable dust mass. Measurement methods also changed over time for each of the cohort studies investigated, generally with impinger sampling performed in earlier decades and gravimetric sampling performed later. Exposure data based on analysis for respirable crystalline silica by x-ray diffraction (the current method of choice) were available only from the study of U.S. industrial sand workers. In order to develop cumulative exposure estimates for all cohort members and pool the cohort data, all exposure information was converted to units of mg/m^3 respirable crystalline silica by generating cohort-specific conversion factors based on the silica content of the dust to which workers were exposed and, in some instances, results of side-by-side comparison sampling. Within each cohort, available job- or process-specific information on the silica composition or nature of the dust was used to reconstruct silica exposures of cohort members. Most of the studies did not have exposure measurement data prior to the 1950s; exposures occurring prior to that time were estimated either by assuming such exposures were the same as the earliest recorded for the

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189 *Id.*

> cohort, or by modeling that accounted for documented changes in dust control measures.[190]

For these reasons, OSHA concluded, "uncertainty in the exposure assessments that underlie each of the 10 studies included in the pooled analysis is likely to represent one of the most important sources of uncertainty in the risk estimates."[191] Thus, it is virtually certain that substantial exposure estimation error infused the pooled analysis, resulting in exposure misclassification that would create a false appearance of a monotonically increasing exposure-response even where none exists.

- OSHA commissioned Dr. Kyle Steenland and Dr. Bartell to perform an uncertainty analysis, but their report does not resolve the issues relating to exposure uncertainty – for two reasons: First, by retaining Dr. Steenland "to opine on the technical robustness and soundness of his own studies and conclusions, rather than choosing independent experts in the relevant area of statistics to scrutinize the statistical approach," OSHA "created opportunities for inadvertent investigator bias and confirmation bias."[192] Second,

> Drs. Steenland and Bartell's analysis of effects of exposure uncertainties did not apply any of the specific methods for uncertainty analysis of exposure estimates appropriate for this area of statistics (e.g., PROC CALIS in SAS, simulation extrapolation (SIMEX) in R or STATA, regression calibration, multiple augmentation, etc.) Nor did their uncertainty analysis correctly analyze the interdependencies among uncertainties in exposure estimation errors, causation, and model form specification errors. Instead, their analysis is based entirely on an inappropriate technical method (Gryparis et al., 2009): Monte Carlo simulation, in which multiple randomly generated (but pretended to be error-free) data sets are analyzed, each time assuming that there are no errors or uncertainties in models or exposure estimates, and the ensemble of results is then used to draw conclusions about the effects of errors in exposure estimates in the real world, where model errors and uncertainties, as well as exposure uncertainties, are important. Conclusions based on such Monte Carlo simulations have no known probative value."[193]

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190 Health Effects Review at 270.

191 *Id.* at 292.

192 See Cox Comments at 59.

193 *Id.*

- Steenland *et al.* (2001) tested a large variety of models and lag periods in analyzing the data before settling on the ones that ultimately were presented as the basis for assessing lung cancer risk.[194] This was true of the other studies OSHA relies on to estimate lung cancer risks as well – where multiple lag periods, multiple exposure groupings, multiple model forms, and multiple exposure data formats (log transformed and untransformed) were examined. This results in "multiple-testing bias, arising from trying multiple modeling choices and then selecting and reporting results from a preferred model without appropriately correcting reported significance levels."[195] Furthermore, the fact that the different models applied to the pooled data of Steenland *et al.* (2001) produced wide differences in results "illustrates the importance of model uncertainty. OSHA should not select a few models that it likes best, but should use model ensemble methods, such as Bayesian Model Averaging (Viallefont) and model cross-validation to explicitly incorporate model uncertainty and to determine whether the final risk estimates that OSHA has obtained . . . are robust to model uncertainties."[196]

- Steenland *et al.* (2001) "conducted much of their analysis and calculated their risk estimates using log-transformed cumulative exposure,"[197] as did OSHA in its risk assessment based on that study. As Dr. Cox notes, however:

> There is no legitimate basis for log-transforming *estimated* cumulative exposures, which is what Steenland et al. actually did, without modeling the effect of the log-transform on the error distribution of exposure estimates. Transforming variables in this way may lead to invalid conclusions, since how the log transform affects unmodeled errors is unknown. That it changes the qualitative conclusions about whether different cohorts have significantly different ER relations (e.g., different slopes of the ER curve at the relatively low exposures of interest) is problematic, since the truth about such substantive questions should remain invariant in any legitimate transformation of the data.[198]

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[194] See 78 Fed. Reg. at 56329-56330; Health Effects Review at 272 ("After testing models based on unlagged exposure and exposure lags of 5, 10, 15, and 20 years to account for disease latency, the authors selected an exposure lag of 15 years for most analyses.").

[195] Cox Comments at 63.

[196] *Id.* at 74.

[197] Health Effects Review at 273.

[198] Cox Comments at 70.

- The pooled data analyzed in Steenland *et al.* (2001) are consistent with the existence of a threshold above 100 μg/m^3. OSHA states that in a subsequent "analyses of the data to determine whether there was any empirical evidence of a threshold," Steenland and Deddens (2002) found that "[t]hreshold models using average exposure or untransformed cumulative exposure did not show any statistically significant improvement in fit over models without a threshold" and "that a threshold model based on the log of cumulative dose (15-year lag) fit better than a no threshold model, with the best threshold at 4.8 log mg/m3-days (representing an average exposure of 0.01 mg/m3 over a 45-year working lifetime."[199] But these analyses, as Dr. Cox points out, do not show either the absence of a threshold or that any such threshold is below 100 μg/m^3. Thus, based on their first finding, "one could equally well state that 'non-threshold models using average exposure or untransformed cumulative exposure did not show any statistically significant improvement in fit over models with a threshold.'"[200] And their second finding is another illustration of the fact "that qualitative conclusions about the true ER relation (e.g., is there a threshold?) change, based on the arbitrary transformations selected by the modeler. Thus, the choice of transformation made by the modeler drives the substantive conclusions reached (e.g., threshold vs. no threshold)."[201] Furthermore, their "estimate of a threshold at 4.8 log mg/m^3-days is not supported either, as it does not correctly adjust for the effects of exposure estimation errors in reducing apparent thresholds to artificially low concentrations."[202] In addition, the fact that their identification of a threshold depended on whether they used log-transformed or untransformed exposures "indicates that their analysis is unsound. A plausible source of the problem is the repeated error of treating exposure *estimates* as if they were correct, accurately measured, exposure *values*. An ordinal feature such as a threshold would not change under log-transformation of the exposure axis if correct exposure values were being used, but it does change when exposure estimates are being used instead."[203]

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199 Health Effects Review at 275.

200 Cox Comments at 71.

201 *Id.* at 72.

202 *Id.*

203 *Id.*

For all these reasons, the pooled analysis by Steenland *et al.* (2001) does not yield credible or reliable estimates of silica-related lung cancer risk. But, even if risk estimates based on Steenland *et al.* (2001) were not so problematic, that study would not demonstrate that reducing the PEL from 100 μg/m^3 to 50 μg/m^3 will result in a substantial reduction in the risk of lung cancer. This can be seen clearly when one examines the corrected estimates of excess lung cancer risk for this ten cohort pooled analysis that were prepared for OSHA by Drs. Steenland and Bartell under a contract with ToxaChemica International, Inc. The ToxaChemica report shows that under the spline model (which the authors prefer over the log cumulative model because of biological plausibility), reducing the PEL from 0.1 mg/m^3 to 0.05 mg/m^3 would make a negligible difference in the excess risk of lung cancer. According to their reanalysis, after 45 years of occupational exposure to crystalline silica, the excess risk of lung cancer would be 0.017 (17/1,000) at an exposure level of 0.1 mg/m^3 and 0.016 (16/1,000) at an exposure level of 0.05 mg/m^3, risk values that are indistinguishable given the overlapping confidence limits of the two estimates.[204] Moreover, their analysis shows that the excess risk at exposure levels of 0.15 mg/m^3 and 0.25 mg/m^3 is the same as the excess risk at 0.05 mg/m^3, while the excess risk at an exposure level of 0.20 mg/m^3 is actually *lower* than the excess risk at 0.05 mg/m^3.[205] All of which indicates two things: (1) Reducing the PEL from 100 μg/m^3 to 50 ug/m^3 would make very little difference in the projected lung cancer risk even if there is no exposure threshold above 100 μg/m^3. (2) Estimates of lung

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[204] See Steenland, N.K. & Bartell, S.M. Silica Exposure: Risk Assessment for Lung Cancer, Silicosis and Other Diseases. Prepared under contract to OSHA by ToxaChemica International, Inc. (Draft Final, December 7, 2004) at 20 & 42, Table 9. Docket ID: OSHA-2010-0034-0469.

[205] See *id.* at 42, Table 9.

cancer risk in the neighborhood of the current general industry PEL are hugely uncertain – with the data suggesting that a greater reduction in lung cancer risk could be achieved by doubling the PEL to 200 ug/m^3 than by cutting it in half to a level of 50 ug/m^3.

For various reasons, the study of diatomaceous earth ("DE") workers by Rice *et al.* (2001) does not provide a reliable basis for estimating lung cancer risk at silica exposures of 100 μg/m^3 and below either. For one thing, as discussed at pages 102-104 below, the exposure assessment for the DE worker cohort is subject to large uncertainties, with a high likelihood of exposure misclassification. Indeed, when Seixas *et al.* (1997) compared the semi-quantitative (low, medium, and high) exposure categories that had been derived for use in the earlier studies of the DE cohort by Checkoway *et al.* (1993) and (1996) to the quantitative estimates that they developed for the period 1974-1988, they found a high degree of overlap between the exposure categories, indicating either that there was a high degree of error in the semi-quantitative estimates or a high degree of uncertainty in the quantitative estimates.[206] Moreover, the practice of "[a]ssigning each worker a single estimated cumulative exposure based on estimated mean values [as was done in Rice *et al.* (2001)] produces biased results and artificially narrow confidence intervals (and hence excess false-positive associations) (Donders et al., 2006). Multiple imputation should have been used instead to obtain unbiased results. As it is, uncertainty in exposures is not modeled, creating unnecessary biases in conclusions."[207]

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[206] See Seixas NS, Heyer NJ, Welp EA, and Checkoway H. (1997). Quantification of historical dust exposures in the diatomaceous earth industry. Ann Occup Hyg 41:591–604. OSHA-2010-0034-0431.

[207] Cox Comments at 76.

Furthermore, RCS exposures of DE cohort members were greatly in excess of OSHA's current general industry PEL of 100 μg/m^3. According to the study's authors, the mean RCS exposure in the DE worker cohort was 0.29 mg/m^3, or three times the current PEL for general industry.[208] And exposures in the early years (for which no measurements existed) probably were underestimated, so that the mean RCS exposure for the cohort as a whole presumably was even higher than 0.29 mg/m^3.[209] Moreover, the rate ratio for lung cancer mortality in Checkoway *et al.* (1997) was significant only in the highest cumulative exposure category of ≥5.0 mg/m^3-years.[210] As the British Health and Safety Executive notes: "The excess of lung cancer was limited or most prominent in those who had joined the industry earlier, particularly before 1930, when dust levels were probably at their highest."[211] In short, the results for the DE worker cohort are fully consistent with the existence of a concentration threshold above 100 μg/m^3.

In addition, the results of Rice *et al.* (2001) were subject to confounding by smoking and possibly by asbestos. Indeed, OSHA itself suggests that unaccounted for smoking habits likely produced exaggerated estimates of lung cancer risk in the DE worker cohort, which is one reason OSHA views the study of British coal workers by Miller and MacCalman (2009)

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208 See Health Effects Review at 279.

209 See pp. 102-103, *infra*.

210 See Checkoway, H., *et al.* (1997). Dose-response Associations of Silica with Nonmalignant Respiratory Disease and Lung Cancer Mortality in the Diatomaceous Earth Industry. Am J Epidemiol 145:680–688 at 685. OSHA-2010-0034-0326.

211 British HSE Phase 2 Report at 32.

as providing a more reliable estimate of lung cancer risk.[212] Thus, as OSHA notes: "Sources of uncertainty in the risk estimates based on the Rice et al. (2001) study include possible error in exposure estimates and confounding of the exposure-response analysis due to smoking and occupational co-exposures such as asbestos."[213]

Rice *et al.* (2001) also has modeling problems. As Dr. Cox observes, the risk estimates for the DE cohort developed by the authors and by OSHA reflect "[u]ncorrected multiple-testing bias, arising from trying multiple modeling choices and then selecting and reporting results from a preferred model without appropriately correcting reported significance levels."[214] In Rice *et al.* (2001), this involved consideration of multiple lag periods, multiple exposure groupings, and multiple model forms.[215] Furthermore, model uncertainty was not addressed in the study. As Dr. Cox notes, there was no "biological (or other fundamental) justification" for the model forms that Rice *et al.* (2001) chose to examine; instead, their approach amounted to "an exercise in curve-fitting/data dredging using various *ad hoc* models."[216] And the model forms they chose to examine "make it impossible to find a threshold or non-monotonic exposure-response relation. They impose

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212 See Health Effects Review at 278 (noting that the much lower lung cancer risk estimates derived from the British coalminer study by Miller and MacCalman (2009) compared to risk estimates based on other studies (including the DE worker study) likely reflect the fact that the estimates in Miller and MacCalman "are adjusted for individual smoking histories so any smoking-related lung cancer risk (or smoking – silica interaction) that might possibly be attributed to silica exposure in the other studies will not be reflected in the risk estimates derived from the study of these coalminers.").

213 *Id.* at 280.

214 See Cox Comments at 63.

215 See *id.* at 63-64.

216 See Cox Comments at 77.

unjustified *a priori* constraints on the conclusions that can be reached."[217] Thus, there is a very real possibility of model specification error that "can allow an exposure variable to be identified as a 'significant predictor' of an outcome (e.g., lung cancer mortality) even if there is no true relation between them. No model diagnostics are reported for the Rice (2001) models to allow model specification errors and their effects to be detected or corrected."[218]

The same types of modeling uncertainties and biases apply to the study of North American industrial sand workers by Hughes *et al.* (2001). For example, as Dr. Cox points out, the model used to estimate risk ($ln\ OR = \alpha + \beta\ x\ Cumulative\ Exposure$)

> guarantees a monotonic exposure-response relation, independent of the data. Model uncertainty and errors in exposure estimates have both been ignored, so the slope estimate from Hughes et al. (2001), as well as the resulting excess risk estimates, are likely to be biased and erroneous. . . . [And the life table approach used by OSHA to calculate risk based on this study] makes unverifiable assumptions, such as that cause-specific times to death are independent).[219]

In addition, as the British Health and Safety Executive notes: "One particular weakness [of this cohort] was that company records from these plants were reported to be often incomplete and lacking adequate information on occupational history and smoking habits. The lack of this type of information is particularly important for unskilled and transient workers [many of whom were present in this cohort] who may smoke more than average and may have been exposed to confounding factors in previous or subsequent employment."[220] The limited information on smoking obtained for the nested case-control

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217 *Id.*

218 *Id.*

219 See *id.* at 85.

220 British HSE Phase 2 Report at 38.

study indicated that the proportion of ever smokers was statistically significantly higher in cases (91%) than in controls (69%).[221] There also was some evidence that workers in this cohort had exposure to asbestos, with three death certificates listing mesothelioma as the cause of death.[222]

Furthermore, there were some puzzling observations. For one thing, as OSHA notes: "There was no consistent correlation between duration of employment and lung cancer risk in this cohort,"[223] which, as Dr. Cox points out, "undermines a causal interpretation of employment-related exposures as causes of increased risk of lung cancer."[224] He continues: "The lack of dependence on length of employment suggests that model specification error or other factors unrelated to occupational exposures (and hence to length of employment) may explain the reported associations."[225] For another: "Most of the lung cancer excess [in this cohort] came from four plants in New Jersey and Illinois (SMRs of 1.83 and 2.23, respectively), but these regions had the lowest risk rates for silicosis (0.21 and 0.27, respectively). The authors could not explain this apparent discrepancy. The difference in lung cancer rate cannot be explained in terms of relative levels of dust exposure in each plant as the risk of silicosis and NMRD was substantially higher in those plants with lowest lung cancer mortality."[226]

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221 See *id.* at 40.

222 See *id.* at 38.

223 Health Effects Review at 285.

224 Cox Comments at 83.

225 *Id.* at 86.

226 British HSE Phase 2 Report at 39.

In sum, none of the studies on which OSHA relies is inconsistent with a concentration threshold above 100 μg/m^3 for any risk of silica-related lung cancer; none demonstrates an increased lung cancer risk in the absence of silicosis; and none provides a sound basis for estimating lung cancer risks at RCS exposure levels of 100 μg/m^3 and below.

C. Exposure to Crystalline Silica Has Not Been Shown To Increase the Risk of Non-Respiratory Cancers.

Although many cancer sites in addition to the lung have been examined in epidemiological studies of silica-exposed workers, the evidence does not establish that crystalline silica causes any non-respiratory cancers – and there certainly has been no showing that silica exposure at a level of 100 μg/m^3 significantly increases the risk of non-respiratory cancers. To be sure, in a few studies, silica-exposed workers or silicotics have exhibited increased mortality from certain cancers other than the lung, but, as noted below, the evidence linking non-respiratory cancers to silica exposure is limited and conflicting as well as subject to the influence of confounders.

In a NIOSH-sponsored case-control analysis looking at mortality odds ratios, G. Calvert and coworkers found no significant association between crystalline silica exposure and esophageal cancer or stomach cancer.[227] No association between quartz exposure and

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[227] Calvert, G.M., *et al.*, Occupational silica exposure and risk of various diseases: an analysis using death certificates from 27 states of the United States. Occup. Environ. Med. 2003; 60:122-129. Similarly, in a study of British coal workers, B. Miller and L. MacCalman found no link between stomach cancer mortality and exposure to respirable quartz or respirable dust. See Miller, B. & L. MacCalman, Cause-specific mortality in British coal workers and exposure to respirable dust and quartz. Occup and Environ Medicine. Published online, doi:10.1136/oem.2009.046151, October 9, 2009 (OSHA-2010-0034-1306). In earlier studies, M. Finkelstein *et al.* had found an increased risk of stomach cancer mortality among compensated silicotics in Ontario, but noted discrepant findings for stomach cancer in different studies. Finkelstein, M., *et al.*, Mortality among workers receiving compensation awards for silicosis in Ontario 1940-85. British Jrnl of Industrial

stomach cancer was found in a study of British coal workers by Miller and MacCalman.[228] In a mortality study of 17,644 medical surveillance participants in the German porcelain industry, T. Birk, *et al.* found that mortality from liver and pancreatic cancer was significantly increased in men.[229] However, when K. Mundt *et al.* further analyzed the German porcelain worker cohort using a job exposure matrix, they found that mortality due to liver and pancreatic cancer was not statistically significantly associated with cumulative silica exposure at any level.[230] The same was true of kidney cancer, cardiovascular disease, digestive diseases, diabetes, and renal disease, as well as of all cancers combined.[231]

Medicine. 1987; 44:588-594. By contrast, F. Forastiere *et al.* found a deficit of digestive tract tumors among compensated silicotics in Latium, Italy. Forastierre, F., *et al.*, Mortality pattern of silicotic subjects in the Latium region, Italy. British Jrnl of Industrial Medicine. 1989; 46: 877-880.

I.T.S. Yu, *et al.* found an increased mortality risk of esophageal cancer among registered male silicotics in Hong Kong who had very heavy exposure to free silica dust in underground caissons (frequently several hundred times the TLV). But after adjusting for smoking and alcohol consumption, the SMR was barely significantly elevated among the caisson workers (lower 95% CI=1.01), and there was no elevated risk among non-caisson worker silicotics. Moreover, trends with duration of silica exposure and severity of radiological silicosis were not statistically significant, and other possible confounders, like low socioeconomic status, also existed. Yu, I.T.S., *et al.*, Further evidence for a link between silica dust and esophageal cancer. Int. J. Cancer. 2005; 114: 479–483, available on-line at http://dx.doi.org/10.1002/ijc.20764.

[228] Miller, B.G. & MacCalman, L. (2010). Cause-specific mortality in British coal workers and exposure to respirable dust and quartz. Occup Environ Med 67:270-276.

[229] Birk, T., *et al.*, Mortality in the German Porcelain Industry 1985-2005: First Results of an Epidemiological Cohort Study. JOEM. 2009; 51, No. 3: 373-385.

[230] Mundt, K. *et al.*, Respirable Crystalline Silica Exposure-Response Evaluation of Silicosis Morbidity and Lung Cancer Mortality in the German Porcelain Industry Cohort. JOEM. 2011; 53(3): 282-289.

[231] *Id.* This was consistent with the earlier work by T. Birk, *et al.* who found that death from lung and renal cancers and from non-malignant renal disease was not associated with employment or silica-exposure surrogates in the German porcelain worker cohort. Birk, T.,

The absence of an association between silica exposure and kidney cancer or non-malignant renal disease also was noted by J.C. McDonald, *et al.* in a study of North American industrial sand workers, where the authors found no relation between end-stage renal disease or renal cancer and cumulative silica exposure.[232] In fact, the trends were opposite (*i.e.*, decreasing odds ratios with increasing cumulative exposure) for both diseases. Similarly, in a mortality study of Vermont granite workers, P. Vacek, *et al.* found no significant elevation in the SMRs for kidney cancer and non-malignant kidney disease (nephritis and nephrosis).[233] And, when they conducted a nested case-control analysis, they observed no significant associations between respirable silica exposure (excluding exposures occurring within 10 years of death and, alternatively, including them) and mortality from lung cancer, kidney cancer or non-malignant kidney disease.[234] The authors concluded that their results yielded no indication of an association between silica exposure and either kidney cancer or non-malignant kidney disease.

Finally, there is very limited evidence regarding a possible association between silica exposure or silicosis and increased risk of laryngeal cancer. In a meta-analysis combining SMRs or SIRs from cohort studies and ORs from case-control studies, M. Chen and L.A. Tse found a significantly increased risk of laryngeal cancer among silica-exposed workers in the

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et al., Mortality in the German Porcelain Industry 1985-2005: First Results of an Epidemiological Cohort Study. JOEM. 2009; 51, No. 3: 373-385.

232 McDonald, J.C., *et al.*, Mortality from lung and kidney disease in a cohort of North American industrial sand workers: an update. Ann Occup Hyg. 2005 (Jul); 49(5):367-73.

233 Vacek, P. *et al.*, Mortality in Vermont granite workers and its association with silica exposure. Occup Environ Med. 2011; 68:312-318. available on-line at http://dx.doi.org/10.1136/oem.2009.054452.

234 *Id.*

case-control studies but not in the cohort studies (either when limited to workers with silicosis or when all silica-exposed workers were included).[235] They concluded that their analysis showed a weak association between silicosis or silica exposure and increased risk of laryngeal cancer. Because of inherent limitations of the underlying studies (*e.g.*, failure to adjust for confounders like smoking, alcohol consumption and asbestos), the authors state that the results of their analysis should be interpreted with caution.

In sum, as NIOSH observed in its comprehensive review of silica-related health effects, no conclusion can be reached about a possible association between silica exposure and non-lung cancers.[236] Environment Canada and Health Canada recently reached a similar conclusion, observing that the "results [of epidemiological studies seeking to identify an association between high silica exposures and tumors outside the lungs] are sparse and often inconsistent and have not been unequivocally linked to exposure to either quartz or cristobalite."[237] OSHA also seems to agree with this view. See 78 Fed. Reg. at 56305. Accordingly, speculation about a possible association between crystalline silica and non-respiratory cancers cannot provide a basis for making a finding of significant risk for silica-exposed workers.[238]

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[235] Chen, M. & L.A. Tse, Laryngeal Cancer and Silica Dust Exposure: A Systematic Review and Meta-Analysis. Am. J. Indust. Med. 2012; 55:669-676, available on-line at http://dx.doi.org/10.1002/ajim.22037.

[236] NIOSH Hazard Review: Health Effects of Occupational Exposure to Respirable Crystalline Silica (2002) at vi, 51.

[237] Environment Canada and Health Canada, Screening Assessment for the Challenge: Quartz and Cristobalite, June 2013 at 42.

[238] Environment Canada and Health Canada make this same point, noting that "sufficient epidemiological or toxicological data do not currently exist for quantitative assessment of the exposure-response relationship for these health effects." *Id.* at 42.

D. Workers Will Not Confront a Significant Risk of Silicosis or Other Non-Malignant Respiratory Disease Mortality if There Is Universal Compliance With the Current General Industry PEL of 100 μg/m^3.

OSHA projects that 45 years of occupational exposure to crystalline silica at a level of 100 μg/m^3 will result in 83 excess deaths from non-malignant respiratory disease (including silicosis) per 1,000 workers. See 78 Fed. Reg. at 56333. That projection, as discussed below, is unsupported.

1. The Threshold for Silicosis and All Other Silica-Related Lung Pathologies Is Likely To Be >100 μg/m^3 – So that the Risk of Silicosis at Exposures Below that Level Will Be Negligible, if it Exists at all.

Exposure-response models based on cumulative exposure assume that the internal biologically significant dose (and associated risk of the adverse health effect) is a linear function of both concentration and exposure time – so that, as long as the resulting cumulative exposure is the same, longer exposure at a lower concentration increases risk to the same extent as shorter exposure at a higher concentration. But for silicosis risk, that assumption has been called into question by a number of studies which have found that *intensity* of exposure, *i.e.*, the rate or concentration at which the exposure is received, affects the risk of contracting silicosis. In particular, investigators have found that the risk of silicosis is greater (increases more steeply) when the same cumulative exposure is received in a shorter period of time at a higher intensity than when it results from longer exposure at a lower average concentration.

In the only two studies that looked carefully at this issue, a dose-rate effect was found. J. Hughes *et al.* (1998) found this to be the case in the diatomaceous earth industry, where workers who had a mean crystalline silica exposure of >0.50 mg/m^3 showed a

substantially higher incidence rate and age-adjusted RR for radiological opacities than workers who had a mean crystalline silica exposure of <0.50 mg/m^3, even when their cumulative exposure was the same.[239] In a subsequent analysis of the diatomaceous earth worker cohort, Park *et al.* (2002) found that, when workers having the same cumulative exposures are compared, the silicosis incidence rate in the 1942-1954 period (when silica exposure levels were high) was *13.3 times higher* than in later years (when silica exposure levels were estimated to be considerably lower), thus indicating a dose-rate effect for silicosis in the cohort.[240] Similarly, in a study of Scottish colliery workers, D. Buchanan *et al.* found that cumulative quartz exposure accumulated at higher concentrations resulted in proportionally greater risks of radiological abnormalities than the same cumulative exposure accumulated at lower concentrations, with the differences being quite dramatic at higher intensities.[241]

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[239] Hughes, J. *et al.*, Radiographic Evidence of Silicosis Risk in the Diatomaceous Earth Industry. Am. J. Respir. Crit. Care Med. 1998; 158:807-814. At a cumulative exposure of 4 mg/m^3-years, those having a mean exposure >0.50 mg/m^3 were almost four times as likely to exhibit radiological opacities as those having a mean exposure <0.50 mg/m^3.

[240] Park, R. *et al.* (2002). Exposure to crystalline silica, silicosis, and lung disease other than cancer in diatomaceous earth industry workers: A quantitative risk assessment. Occup Environ Med 59:36–43 at 41. OSHA-2010-0034-0405. See also Peer review comment of Kenny Crump in Peer Review Comments, Silica Docket Item OSHA-2010-0034-1716, at 166-167.

[241] Buchanan, D. *et al.*, Quantitative relations between exposure to respirable quartz and risk of silicosis. Occup Environ Med 2003; 60:159–164 (finding that at comparable levels of cumulative exposure, an average silica concentration above 2 mg/m^3 is three times more effective in producing silicosis than concentrations below that level). See also Buchanan, D. *et al.*, Quantitative Relationships between Exposure to Respirable Quartz and Risk of Silicosis at One Scottish Colliery. Institute of Occupational Medicine Research Report TM/01/ 03; Buchanan, D. *et al.*, Observed and predicted silicosis risks in heavy clay workers. Occupational Medicine. 2007; 57:569-574, available on-line at http://dx.doi.org/10.1093/occmed/kqm080.

By the same token, N. Cherry *et al.* found that in a cohort of Stoke-on-Trent pottery, refractory and sandstone industry workers, cumulative exposure and average concentration were strongly related to presence of small opacities, but duration of exposure was not – suggesting that intensity of exposure was a more significant factor than length of exposure.[242] K. Steenland *et al.* made a similar finding in their pooled analysis of ten cohorts, where they observed no clear trend for silicosis mortality with quintile of duration of exposure, and where models based on duration of exposure did not fit the lung cancer data well – "indicating [to the authors] the importance of incorporating intensity of exposure into the exposure metric."[243] And in a mortality study of Vermont granite workers, P. Vacek *et al.* found that after adjustment for the years worked at other levels of intensity, only employment at high intensities (> 0.20 mg/m^3 for silicosis and > 0.30 mg/m^3 for the category of non-malignant respiratory disease) was significantly associated with increased mortality.[244]

Results such as these have persuaded various investigators to conclude that it is too simplistic to predict silicosis risks based on cumulative exposure alone (or on "average" exposure calculated by dividing cumulative exposure by 40 or 45 years), because "[a]short

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[242] Cherry, N.M. *et al.*, Crystalline silica and risk of lung cancer in the potteries. Occup. Environ. Med. 1998; 55:779-785.

[243] Steenland, K. *et al.*, Pooled exposure-response analyses and risk assessment for lung cancer in 10 cohorts of silica-exposed workers: an IARC multicentre study. Cancer Causes and Control. 2001; 12: 773-784. In a subsequent report to OSHA, Steenland and Bartell noted that "duration of exposure per se (including variants such as lagging or logging duration) was not a significant predictor of lung cancer in this data set." Steenland, N.K. & Bartell, S.M. Silica Exposure: Risk Assessment for Lung Cancer, Silicosis and Other Diseases. Prepared under contract to OSHA by ToxaChemica International, Inc. (Draft Final, December 7, 2004) at 3. Docket ID: OSHA-2010-0034-0469.

[244] Vacek, P. *et al.*, A Study of the Relationship between Mortality and Silica Exposure in the Vermont Granite Industry: Final Report (November 16, 2009). A copy of this report is submitted herewith as Attachment 6.

but high exposure seems to produce a greater risk of developing silicosis than a long but low exposure at a similar cumulative level."[245] And if a dose-rate effect exists for silicosis morbidity, it presumably exists for silicosis mortality as well.[246] Thus, even if they were not subject to criticism in other respects, predictions of silicosis risk based solely on estimates of cumulative exposure would be questionable because they undervalue the role that *intensity* of exposure plays as a factor in silicosis risk. OSHA acknowledges that a dose-rate effect involving intensity of exposure is biologically plausible but says there is little evidence it exists at concentrations in the range of the current PEL.[247] But that largely reflects the nature of the two studies in which the effect was specifically looked for. And, in any event, as OSHA's peer reviewer Kenny Crump points out, even if a dose-rate effect has been established only at higher concentrations, "risks in the range of the current PEL are being estimated from studies that involved higher air concentrations. Not accounting for a dose-rate effect, if one exists, could overestimate risk at lower concentrations."[248]

Predictions of silicosis risk also must be consonant with the existence of cumulative exposure or concentration level thresholds for silica-related respiratory disease. OSHA acknowledges that the studies it relies on to predict the risk of silicosis morbidity at exposure levels of 100 $\mu g/m^3$ and below involved "populations of workers exposed to average

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245 Ulm, K. *et al.*, Silica, silicosis and lung-cancer: results from a cohort study in the stone and quarry industry. Arch Occup Environ Health. 2004; 77:313-318.

246 See Peer review comment of Kenny Crump in Peer Review Comments, Silica Docket Item OSHA-2010-0034-1716, at 167.

247 Health Effects Review at 344.

248 Peer review comment of Kenny Crump in Peer Review Comments, Silica Docket Item OSHA-2010-0034-1716, at 167.

concentrations of respirable crystalline silica above those permitted by OSHA's current general industry PEL [of 100 μg/m^3]."[249] That is an understatement. In fact, the average exposures of these cohorts tended to be very much higher than the current general industry PEL of 100 μg/m^3. For example, in the British coal worker cohort that OSHA identifies as providing the most reliable basis for estimating risks of silicosis morbidity, "there were periods of extremely high exposure to respirable quartz in the mine (greater than 2 mg/m^3 in some jobs between 1972 and 1976, and more than 10 percent of exposures between 1969 and 1977 were greater than 1 mg/m^3)."[250] Indeed, 10 percent of the quarterly mean measurements for a portion of this period were over 10 mg/m^3.[251] These exposure concentrations are 10-20 (and as much as 100) times higher than the current general industry PEL. When combined with the evidence of a dose-rate effect for silicosis morbidity, they are quite consistent with the existence of an exposure concentration threshold for silicosis that is greater than 100 μg/m^3.

This last point is important to bear in mind when considering OSHA's assertion "that silicosis remains a significant cause of early mortality and of serious morbidity, despite the

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249 78 Fed. Reg. at 56324.

250 *Id.* As the British Health and Safety Executive explains: "Records showed that in the 1970's, exceptionally high levels of respirable quartz were generated by mechanical cutting into sandstone strata above and below a coal seam (seam B), where >10% of mean exposures exceeded 1 mg.m^{-3} respirable quartz (some exposures as high as 10 mg.m^{-3})." British Health and Safety Executive, Respirable crystalline silica – Phase 1: Variability in fibrogenic potency and exposure-response relationships for silicosis (2002) ("British HSE Phase 1 Report") at 41. Available on-line at http://www.whitehouse.gov/sites/default/files/omb/assets/oira_1218/1218_meeting_04072011-7.pdf.

251 See Health Effects Review at 334.

existence of an enforceable exposure limit over the past 40 years." That statement needs to be put in context. First, as discussed in section I. above, there has been a dramatic reduction in silicosis mortality rates over the past four decades. Second, while the 100 μg/m^3 exposure limit for general industry may have been *enforceable* for the last four decades, it clearly was not universally *enforced* – as evidenced by the widespread overexposures (averaging more than 30 percent of OSHA's compliance samples for most of this period).[252] And, as OSHA notes, for the construction and maritime industries, the "enforceable exposure limit" over the last four decades has been equivalent to 250-500 μg/m^3. See 78 Fed. Reg. at 56328. So, even if this *enforceable* exposure limit had been rigorously enforced (which it was not), the cases of silicosis that are found today would tell us little about whether a universally enforced exposure limit of 100 μg/m^3 is protective against silicosis.

These points are significant because, as discussed above (see pp. 52-65, *supra*), mechanistic and empirical evidence as well as theoretical considerations point to the existence of what Dr. Cox terms a "tipping point" threshold above 0.1 mg/m^3 for the risk of any malignant as well as non-malignant respiratory pathologies that may be associated with prolonged exposure to crystalline silica.[253] As Dr. Cox points out, to do harm, exposures to poorly soluble particulates (PSPs) such as crystalline silica must be intense enough and last long enough to trigger the chronic inflammatory responses and progression to a high-ROS (reactive oxygen species) state that can eventually lead to disease. Risk of disease is not increased by exposures while homeostasis is maintained, and disrupting normal homeostasis

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[252] See pp. 11-19, *supra*.

[253] Cox, L.A. Jr., An Exposure-Response Threshold for Lung Diseases and Lung Cancer Caused by Crystalline Silica. Risk Analysis. 2011; 31(10):1543-1560. Available on-line at http://dx.doi.org/10.1111/j.1539-6924.2011.01610.x.

requires activating positive feedback loops capable of damaging tissue (respiratory epithelium) and overwhelming normal repair processes. Both rat data and mathematical modeling of inflammation-mediated lung diseases indicate that these responses to PSPs have exposure-response thresholds. For respirable crystalline silica, Dr. Cox notes, the threshold for turning on possible disease processes appears to be above 0.1 mg/m^3 and may be as high as 0.4 mg/m^3.[254] Consequently, if long-term average silica exposures are maintained at or below 0.1 mg/m^3, the risk of developing silicosis should be negligible or nonexistent.

A number of studies provide evidence that this is, indeed, the case. Thus, in a study of British industrial sand workers, T. Brown and L. Rushton found no exposure-related effect with respect to lung cancer mortality in a cohort whose geometric mean exposure to respirable quartz was 0.09 mg/m^3.[255] And an update of the Vermont granite worker study by W. Graham *et al.* found that in workers hired after 1940 (when the mean silica exposure for most workers was reduced to below 0.1 mg/m^3, with approximately 11% of workers having exposures above that level), the SMRs for tuberculosis and non-malignant respiratory disease were not elevated, and there were no silicosis deaths among workers whose quartz exposures were only in the Vermont granite industry.[256] Similarly, in a more recent and comprehensive

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[254] See *id.* The reason Dr. Cox places the threshold in the range of 0.1 mg/m^3 - 0.4 mg/m^3 is because of uncertainty regarding the distribution of exposure estimation errors around true values in the various epidemiological studies of silica-exposed workers. See *id.*

[255] Brown TP and Rushton L. (2005). Mortality in the UK industrial silica sand industry: 1.Assessment of exposure to respirable crystalline silica. Occup Environ Med 62:442–445. OSHA-2010-0034-0303; Brown TP and Rushton L. (2005b). Mortality in the UK industrial silica sand industry: 2. A retrospective cohort study. Occup Environ Med 62:446–452. OSHA-2010-0034-0304.

[256] W. Graham *et al.*, Vermont Granite Mortality Study: An Update With an Emphasis on Lung Cancer. JOEM. 2004; 46(5): 459-466.

mortality study of the Vermont granite worker cohort, P. Vacek and colleagues found that after adjustment for the years worked at other levels of intensity, only employment at intensities >0.20 mg/m^3 were significantly associated with silicosis mortality.[257]

Recent studies of almost 18,000 German porcelain industry workers have yielded similar results. Thus, K. Mundt *et al.* found that the relationship between radiological silicosis and respirable silica was statistically significant only at levels greater than 0.15 mg/m^3 (average exposure) or 4 mg/m^3-years (cumulative exposure) – suggesting a threshold for silicosis roughly at or above those levels.[258] OSHA expressed skepticism about the significance of these findings, arguing that no formal threshold analysis was performed.[259] But precisely such an analysis subsequently was performed by P. Morfeld and co-workers, who used hockey-stick regression modeling to estimate a NOAEL (no observed adverse effect level) for the respirable silica concentration associated with silicosis.[260] While cumulative exposure was the driving variable in the models used by Morfeld *et al*. 2013, they sought "to account for the effect of intensity of exposure," by extend[ing] the modeling and search[ing] for a threshold for RCS dust concentration nested within the cumulative exposure

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[257] P. Vacek *et al*., A Study of the Relationship between Mortality and Silica Exposure in the Vermont Granite Industry: Final Report (November 16, 2009). A copy of this report is submitted herewith as Attachment 6.

[258] Mundt, K. *et al.*, Respirable Crystalline Silica Exposure-Response Evaluation of Silicosis Morbidity and Lung Cancer Mortality in the German Porcelain Industry Cohort. JOEM. 2011; 53(3): 282-289. Their analysis also suggested that the relationship between cumulative respirable silica exposure and silicosis is not linear.

[259] See Supplemental Health Review at 11.

[260] Morfeld, P. *et al.*, Threshold Value Estimation for Respirable Quartz Dust Exposure and Silicosis Incidence Among Workers in the German Porcelain Industry. JOEM. 2013; 55(9):1027-1034.

modeling."[261] They found that when no threshold was accounted for, "the relative risk estimates for radiographic opacities were similar to those published by Park et al 2002a." However, when they used the hockey-stick regression modeling procedure, they found that a threshold Cox model fit the radiographic silicosis data of the cohort significantly better overall than a non-threshold model – and that the best estimate for the threshold was 0.25 mg/m^3 (95%-CI: 0.15 mg/m^3, 0.30 mg/m^3) measured as an annual average concentration.[262] The authors explored a latency effect by lagging exposures and found that the threshold estimates did not differ when using unlagged silica exposures and silica exposures lagged by ten years. They went on to note that since their analysis focused on *annual average* concentrations, they may have underestimated the threshold based on *shift averages* by at least a factor of 2. This is significant because shift averages are most relevant to setting a PEL.

In addition to matching up well with epidemiological findings and mechanistic considerations, the existence of a concentration threshold above 100 $\mu g/m^3$ for silicosis goes a long way to explaining the dramatic decline of more than 90 percent in rates of mortality from silicosis and silicosis-respiratory tuberculosis (and the decline of more than 83 percent in the annual rate of silicosis-related discharges from short-stay non-federal hospitals) that

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261 See Morfeld Comment at 20-21.

262 See Morfeld, P. *et al.*, Threshold Value Estimation for Respirable Quartz Dust Exposure and Silicosis Incidence Among Workers in the German Porcelain Industry. JOEM. 2013; 55(9):1027-1034; Morfeld Comment at 20. That threshold value is consistent with the apparent intensity thresholds of 0.20 mg/m^3 for silicosis mortality and 0.30 mg/m^3 for non-malignant respiratory disease mortality suggested in the study of Vermont granite workers by Vacek *et al.* See Vacek, P. *et al.*, A Study of the Relationship between Mortality and Silica Exposure in the Vermont Granite Industry: Final Report (November 16, 2009) (Attachment 6 hereto) at 19.

the United States has witnessed over the course of the four decades during which the general industry PEL of 100 μg/m^3 has been in effect.[263] The fact that silicosis mortality and morbidity continue to be found (though at greatly reduced rates) is perfectly consistent with the existence of a concentration threshold above 100 μg/m^3 – because the silicosis cases that still arise almost certainly are attributable to the widespread and often extreme exceedances of the 100 μg/m^3 PEL that stubbornly persist year after year at rates of 30 percent or more based on OSHA's own sampling.[264] The fact that silicosis rates have fallen by over 90 percent despite these widespread overexposures provides real life confirmation of the point that when exposures do not exceed 100 μg/m^3, they are below the threshold at which a risk of silicosis arises. If that were not the case, one would hardly expect to see a reduction of more than 90 percent in silicosis mortality rates when, by OSHA's own reckoning, more than 500,000 employees are exposed to RCS concentrations greater than 100 μg/m^3 (with 265,000 of these being exposed above 250 μg/m^3), and more than 1,643,000 others have RCS exposures ranging up to 100 μg/m^3.[265] Given exposure numbers like these, it is hard to explain how there could have been more than a 90 percent decline in the number of annual deaths from silicosis on a multiple-cause basis (falling from 1,065 in 1968 to 101 in 2010) if there were not an exposure concentration threshold above 100 μg/m^3.[266]

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263 See pp. 8-10, *supra*.

264 See pp. 11-19, *supra*.

265 See 78 Fed. Reg. at 56352, Table V-5.

266 See pp. 8-9, *supra*. Nor would one have seen the 83 percent decline in the annual rate of silicosis-related discharges from short-stay non-federal hospitals. See p. 11, n. 13 *supra*.

While the Morfeld *et al.* (2013) analysis focused on identifying a concentration threshold for *silicosis*, it is consistent with the finding of apparent cumulative exposure thresholds for *lung cancer* ranging from >4 mg/m^3-years to 10 mg/m^3-years in the German porcelain worker cohort and a number of others.[267] Since silica-related lung cancer and silicosis appear to operate through the same inflammation-mediated mechanism,[268] it is not surprising to find essentially the same concentration threshold for the two endpoints. Thus, the concentration threshold for silicosis identified by Morfeld *et al.* (2013) can reasonably be applied to lung cancer and other non-malignant respiratory effects of silica exposure as well as to radiological silicosis.

The fact that Morfeld *et al.* (2013) contains the most explicit finding of an exposure concentration threshold for silicosis is not surprising. As Dr. Morfeld explains, other investigators have not explicitly searched for a concentration threshold, so one would not expect them to find one.[269] But, as he and Dr. Cox point out, the studies of other investigators are not incompatible with the existence of a concentration threshold in the low exposure region.[270] This is particularly true since, as noted above, a number of authors have found that intensity of exposure has a decided impact on silicosis risk.

Dr. Morfeld sums up his analysis with the observation that "every quantitative risk assessment of the association between RCS dust exposure and silicosis or lung cancer should

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[267] See pp. 56-59, *supra*.

[268] See pp. 52-55, *supra.*

[269] See Morfeld Comment at 21-22.

[270] See *id.*; Cox Comments at 2, 30-31, 38-39, 40-42, 46, 66-67, 71-72, 78, 85, 88, 90, 96, 98, 99.

allow for the existence of thresholds – otherwise the numbers returned are potentially biased. . . . If the existence of a threshold is considered, the findings [of Morfeld *et al.* (2013)] point to the conclusion that no effects will be observed if the RCS dust concentration is kept below 0.1 mg/m^3 in every working shift."[271] Since the risk assessment models used by OSHA "did not consider thresholds, . . . its risk estimates are potentially biased," and its projections of silicosis morbidity and mortality at the current general industrty PEL of 100 μg/m^3 and the proposed PEL of 50 μg/m^3 "are unreliable," as they ignore "the apparent existence of a RCS concentration threshold >0.1 mg/m^3."[272]

2. OSHA's Projections of NMRD Mortality Risks at Exposure Levels of 100 μg/m^3 and Below Are Not Credible or Reliable.

Based on the study of diatomaceous earth (DE) workers by Park *et al.* (2002), OSHA projects an excess NMRD mortality risk of 83 per 1,000 workers exposed to crystalline silica for 45 years at a level of 100 μg/m^3.[273] For a variety of reasons, this projection rests more on speculation than on solid evidence.

For one thing, the exposure assessment for this cohort was subject to considerable uncertainty and likely exposure misclassification. There were no dust monitoring data for the years before 1948 – with the majority of measurements dating from the early 1960s – so

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271 See Morfeld Comment at 23.

272 *Id.*

273 See Occupational Exposure to Respirable Crystalline Silica – Review of Health Effects Literature and Preliminary Quantitative Risk Assessment ("Health Effects Review") at 351. The study by Park *et al.* refers to NMRD mortality as LDOC (lung disease other than cancer) mortality. Since OSHA refers to this health outcome as NMRD mortality, we will do so in these Comments as well.

exposures estimates for the earlier years had to be extrapolated.[274] Also, there was uncertainty about conversion factors from particle count to gravimetric values.[275] Thus, Park *et al.* had to acknowledge that there may have been exposure misclassification, particularly in the early years when sampling data were lacking.[276] Supporting this supposition is the fact that the silicosis incidence rate in the 1942-1954 period was found to be *13.3 times higher* than in later years when workers having the same cumulative exposures are compared. In fact, 73 percent of the silicosis morbidity incidence cases occurred during the first 13 of 53 years of follow-up (from 1942 through 1954), *i.e.*, 73 percent of these cases occurred during just 25 percent of the total follow-up period, which extended from 1942 through 1994.[277] While other factors may have contributed to these rather anomalous findings, exposure misclassification very likely played a major role. The fact that Park *et al.* found no deaths from NMRD in the highest cumulative exposure group also suggests the existence of exposure misclassification in this study.[278]

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274 See Checkoway, H., *et al.* (1997). Dose-response Associations of Silica with Nonmalignant Respiratory Disease and Lung Cancer Mortality in the Diatomaceous Earth Industry. Am J Epidemiol 145:680–688 at 685. OSHA-2010-0034-0326.

275 See *Id.*

276 Park, R. *et al.* (2002). Exposure to crystalline silica, silicosis, and lung disease other than cancer in diatomaceous earth industry workers: A quantitative risk assessment. Occup Environ Med 59:36–43 at 41. OSHA-2010-0034-0405.

277 *Id.* at 39.

278 It should be noted that the exposure assessment in the Park *et al.* (2002) study, as well as the exposure assessment in the Rice *et al.* (2001) study of lung cancer in DE workers and the Hughes *et al.* (1998) study of radiographic silicosis in DE workers, was based on the exposure assessment developed in an earlier study of the DE worker cohort by Checkoway *et al.* (1997) and Seixas *et al.* (1997). See Checkoway, H., *et al.* (1997). Dose-response Associations of Silica with Nonmalignant Respiratory Disease and Lung Cancer Mortality in the Diatomaceous Earth Industry. Am J Epidemiol 145:680–688. OSHA-2010-0034-0326;

Another reason for uncertainty in the exposure assessment is a lack of clarity as to the silica percentage in respirable dust. Checkoway *et al.* (1997) based their estimates on the assumption that the silica content of the various products was 1% for uncalcined DE (quartz content of ore), 10% for calcined DE, and 20% for flux-calcined DE. By contrast, Hughes *et al.* (1998) used silica content values of 3%, 20% and 60%, respectively, for those products – and they dichotomized the cohort into those who were exposed to dust with a silica content of <35% vs. >35%.[279] Park *et al.* (2002) appear to have used values ranging from 1% to 25%, which would tend to understate silica exposures based on low estimates of silica content.

As Dr. Cox points out, despite these exposure uncertainties:

> None of the models considered by Park et al. (1a-1f on p. 37) includes the possibility that estimated cumulative exposures to silica might differ from the corresponding correct values. (The term E in Park et al.'s model forms 1a-1f does not contain any model for exposure estimation errors or uncertainties). This alone invalidates the model parameter estimates and interpretation, since correct individual exposures are not known and may differ significantly from estimated values based on estimated average exposure concentrations for job categories.[280]

In addition to considerable uncertainty in the exposure assessment and likely exposure misclassification, there is a real question as to whether results for the DE worker

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Hughes, J.M. *et al.* (1998). Radiographic Evidence of Silicosis Risk in the Diatomaceous Earth Industry. Am J Respir Crit Care Med 158:807–814. OSHA-2010-0034-1059; Seixas, N.S., *et al.* (1997). Quantification of historical dust exposures in the diatomaceous earth industry. Ann Occup Hyg 41:591–604. OSHA-2010-0034-0431.

[279] Hughes, J.M. *et al.* (1998). Radiographic Evidence of Silicosis Risk in the Diatomaceous Earth Industry. Am J Respir Crit Care Med 158:807–814. OSHA-2010-0034-1059.

[280] Cox Comments at 31.

cohort, whose mean silica exposure levels were very high, can appropriately be extrapolated to workers whose mean exposures are 100 μg/m^3 and below. By the authors' own estimate, the mean RCS exposure in the DE worker cohort was 0.29 mg/m^3, which is three times the current PEL for general industry. Since exposures in the early years (for which no measurements existed) probably were underestimated (as indicated by the silicosis data discussed above), the mean RCS exposure for the cohort as a whole presumably was even higher than the 0.29 mg/m^3 level estimated by the authors. Moreover, the mean RCS exposure of the 51 silicosis incident cases in the 1942-1954 period was estimated to be 7.1 mg/m^3-years; for the 5 cases in the subsequent 10 years, it was 5.4 mg/m^3-years.[281] If these individuals had the same mean duration of employment as the cohort as a whole (7.4 years), their mean RCS exposure would have approached 1.0 mg/m^3, or almost 10 times the level of the current PEL for general industry. The importance of high mean exposures in this cohort also was demonstrated in the earlier study by Hughes *et al.* (1998), where workers who had a mean crystalline silica exposure of >0.50 mg/m^3 showed a much higher incidence rate and age-adjusted RR for radiological opacities than workers who had a mean crystalline silica exposure of <0.50 mg/m^3, even when their cumulative exposure was the same.[282] Similarly, in the earlier study of NMRD mortality in the DE worker cohort, Checkoway *et al.* (1997) found that the SMR for NMRD mortality was significantly elevated only in workers whose

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[281] Park, R. *et al.* (2002). Exposure to crystalline silica, silicosis, and lung disease other than cancer in diatomaceous earth industry workers: A quantitative risk assessment. Occup Environ Med 59:36–43 at 39-40. OSHA-2010-0034-0405.

[282] At a cumulative exposure of 4 mg/m^3-years, those having a mean exposure >0.50 mg/m^3 were almost four times as likely to exhibit radiological opacities as those having a mean exposure <0.50 mg/m^3. See Hughes, J.M. *et al.* (1998). Radiographic evidence of silicosis risk in the diatomaceous earth industry. Am J Respir Crit Care Med 158:807–814. OSHA-2010-0034-1059.

year of hire was before 1950 (when exposures were particularly high).[283] By the same token, using the unlagged model that Park *et al.* say fits the data better and is more biologically plausible, the relative risk for NMRD mortality in Checkoway *et al.* (1997) was statistically significant only in the highest cumulative exposure category (>5 mg/m^3-years RCS).[284]

A similar finding was made by Vacek *et al.* (2011) in the Vermont granite worker cohort, where there was no positive association between cumulative silica exposure and NMRD mortality except in the highest exposure category (>5.41 mg/m^3-years) – where a non-significant elevated odds ratio was observed[285] – and the "trend test was far from being significant (p=0.32)."[286] The mean average exposure for workers in the high cumulative exposure group was 220 μg/m^3, and the minimum average exposure for any worker in that group was 110 μg/m^3.[287] Similarly, in a recent update of the Stoke-on-Trent pottery workers cohort, Cherry *et al*. (2012) found a lack of dose-response between cumulative silica exposure and mortality from COPD. As the authors put it: "The lack of dose–response for lung cancer or COPD in more recent periods, or for cNMRD [chronic non-malignant

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283 See Checkoway, H., *et al.* (1997). Dose-response Associations of Silica with Nonmalignant Respiratory Disease and Lung Cancer Mortality in the Diatomaceous Earth Industry. Am J Epidemiol 145:680–688 at 685, Table 4. OSHA-2010-0034-0326.

284 See *id.* at 686, Table 5.

285 See Vacek, P. *et al.*, Mortality in Vermont granite workers and its association with silica exposure. Occup Environ Med. 2011; 68:312-318, Table 5. Available on-line at http://dx.doi.org/10.1136/oem.2009.054452.

286 Morfeld Comment at 29.

287 Personal communication from Pamela Vacek, Ph.D., November 25, 2013.

respiratory disease] at any period, requires some consideration."[288] The results in Vacek *et al.* (2011) and Cherry *et al.* (2012) throw into question the finding of an association between silica exposure and NMRD mortality in Park *et al.* (2002) and suggest that if there is an association, it occurs only above an average exposure threshold that exceeds 100 $\mu g/m^3$.[289]

In short, the Park *et al.* (2002) study is fully consistent with the existence of an exposure threshold above 100 $\mu g/m^3$ for any NMRD mortality effects of crystalline silica. This is particularly true since Park *et al.* (2002), as Dr. Peter Morfeld points out, "did not search for threshold effects."[290] And, as Dr. Cox notes, the model forms they used "assume no thresholds or J-shaped relations, independent of what the data show, and in conflict with biologically-based evidence and the findings of Health Canada and others (Cox, 2011) that a threshold approach to risk assessment is appropriate for crystalline silica because lung cancer (and other inflammation-mediated lung diseases) have an etiology in which positive feedback loops play a prominent role."[291] For these reasons alone, "risk estimates [based on Park *et al.* (2002)], in particular for low level exposures, are questionable."[292]

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288 Cherry, N. *et al.*, Mortality in a cohort of Staffordshire pottery workers: follow-up to December 2008. Occup. Environ. Med., published online October 26, 2012. Available on-line at http://oem.bmj.com/content/early/2012/10/25/oemed-2012-100782.full.html.

289 The deficit in mortality from emphysema found by Vacek *et al.* (2011) is consistent with the only longitudinal studies for emphysema, Hnizdo (1991) and (1994), which did not find an association of dust or silica with emphysema, particularly among non-smokers. See 78 Fed. Reg. at 56305. Indeed, in a subsequent study, Hnizdo (2000) found that emphysema prevalence decreased with increasing dust exposure. See *id.*

290 Morfeld Comment at 28.

291 Cox Comments at 91.

292 Morfeld Comment at 28.

Quite apart from the exposure and threshold issues discussed above, the results of Park *et al.* (2002) may reflect confounding by smoking and possibly by asbestos. In the Park *et al.* (2002) study, data on smoking habits was available for only 50 percent of the cohort and, even then, only on an "ever-versus-never" smoked basis. Thus, as Checkoway *et al.* (1997) acknowledged: "Our ability to assess potential confounding by smoking [in the DE worker cohort] was limited by incomplete, crude data."[293] Among the factors making the smoking data incomplete was the fact that *smoking habits were unknown for 67 percent of the workers who died from NMRD.* What is known is that there was a lower prevalence of smoking in workers with the lowest cumulative exposures, which could have confounded the internal comparisons in the DE worker cohort.[294] Also, the NMRD mortality rate for Hispanic workers in the cohort was about half that of other workers – a finding which, as the authors point out, probably reflected the lower smoking rates among Hispanic workers.[295] This, too, suggests that smoking was a likely confounder here. In fact, OSHA itself suggests that unaccounted for smoking habits likely produced exaggerated estimates of lung cancer risk in the DE worker cohort, and the same presumably would be true of NMRD mortality.[296]

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[293] Checkoway, H., *et al.* (1997). Dose-response associations of silica with nonmalignant respiratory disease and lung cancer mortality in the diatomaceous earth industry. Am J Epidemiol 145:680–688 at 687. OSHA-2010-0034-0326.

[294] See *id.*

[295] See Park, R. *et al.* (2002). Exposure to crystalline silica, silicosis, and lung disease other than cancer in diatomaceous earth industry workers: A quantitative risk assessment. Occup Environ Med 59:36–43 at 38. OSHA-2010-0034-0405.

[296] See Health Effects Review at 278 (noting that the much lower lung cancer risk estimates derived from the British coalminer study by Miller and MacCalman (2009) compared to risk estimates based on other studies (including the DE worker study) likely reflect the fact that the estimates in Miller and MacCalman "are adjusted for individual smoking histories so any smoking-related lung cancer risk (or smoking – silica interaction)

According to OSHA, "it appears that the silica-related risk [of NMRD mortality] is strongly influenced by smoking, and the effects of smoking and silica exposure may be synergistic."[297] That being the case, the limited smoking data available for the DE worker cohort and the indications that lower smoking rates were associated with lower NMRD mortality suggest that smoking very well may have confounded the NMRD mortality results reported by Park *et al.* (2002).

Similarly, while Park *et al.* dismissed asbestos as a potential confounder and omitted asbestos exposure in their final models, the situation is not as clear-cut as they would have one believe. They rely on Checkoway *et al.* (1997), which they say "found no evidence that exposure to asbestos accounted for the observed association between mortality from LDOC and cumulative exposure to silica in the diatomaceous earth cohort."[298] But, while Checkoway *et al*. (1997) attempted to control for asbestos exposure, there was limited data – so that, as the authors acknowledged, "misclassification of asbestos exposure may have hindered our ability to control for asbestos as a potential confounder."[299] Finally, as Dr. Cox points out, there also may have been uncontrolled residual confounding by age because it is likely that older workers within a five-year age group category have had higher exposures

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that might possibly be attributed to silica exposure in the other studies will not be reflected in the risk estimates derived from the study of these coalminers.").

[297] Health Effects Review at 206.

[298] Park, R. *et al.* (2002). Exposure to crystalline silica, silicosis, and lung disease other than cancer in diatomaceous earth industry workers: A quantitative risk assessment. Occup Environ Med 59:36–43 at 41. OSHA-2010-0034-0405.

[299] Checkoway, H., *et al.* (1997). Dose-response associations of silica with nonmalignant respiratory disease and lung cancer mortality in the diatomaceous earth industry. Am J Epidemiol 145:680–688 at 685. OSHA-2010-0034-0326.

and "have higher probabilities of lung cancer and other respiratory illnesses than younger workers."[300]

In addition to the points discussed above, Park *et al.* (2002), as Dr. Peter Morfeld notes,

> suffers from a methodological drawback: the final models and the excess lifetime risk calculations based on these models do not use the full data available but evaluated a selected subset. The authors truncated the cumulative RCS dust exposures before doing the final analyses based on their observation of where the cases were found. The maximum in the study was 62.5 mg/m^3-years but exposures were only used up to 32 mg/m^3-years because no LDOC deaths occurred at exposures higher than that level. Such a selection distorts the estimated exposure-response relationship because it is based on the outcome of the study and on the exposure variable. Because high exposures with no effects were deliberately ignored, the exposure-response effect estimates are biased upward.[301]

Dr. Morfeld continues: "The authors justified the procedure as an adjustment for the healthy-worker survivor effect. The truncation procedure they used, however, is not appropriate. Techniques are available to control for the healthy-worker survivor effect without excluding any data or making biased selections (Robins 1998, Joffe 2012) and should have been used instead."[302] The authors, however, did not do so – preferring, instead, to explain away non-conforming data. But, as Dr. Cox observes: "*Post hoc* deletion of one sixth of the deaths in order to achieve a monotonically increasing exposure-response relation invalidates the significance of this reported relation: the data were edited as necessary to achieve the

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300 See Cox Comments at 32-33.

301 Morfeld Comment at 27.

302 *Id.*

result."[303] And "[m]aking up hypothetical explanations for why the data do not support one's preferred beliefs is a recipe for confirmation bias."[304]

In addition to the foregoing, "general dust exposures," as Dr. Morfeld notes, "can cause non-malignant respiratory diseases (covering chronic bronchitis, emphysema and pneumoconiosis, Cherrie et al 2013)," and "it is difficult to disentangle the effect of general dust exposures and the effect of dust components, like RCS, on non-malignant respiratory diseases because the correlations between both entities are usually rather high or uncertain (Dahmann et al 2008b)."[305] Park *et al.* (2002), as Dr. Morfeld points out, "did not take account of the effect of general dust exposure on non-malignant respiratory disease when estimating the effect of RCS dust exposures. Thus, it is unclear how much the risk estimates based on that study might change if adjustments were made for the general dust exposures of the cohort."[306] Particularly given the considerable uncertainties as to the silica content of the dust to which the DE workers were exposed and their overall levels of respirable silica exposure (see pp. 102-103, *supra*), "the cause of an observed excess of non-malignant respiratory disease mortality (other than silicosis) after occupational dust exposure in the DE worker cohort remains unclear, as does the shape of the relationship between RCS exposure and NMRD mortality (including the concentration level that may be a threshold for any such relationship)."[307]

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303 Cox Comments at 32.

304 *Id.* at 92.

305 *Id.* at 31.

306 *Id.* at 32.

307 *Id.* at 31.

Questions surround the approach to modeling in Park *et al.* (2002) as well. The authors selected a single model (the linear relative rate model) and unlagged cumulative exposure to assess risks of NMRD mortality in the cohort. But, as Dr. Cox explains: "Selecting a single best-fitting model form (e.g., the linear relative rate model) and exposure metric is a recipe for over-fitting bias, model selection bias, model specification error, biases due to ignored model uncertainty, and errors and biases due to failure to use multiple imputation of uncertain exposure values."[308] Model uncertainty, as Dr. Cox notes, is likely to be of particular concern in this study because "Figures 1 and 2 of Park et al. show that the choice of model form makes a huge difference to results," and "[t]here is no guarantee that any of the models considered provides an approximately correct description of the true exposure-response association, nor that other, better-fitting models might not provide risk estimates very different from any of those shown."[309] Moreover, use of an unlagged exposure model is "not very biologically plausible for dust-related LDOC deaths (if any) caused by exposure concentrations in the range of interest. Unresolved chronic inflammation and degradation of lung defenses takes years to decades to manifest."[310] In any event, no justification was provided for selecting these model forms, which "assume no thresholds or J-shaped relations, independent of what the data show, and in conflict with biologically-based evidence and the findings of Health Canada and others (Cox, 2011) that a threshold approach to risk assessment is appropriate for crystalline silica because lung cancer (and other

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308 Cox Comments at 92.

309 *Id.* at 31.

310 *Id.* See also *id.* at 92.

inflammation-mediated lung diseases) have an etiology in which positive feedback loops play a prominent role."[311]

Finally, as Dr. Cox reminds us:

> It is inappropriate to use a study of statistical associations, such as the Park et al. study, to estimate the risk of NMRD mortality caused by exposure. Association is not causation. Also, the lifetable analysis [which OSHA employed] does not model dependencies among competing risks, and hence its results may not reflect reality (Tsiatis, 1975). The linear relative risk model has no justification for this application, and is inconsistent with the underlying biology of threshold-like responses for exposure-related lung damage and disease induction (e.g., Mauderly et al., 1997; Oberdorster, 1996; Nikula et al., 1997; Cox, 2011).[312]

In light of the foregoing points, OSHA's projection that 45 years of occupational exposure to respirable crystalline silica at a level of 0.1 mg/m^3 will result in 83 excess deaths from non-malignant respiratory disease per 1,000 workers is unsupported and unreliable.

The same is true of OSHA's estimates of silicosis mortality based on Mannetje et al (2002), which involved a pooled analysis of data from six of the ten cohorts that were included in the pooled analysis of lung cancer in Steenland *et al.* (2001). As in the case of the pooled lung cancer analysis, there is enormous uncertainty in the exposure assessment used by Mannetje et al (2002) and a high likelihood of exposure misclassification. Also, Mannetje et al (2002) provided no justification for the relative rate model forms they used to evaluate exposure-response, all of which "assume no thresholds or J-shaped relations, independent of what the data show."[313] Thus, "the conditional logistic regression model form [used by Mattetje *et al.* (2002)] *assumes* a monotonic relationship between each

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311 *Id.* at 91.

312 *Id.* at 93.

313 See *id.* at 91.

continuous predictor and the probability of response, so . . . the statistical models and methods used [in the pooled analysis] . . . cannot reliably discriminate between monotonic and non-monotonic relationships."[314] Accordingly, the apparent monotonic response that Mannetje et al (2002) claimed to find with *estimates* of increasing cumulative exposure "could easily be found even if the true response-vs.-exposure relation is not monotonic (e.g., is J-shaped, or increasing only above a threshold value), due to effects of exposure estimation error."[315]

In any event, there is no statistically significant difference in the odds ratios for silicosis mortality associated with different estimated cumulative exposures in the pooled analysis of Mannetje *et al.* (2002). Despite order-of-magnitude differences between high and low estimated exposures, their confidence intervals all overlap, as shown below, and the exposure-response relation is not even fully monotonic.

- 4.45 mg/m^3-years, OR = 3.1 (95% CI, 2.5-4.0);
- 9.08 mg/m^3-years, OR = 4.6 (95% CI, 3.6-5.9);
- 16.26 mg/m^3-years, OR = 4.5 (95% CI, 3.5-5.8); and
- 42.33 mg/m^3-years, OR = 4.8 (95% CI, 3.7-6.2).

As Dr. Cox notes:

> There is no evidence here that reducing cumulative exposure from 10 mg/m^3-years to 5 mg/m^3-years would reduce the odds ratio (or that increasing cumulative exposure to 40 mg/m^3-years would increase the odds ratio). Interpreting such results as evidence for a monotonic exposure-response relation, in which halving the PEL will approximately halve excess risks, over-interprets what these data actually show, which is no clear effect of exposure on odds ratios over this entire range.[316]

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314 *Id.* at 64.

315 *Id.* at 65.

316 *Id.* at 66.

Thus, Mannetje *et al.* (2002) does not provide a reliable basis for estimating the risk of silicosis mortality.[317] This is particularly true with respect to exposures of 100 μg/m^3 and below – because the exposure-response models considered by the authors and by OSHA do not allow for an exposure threshold above 100 μg/m^3 – even though, as discussed above, the evidence points to a threshold of approximately 250 μg/m^3 for radiological silicosis, and the decline of more than 90 percent in silicosis mortality rates during the time in which the current general industry PEL of 100 μg/m^3 has been in effect appears to confirm the point.

3. OSHA's Projections of Silicosis Morbidity Risks at Exposure Levels of 100 μg/m^3 and Below Are Not Credible or Reliable.

OSHA projects that after 45 years of occupational exposure to respirable crystalline silica at a concentration of 100 μg/m^3 anywhere from 60 out of 1,000 workers (based on a study of the Chinese pottery industry) to 773 out of 1,000 workers (based on a study of South African gold miners) will develop radiological silicosis.[318] For a variety of reasons, those projections are not credible.

First and most fundamentally, as discussed in section II.C.1. above, silicosis is an inflammatory/fibrotic response having a concentration threshold above 100 μg/m^3 (most

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[317] In this connection, it should be noted that Y. Sun & F. Bochmann applied the exposure-response relationship estimated by Mannetje *et al.* (2002) to recalculate the lifetime risk of silicosis mortality for quartz-exposed workers using life table data for the German population in 1995. When they did this without allowing for latency from first exposure, they came up with risk estimates very close to those of Mannetje *et al.*. But when they allowed for a latency period of 28 years (based on the Mannetje *et al.* cohorts), the lifetime risk of silicosis mortality was only 1.6 and 0.7 per 1,000 workers for 45 years exposure at 0.1 mg/m^3 and 0.05 mg/m^3, respectively. See Sun, Y. & Bochmann, F., Letter to the Editor of Occup. Environ. Med. 2004; 61: 374-375.

[318] See Health Effects Review at 351-52, Table II-12.

likely in the neighborhood of 250 μg/m^3). Accordingly, whatever validity OSHA's risk estimates for silicosis might or might not have for workers exposed to high concentrations of RCS, they have no validity as applied to workers whose RCS exposures do not exceed the current general industry PEL of 100 μg/m^3. To be sure, the authors of the studies and OSHA itself purport to model silicosis risks to levels below 100 μg/m^3. But, as Dr. Cox observes, the models used in all of the studies relied on by OSHA assume that there is no exposure threshold for silicosis or the other health endpoints investigated. None of the authors investigated threshold models or searched for a concentration threshold in their studies, and their exposure-response coefficients and resulting risk estimates are predicated on the absence of a threshold.[319] In that sense, as Dr. Cox notes: "None of the studies relied on by OSHA for estimating silicosis morbidity risks is inconsistent with (or rules out) the existence of an exposure threshold above 100 μg/m^3."[320] And, as noted above, when Morfeld *et al.* (2013) explicitly searched for a concentration threshold nested within the cumulative exposure modeling in the German porcelain worker cohort, they found it at a level of 0.25 mg/m^3.[321]

Second, OSHA's projections of silicosis morbidity at any given exposure level cover a vast range of estimated risks. Even when limited to cumulative risk studies with post-employment follow-up, OSHA's risk estimates span more than an order of magnitude at the

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[319] See Cox Comments at 2, 30-31, 38-39, 40-42, 46, 66-67, 71-72, 78, 85, 88, 90, 96, 98, 99.

[320] *Id.* at 99.

[321] See pp. 98-99, *supra.*

100 μg/m^3 level.[322] The varying estimates are based on a variety of studies, all of which claim to have obtained statistically significant results. As Dr. Cox points out, however,

> obtaining *significant* results is not the same as obtaining *correct* results. We agree that OSHA has reported a large number of results which, while yielding mutually inconsistent (i.e., significantly different) risk estimates, all claim to be statistically significant – at least in the absence of any corrections for biases introduced by model misspecification, multiple testing, and post-hoc selection of data and models to guarantee this outcome. But the fact that they disagree with each other suggests that none of them is a reliable guide to a correct quantification of ER associations, i.e., the association that would be achieved (and, presumably, that could be replicated) in the absence of different modeling assumptions and choices designed to produce significance but not necessarily correctness of results.[323]

Third, the estimates of silicosis morbidity risks in most of the studies relied on by OSHA are based on highly uncertain (and very likely significantly understated) estimates of exposure. The three studies that produce the highest projections of silicosis morbidity – Hnizdo and Sluis-Cremer (1993), Steenland and Brown (1995), and Chen *et al.* (2001) – are good examples of this point.

Hnizdo and Sluis-Cremer (1993): The exposure assessment used in the study of South African gold miners by Hnizdo and Sluis-Cremer (1993) could hardly be more problematic and uncertain. Hnizdo and Sluis-Cremer (1993) did not do any exposure sampling or reconstruction of their own. Instead, they used exposure data from the late 1950s through the mid 1960s that was collected and organized by Beadle and Bradley (1970)

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322 See Health Effects Review at 337, Table II-11.

323 Cox Comments at 98. A related observation applies to the varying results of the Chinese mining and pottery worker study by Chen *et al.* (2005) – *viz.*: "The more than five-fold difference in risk estimates for the same exposure across industries suggests that silicosis morbidity risk is not well explained by estimated cumulative exposure, but reflects effects of other variables. It is a mistake to attribute these exposure-associated risks entirely to exposure, given these inconsistencies in the estimated ER relation across studies." *Id.* at 99.

as further analyzed by Page-Shipp & Harris (1972) and DuToit (1991).[324] The Beadle and Bradley data reflected shift-long samples of approximately 650 men collected at a random sample of 20 gold mines representing different geographical areas and mining conditions.[325] These were not gravimetric samples. Rather, they were particles collected with a Konimeter, a standard thermal precipitator, and a modified thermal precipitator. From the particle count values, estimates of "respirable surface area" ("R.S.A.") were calculated. These values were converted to "respirable mass" values "on a theoretical basis" – and, as Beadle acknowledged, "the accuracy and precision associated with it may not be comparable with results obtained by actually weighing samples."[326] Moreover, they noted, R.S.A. "is not an actual measure of surface area either, but is based on a theoretical calculation."[327] As the British Health and Safety Executive concluded: "The reliability of the conversion to

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[324] See Beadle, D.G. and Bradley, A.A. (1970). The Composition of Airborne Dust in South African Gold Mines. In Shapiro, H.A. (ed.): *Pneumoconiosis. Proceedings of the International Conference. Johannesburg 1969*. Oxford: Oxford University Press (1970), pp. 462-466; Page-Shipp, R.J. and Harris, E. (1972). A study of the dust exposure of South African white gold miners. Journal of the South African Institute of Mining and Metallurgy. 73(1):10-24; Du Toit, R.S.J., The shift mean respirable mass concentration of eleven occupations of Witwatersrand gold miners. NCOH Report No. 4/91 (February 1991).

[325] *See* Beadle, D.G. and Bradley, A.A. (1970), *supra*; Beadle, D.G., Harris, E. and Sluis-Cremer, G.K. (1970). The Relationship Between the Amount of Dust Breathed and the Incidence of Silicosis: An Epidemiological Study of South African European Gold Miners. In Shapiro, H.A. (ed.): *Pneumoconiosis. Proceedings of the International Conference. Johannesburg 1969*. Oxford: Oxford University Press (1970) , pp. 473-477; Beadle, D.G., The Relationship Between the Amount of Dust Breathed and the Development of Radiological Signs of Silicosis: An Epidemiological Study in South African Gold Miners. In Walton, W.H. (ed.): *Inhaled Particles III*. Oxford: Pergamon Press (1971), pp. 953-964; Page-Shipp and Harris (1972), *supra*.

[326] Beadle, D. and E. Harris, The Dust Miners Breathe (Chamber of Mines Contract 6/135/69), January 14, 1970, Appendix I.

[327] *Id.*

gravimetric units is uncertain, but is considered by HSE to represent a likely source of error in the exposure estimates."[328] In short, the exposure data developed by Beadle and Bradley and their colleagues (and used by Hnizdo and Sluis-Cremer) was uncertain even as applied to the period in the early to mid-1960s when the data were collected.

In addition to the questionable gravimetric values generated by Beadle and Bradley based on 1960s data, there are various reasons why the exposure values used by Hnizdo and Sluis-Cremer (1993) are questionable. These include the assumption that exposures remained essentially unchanged from the 1930s to the 1960s, the failure to consider differences in mining operations over time or inter-mine differences in exposure, and the fact that only particles in the size range of 0.5 – 5.0 µm were counted, whereas respirable dust includes particle sizes up to 10 µm .[329] These factors would tend to produce underestimates of exposure and overestimates of risk. Thus, as the British Health and Safety Executive points out:

> The authors [Hnizdo and Sluis-Cremer] indicated that if conditions prior to the 1960s had been dustier then this exposure estimate would lead to an underestimate of exposure, and hence an overestimate of the risk of silicosis. This possibility is supported by an earlier report cited by the authors which stated that from 1938 through the 1970s the average respirable silica concentrations in these mines would have been between 0.3 and 0.5 $mg.m^{-3}$, considerably above the 0.09 $mg.m^{-3}$ value used for miners in this study. Since miners in this study were employed from 1947 for an average of 24 years, many in this population would probably have received higher exposures in the past than indicated from the 1960s survey.[330]

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328 British HSE Phase 1 Report at 48.

329 See Hnizdo, E. and Sluis-Cremer, G., Risk of Silicosis in a Cohort of White South African Gold Miners. American Journal of Industrial Medicine. 1993; 24: 447-457; Gibbs, G.W. and du Toit, R.S.J., Estimating the Quartz Exposure of South African Gold Miners. Annals of Occupational Hygiene. 2002; 46:597-607; British HSE Phase 1 Report at 50.

330 British HSE Phase 1 Report at 48-49.

Furthermore, when R.J. Page-Shipp and E. Harris examined the data, they were struck by the fact that "[t]he range of exposures in any occupational group is very wide," an observation that they attributed primarily to two factors: (i) different mining methods are embraced by each occupational group, and (ii) the efforts of individuals are of primary importance in determining the dust level of their environment."[331] This wide variability in the measured dust levels within jobs suggests that among workers labelled by Hnizdo and Sluis-Cremer (1993) as having a given (average) exposure level, many would have been exposed to much higher or much lower levels. This likely would result in overestimates of the observed risk at a given (average) exposure level, since the miners within each exposure level grouping who developed silicosis most likely are those whose exposures were much higher than the average values used by Hnizdo and Sluis-Cremer (1993) to describe them.

The factor that appears to have the greatest impact on Hnizdo and Sluis-Cremer's risk estimates – and the one that is most readily quantifiable – is their assumption that the respirable gold mine dust to which cohort members were exposed had a 30% quartz content *after being incinerated and acid washed*.[332] That assumption is incorrect. As Beadle & Bradley (1970) explained, with the advent of a gravimetric sampler and a suitable X-ray technique, it was possible to determine the mass concentration of the total dust and of the quartz "*in mine air*." Based on the results for 6 mines surveyed, they reported that the

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331 Page-Shipp, R.J. and E. Harris (1972), *supra*.

332 See Hnizdo, E. and Sluis-Cremer, G., Risk of Silicosis in a Cohort of White South African Gold Miners. American Journal of Industrial Medicine. 1993; 24: 447-457 at 453.

average percentage of quartz *in the mine dust* was 31%.[333] By contrast, the average quartz percentage in the residue (after incineration and acid treatment) of the dust collected with an electrostatic precipitator was 54%.[334] And the particle count and respirable surface area data of Beadle and Bradley were converted to respirable mass *after the dust had been incinerated to remove combustibles and acid treated to remove soluble salts*.[335] Thus, the respirable mass dust concentrations and cumulative dust exposure ("CDE") values presented by Hnizdo & Sluis-Cremer (1993) reflected dust concentrations *after incineration and acid treatment*, rather than the actual mine dust concentrations to which the cohort members were exposed, and the silica content of the dust at that point was 54%, not 30%.[336] Because Hnizdo & Sluis-Cremer (1993) assumed that the quartz content of the incinerated and acid washed dust

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[333] See Beadle, D.G. and Bradley, A.A. (1970), *supra* at 462, 464, 465. See also British HSE Phase 2 Report at 47 (noting that crystalline silica was . . . thought to be about 30% of respirable dust" in the South African gold mines).

[334] See Beadle, D.G. and Bradley, A.A. (1970), *supra* at 465, Table II (average of last column). This last figure reflects data on samples collected at 4 mines from 1965-1967 and at 53 mines from 1958-1960.

[335] See Gibbs, G.W. & Du Toit, R.S.J., Estimating the Quartz Exposure of South African Gold Miners. Annals of Occupational Hygiene. 2002; 46:597-607.

[336] See Gibbs, G.W. & du Toit, R.S.J., Estimating the Quartz Exposure of South African Gold Miners. Annals of Occupational Hygiene. 2002; 46:597-607. In a 1977 report on a survey of 2,209 South African gold miners paper, Wiles and Faure stated that dust in the South African gold mines "contains about 75% free silica." Wiles, F.J. and Faure, M.H., Chronic Obstructive Lung Disease in Gold Miners. In Walton, W.H. (ed.): *Inhaled Particles IV, Part 2*. Oxford: Pergamon Press (1977), pp. 727-735 at 727. In a discussion note, R. Du Toit pointed out that the 75% figure refers to the free silica content of samples that have been heated and acid treated. *Id.* at 735. Even so, the 75% free silica value referred to by Wiles and Faure is significantly higher than the 54% silica content that Beadle & Bradley (1970) reported in the dust after incineration and acid treatment.

was 30%, rather than 54%, they appear to have underestimated quartz exposures of the miners by a factor of almost 2 (54/30 = 1.8) for that reason alone.[337]

As noted above, there are a number of additional reasons why the quartz exposures of gold miners in the cohort studied by Hnizdo and Sluis-Cremer were underestimated,[338] so the actual RCS exposures of cohort members may very well have been more than twice as high

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[337] See Gibbs, G.W. & Du Toit, R.S.J., Estimating the Quartz Exposure of South African Gold Miners. Annals of Occupational Hygiene. 2002; 46:597-607. Referencing a 1997 report by Kielblock *et al.* (indicating that mine dust sampled in the late 1980s and early 1990s contained about 15% silica) and a later report by Churchyard *et al.* (indicating a median silica content of 13.2% and 16.1% in samples taken in 2001-2002), OSHA suggests that the 30% value used by Hnizdo and Sluis-Cremer (1993) for the silica content of heat treated and acid washed dust may conceivably have been correct. See Health Effects Review at 332. It is not clear what accounts for the differences between what Beadle and Bradley reported as silica content (31% in the mine dust and 54% in the residue following incineration and acid treatment) and the lower values reported three and four decades later by Kielblock and Churchyard. But, since Hnizdo and Sluis-Cremer (1993) rely on the Beadle and Bradley data as the basis for estimating CDE (cumulative dust exposure) in their risk assessment, it seems appropriate for them to use the Beadle and Bradley quartz content information as well – unless it can be shown that the Beadle and Bradley analysis of quartz content was faulty, and OSHA has made no such showing. In addition, as noted in the previous footnote, Wiles and Faure reported a free silica content of about 75% in South African gold mine dust in 1977 (apparently after heat and acid treatment), and a recent paper by Gulumian, M, Semano, M. and Vallyathan, V. (*Surface Activity of Silica Dusts Collected from Different Mines in South Africa*, SIMRAC Project 020605) found that the quartz content of bulk dust samples from 10 different South African gold mines varied from 25% to 58%, the average being 33% – with most of the values clustering in the 27% - 35% range. Those values are consistent with the 31% silica content of mine dust reported by Beadle and Bradley.

[338] See p. 118, *supra*. It also is worth noting that in a discussion of the 1969 paper by Beadle, Harris and Sluis-Cremer, a Mr. Martinson mentioned that a "new microscopes revealed 33% more particles than did the old ones…" Beadle, D.G., E. Harris & G.K. Sluis-Cremer. The relationship between the amount of dust breathed and the incidence of silicosis: An epidemiological study of South African European gold miners. in Shapiro, H.A., ed.: Pneumoconiosis. Proceedings of the International Conference. Johannesburg 1969. Oxford: Oxford University Press, pp. 473-477 (1969) (discussion at p. 480). We do not know whether Beadle's estimates were affected by the "old microscopes" that appeared to miss a significant number of particles, but it certainly is possible that the exposure estimates used by Hnizdo and Sluis-Cremer (1993) were understated for that reason as well.

as Hnizdo and Sluis-Cremer assumed. But, even if they were understated by a factor of just 2, the resulting impact on estimated risks could be by a factor of as much as 10.[339] Indeed, OSHA itself projects that the risk of silicosis based on Hnizdo and Sluis-Cremer (1993) is six times greater at an exposure level of 0.1 mg/m^3 (4.5 mg/m^3-years of cumulative exposure) than at an exposure level of one-half that concentration, *i.e.*, at a level of 0.05 mg/m^3 (2.25 mg/m^3-years of cumulative exposure).[340] Whatever the exact impact might be, it is clear that the study by Hnizdo and Sluis-Cremer (1993), in the words of the British Health and Safety Executive, "is limited by substantial weaknesses in the exposure estimates."[341] OSHA appears to agree, noting that "the need to rely on particle count data that was generated over a fairly narrow production period and the need to make assumptions about the quartz content of the dust to which workers were exposed does add uncertainty to the exposure estimates."[342] All of this was summed up well by the British Health and Safety Executive, which put the point this way:

> In view of the considerable uncertainties surrounding the exposure assessment the silicosis risk estimates [in Hnizdo and Sluis-Cremer (1993)] are considered relatively unreliable. It seems highly likely that early quartz

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339 See Hughes, J.M.., Radiographic Evidence of Silicosis in Relation to Silica Exposure. Appl. Occup. Environ. Hyg. 1995; 10(12): 1064-1069 (noting that a twofold underestimation of exposure in the Hnizdo *et al.* (1993) cohort "could account for more that a tenfold overestimation in risk"); Hughes, J. & Weill, H.., Letter to the Editor: Silicosis Risk: Canadian and South African Miners. Amer. Journal of Industrial Medicine. 1995; 27: 617-618.

340 See Health Effects Review at 351-52, Table II-12 (projecting a risk of 773/1,000 workers at an exposure level of 0.1 mg/m^3 and a risk of 127/1,000 workers at an exposure level of 0.05 mg/m^3).

341 British HSE Phase 1 Report at 48.

342 Health Effects Review at 332. See also *id.* at 341 (noting "it must be acknowledged that there is uncertainty in the exposure estimates of the South African miner study").

> exposures in this study were underestimated probably by 2-3 fold. Hence, while it is clearly apparent that there was a high proportion of silicosis cases (1/1+) among this workforce, it seems fairly probable that the quantitative predictions of risk in relation to cumulative exposure to respirable quartz are likely to have been overestimated.[343]

For the foregoing reasons, Hnizdo and Sluis-Cremer (1993) does not provide a reliable basis for projecting risks of silicosis morbidity and should not be used for that purpose.[344]

Steenland and Brown (1995): As in the case of Hnizdo and Sluis-Cremer (1993), the exposure assessment in the study of Homestake gold miners by Steenland and Brown (1995)[345] suffers from enormous uncertainty and a high likelihood of underestimation. Job-exposure matrices expressed in terms of respirable silica mass per cubic meter were developed on the basis of particle count measurements taken in the years 1937-1975. The particle count values were converted to respirable mass using a conversion factor. For the years prior to 1937 (when a significant number of cohort members were first exposed), no exposure measurements were available; instead, based on estimates by industrial hygienists, exposures were assumed to average 25 million particles per cubic foot ("mppcf") prior to 1920, and the estimate was decreased gradually from 1920-1937 as a function of decreased

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343 British HSE Phase 1 Report at 49.

344 Another reason to question silicosis risk estimates based on Hnizdo and Sluis-Cremer (1993) is that the radiographs in this study were interpreted by a single reader. See Health Effects Review at 331. As OSHA notes, "Finkelstein (2000) also pointed to uncertainties in the exposure estimates as well as potential uncertainty introduced by the radiographs having been read by a single reader." *Id.* at 332.

345 Steenland, K. and Brown, D., Mortality Study of Gold Miners Exposed to Silica and Nonasbestiform Amphibole Minerals: An Update With 14 More Years of Follow-Up. American Journal of Industrial Medicine. 1995; 27: 217-229; Steenland, K. and Brown, D., Silicosis Among Gold Miners: Exposure-Response Analyses and Risk Assessment. Am J. Public Health. 1995; 85:1372-1377.

time spent underground.[346] As NIOSH observes, the lack of dust measurements before 1937 could have affected risk estimates for this cohort.[347] This is particularly true because 92% of the "silicotic" miners in the study were exposed prior to 1937, with an average of 50% of their work history occurring prior to that year. In fact, the mean year of first exposure for the "cases" was 1926, and, as OSHA notes, "exposures [in that earlier period] were likely higher than in more recent years."[348] That is an understatement. According to estimates of the study's authors, the median average exposure level of men hired prior to 1930 was more than seven times higher than the median average exposure level of men hired after 1950 (0.15 mg/m^3 versus 0.02 mg/m^3).[349]

Particle count measurements were taken in each year from 1937 to 1975. However, the study's authors apparently had no exposure measurements or job history data for the years after 1975, so they assigned zero exposure to the post-1975 period, even though 15% of the cohort was still employed at that time.[350] In addition, as more work was performed above ground over the years, a decreasing weighting factor was applied to the exposures compared to underground work – on the apparent assumption that above-ground jobs were unexposed. Yet primary crushing of the ore was transferred above ground in the mid-1930s, and an

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346 *See* Steenland, K. and Brown, D., Silicosis among Gold Miners: Exposure-Response Analyses and Risk Assessment. American Journal of Public Health. 1995; 85(10):1372-1377.

347 See NIOSH, Hazard Review: Health Effects of Occupational Exposure to Respirable Crystalline Silica (2002) at p. 32, Table 13.

348 78 Fed. Reg. at 56309.

349 *See* Steenland, K and Brown, D., Mortality Study of Gold Miners Exposed to Silica and Nonasbestiform Amphibole Minerals: An Update With 14 More Years of Follow-Up. American Journal of Industrial Medicine. 1995; 27: 217-229 at 221.

350 *See id.* at 219.

industrial hygiene survey performed by NIOSH in 1977 indicated that workers engaged in crushing operations at the surface had higher time-weighted average dust exposures than underground workers.[351] As noted by the authors of the NIOSH survey: "Many of the employees in the surface crushing mills are subject to high dust concentrations during various work activities."[352]

The particle count values from 1937 to 1975 were converted to respirable silica mass by use of a conversion factor of 10 mppcf = 0.1 mg/m^3 of respirable silica. This conversion factor was *not* based on side-by-side comparisons of samples taken at the Homestake mine. Instead, the authors used a conversion factor of 10 mppcf = 0.075 mg/m^3 proposed by Davis *et al*. (1983) for Vermont granite workers[353] and increased it by the ratio of 13/9.5 to reflect what they believed was the respirable silica content of the dusts at the Homestake mine and in the Vermont granite industry, respectively.[354] There are substantial questions about use of this conversion factor:

- First, use of 10 mppcf = 0.075 mg/m^3 respirable silica proposed by Davis *et al*. as the starting point for a conversion factor at the Homestake mine is problematic. Three different conversion factors have been proposed for the Vermont granite industry. In addition to the factor proposed by Davis, Sutton and Reno proposed a conversion factor of 10 mppcf = 0.1 mg/m^3 respirable silica,[355] and Ayer *et al*. found a conversion factor of 10 mppcf = 0.2 mg/m^3 of respirable silica based on

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[351] *See* Zumwalde, R.D., *et al*., *Industrial Hygiene Report: Homestake Mining Company, Lead, South Dakota* (Final Report January 30, 1981), p. 52, Table 23, p. 53.

[352] *Id*. at 78.

[353] Davis, LK, *et al*. (1983). Mortality Experience of Vermont Granite Workers. *Am. J. Indus. Med.* 4:705-723.

[354] *See* Steenland, K. and Brown, D., Silicosis among Gold Miners: Exposure-Response Analyses and Risk Assessment. American Journal of Public Health. 1995; 85(10):1372-1377.

[355] *See* Davis, *et al*., Table VIII (presenting data from Sutton & Reno).

side-by-side comparisons of granite dust generated in operations at a reconstructed 1920s shed.[356] After reviewing the history of studies correlating particle count with respirable mass and assessing geometric considerations, NIOSH recommended that "unless there are other compelling data to support a different" value, a conversion factor of 1 mppcf = 0.1 mg/m^3 respirable dust should be used.[357] OSHA adopted that recommendation shortly thereafter and uses it today; indeed, OSHA has applied that conversion factor in this very rulemaking.[358] For respirable dust having a silica content of 13% (as Steenland and Brown assumed was the case in the Homestake gold mine), rather than the 9.5% silica content that Steenland and Brown attributed to Vermont granite dust, this translates into a conversion factor of 10 mppcf = 0.137 mg/m^3 respirable silica, a value 37% higher than the value used by Steenland and Brown.

- Second, the adjustment to the Vermont granite conversion factor that Steenland and Brown made based on an assumed 13% silica content of the dust at the Homestake mine is questionable. The 13% figure is an average of 82 samples (ranging from 1% to 48%) taken in two surveys in the 1970s. The authors do not know whether the percentage of respirable quartz in the dust differed in earlier years, which, as NIOSH points out, could have affected their risk assessment results.[359] And their sensitivity analysis showed that if they underestimated the percentage of quartz, it would have a bigger impact on their risk estimates than if they overestimated the percentage of quartz by the same amount.[360]

- Third, it seems doubtful that a conversion factor derived from measurements of granite dust can properly apply (even with a quartz content adjustment) to a gold

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[356] Ayer, H.E., *et al.* (1973). A Monumental Study -- Reconstruction of a 1920 Granite Shed. *Am. Indus. Hygiene Assn. J.* 206-211.

[357] NIOSH, Recommended Conversion Factors to Derive mccpf Equivalents from Samples of Silica-containing Dusts using the Gravimetric Method, April 2000.

[358] See September 4, 2001 Memorandum for Regional Administrators and Silica Coordinators from Richard E. Fairfax, Director of OSHA's Directorate of Compliance Programs (directing OSHA regional offices to apply "a conversion factor of 0.1 mg/m^3 per mppcf . . . when converting between gravimetric sampling and particle count sampling results for silica-containing dust."); 78 Fed. Reg. at 56445 (using a conversion factor of 1 mppcf = 0.1 mg/m^3 respirable dust to characterize exposures in construction and shipyard operations).

[359] See NIOSH, Hazard Review: Health Effects of Occupational Exposure to Respirable Crystalline Silica (2002) at p. 32, Table 13.

[360] *See* Steenland, K. and Brown, D., Silicosis among Gold Miners: Exposure-Response Analyses and Risk Assessment. American Journal of Public Health. 1995; 85(10):1372-1377.

> mining operation where the rock is different, the operations performed are different, the particle size distribution probably is different, etc. As NIOSH stated in its 1974 Criteria Document in explaining the particle count-to-gravimetric conversion factor based on Vermont granite industry studies: "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."[361]

Based on the foregoing approach to exposure assessment, Steenland and Brown stated that for the cohort as a whole, the median intensity of exposure to silica was 0.15 mg/m^3 for men hired before 1930, 0.07 mg/m^3 for men hired between 1930 and 1950, and 0.02 mg/m^3 for men hired after 1950.[362] And they assumed zero exposure after 1975 even though 14-15% of cohort members continued to work at the mine after 1975 when the average RCS exposure level based on Mine Safety and Health Administration (MSHA) compliance sampling at the Homestake facility was in the range of 0.06 mg/m^3 to more than 0.07 mg/m^3.[363] For the reasons discussed above, these exposure values are suspect. Moreover, a Health Hazard Evaluation conducted at the Homestake mine in March 1978 by NIOSH found that respirable silica exposures for all six personal samples taken in the assay department exceeded the NIOSH REL, the OSHA PEL, and the TLV for quartz—with values ranging from 0.15 mg/m^3 to 1.33 mg/m^3.[364] In a follow-up survey conducted in May 1978 (after the

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[361] NIOSH, Doc. No. 75-120, Criteria for a Recommended Standard: Occupational Exposure to Crystalline Silica, p. 75 (1974).

[362] *See* Steenland, K and Brown, D., Mortality Study of Gold Miners Exposed to Silica and Nonasbestiform Amphibole Minerals: An Update With 14 More Years of Follow-Up. American Journal of Industrial Medicine. 1995; 27: 217-229.

[363] Calculations made by K. Bailey of Vulcan Materials Company show that the average exposure level in 1978 based on the MSHA compliance data was 0.073 mg/m^3, while for 686 MSHA samples collected from 1978 – 2001, the average exposure level was 0.061 mg/m^3. Personal communication from K. Bailey, January 23, 2014.

[364] *See* Health Hazard Evaluation Report, Homestake Mining Company (HHE 78- 034-930) at 1, 10 & Table 5.

company had implemented various of NIOSH's engineering recommendations), two of the four respirable silica samples still exceeded 0.1 mg/m^3, and all four exceeded 0.05 mg/m^3—ranging from 0.07 mg/m^3 to 0.24 mg/m^3.[365] While these samples related to assay department workers rather than miners, the fact that they ranged from 0.15 mg/m^3 to 1.33 mg/m^3 on the initial survey in 1978 makes one skeptical of the much lower values that Steenland and Brown used for miners employed during the preceding 50-60 years.[366] Furthermore, an industrial hygiene survey conducted by NIOSH in 1977 concluded that "before approximately 1952 there is an additional risk of over exposure to free silica [TWA > 0.1 mg/m^3] because of the elevated dust concentrations caused by underground blasting procedures and the lack of efficient dust suppressive techniques."[367] In light of these data, Steenland and Brown's estimate that the median intensity of exposure for men hired between 1930 and 1950 was 0.07 mg/m^3 and for those hired after 1950 it was 0.02 mg/m^3 hardly seems credible.

In sum, contrary to OSHA's belief, the exposure assessment for the Homestake gold mine cohort is of questionable quality. As the British Health and Safety Executive observes: "The exposure assessment for this study was weak and was based on a number of

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365 See id.

366 As noted above, alculations made by K. Bailey of Vulcan Materials Company show that the average exposure level in 1978 based on the MSHA compliance data was 0.073 mg/m^3, while for 686 MSHA samples collected from 1978 – 2001, the average exposure level was 0.061 mg/m^3. Personal communication from K. Bailey, January 23, 2014.

367 See Zumwalde, R.D., et al., Industrial Hygiene Report: Homestake Mining Company, Lead, South Dakota (Final Report January 30, 1981), p. 77. See also id., pp. 14-25.

unverifiable assumptions."[368] Indeed, OSHA itself identifies weakness of the exposure assessment as a major limitation of this study.[369] Moreover, the exposure assessment used by Steenland and Brown (1995) almost certainly understates the silica exposures of cohort members. Indeed, if the exposure data were accurate – and if OSHA's view as to the association between silica exposure and increased lung cancer risk is correct – the study should have found a positive exposure-response trend for lung cancer, particularly since there was an excess of lung cancer in the cohort and, as the authors state, "[l]evels of exposure to crystalline silica in our study were high."[370] Yet a variety of different analyses failed to find a relation between lung cancer risk and exposure to silica dust. In fact, a nested case-control analysis "revealed a negative nonsignificant trend with either estimated cumulative dust exposure or the log of estimated dust exposure."[371] Thus, OSHA's confidence in the "quality of underlying exposure and job history information"[372] in the study of Homestake gold miners cannot be reconciled with its own theory of silica-related lung cancer.

Furthermore, there are problems with silicosis ascertainment in this study. The 170 silicosis cases were identified through a mix of death certificates and two cross-sectional radiological surveys in 1960 and 1976. The majority of cases (128 out of 170) came from

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368 British HSE Phase 1 Report at 66.

369 See Health Effects Review at 341 (contrasting the high quality of the exposure assessment of the British coalworker cohort to the poor quality of the exposure assessments in Steenland and Brown (1995) and Chen *et al.* (2001) and Chen *et al.* (2005)).

370 Steenland, K. and Brown, D., Mortality Study of Gold Miners Exposed to Silica and Nonasbestiform Amphibole Minerals: An Update With 14 More Years of Follow-Up. American Journal of Industrial Medicine. 1995; 27: 217-229 at 227.

371 *Id.* at 223.

372 Health Effects Review at 357.

death certificates alone, 29 came from x-rays only, and 13 were identified both ways. As the British Health and Safety Executive notes, diagnosis by death certificate in this study "was fraught with interpretational problems and very likely subject to bias," as "[t]he death certificate information was not backed up by autopsy data, and a physician's diagnosis on the death certificate may well have been influenced by knowledge of past occupational history [or workman's compensation claims]. Hence, the assumption that silicosis could be reliably diagnosed by death certificate is a major weakness in the study."[373] Moreover, as the authors themselves note, some deaths due to chronic obstructive pulmonary disease may have been "misdiagnosed as silicosis."[374] And some of the 5 cases of silicosis with a low cumulative exposure (< 0.2 mg/m^3-years) may have been due to silica exposure "before or after working at the gold mine."[375] Furthermore, examining risks using a life table approach, as the authors did, is invalid because the observations in the study were of prevalence, not incidence, and the dates of incidence were unknown.

In the words of the British Health and Safety Executive, "there are too many weaknesses associated with this study to permit any confident predictions of the risk of

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[373] British HSE Phase 1 Report at 49, 66.

[374] Steenland, K. and Brown, D., Silicosis among Gold Miners: Exposure-Response Analyses and Risk Assessment. American Journal of Public Health. 1995; 85(10):1372-1377 at 1373. According to OSHA, this happened in South Africa as well. See 78 Fed. Reg. at 56308 (discussing Wyndham *et al.* (1986)).

[375] Steenland and Brown (1995), *supra*, at 1375.

silicosis in relation to cumulative exposure."[376] Accordingly, "no confidence can be attached to the predicted risk estimates from this study."[377]

Chen *et al.* (2001): The study of Chinese tin miners by Chen *et al.* (2001) suffers from what the British Health and Safety Executive describes as "major limitations in the exposure assessments."[378] These limitations also apply to the subsequent study of Chinese tin and tungsten miners and pottery workers by Chen *et al.* (2005) – on which OSHA also relies – because RCS exposure estimates of cohort members in the latter study "were based on the same data as described by Chen et al. (2001)."[379]

The exposures assigned to cohort members in this study were based on Chinese total dust ("CTD") samples collected by high volume, short duration *area* samplers that "were typically run during active working periods at a flow rate of 25 l/min, for about 15–20 minutes."[380] As the British Health and Safety Executive points out, the relation of these area samples:

> to personal inhalation exposures is unknown. No precise information was provided concerning the nature of the mining work, but in these underground Chinese tin mines it is likely to have involved (at least to some extent) hand-held tools with workers being in close proximity to the sites of dust generation. The area samplers (said to be placed in three monitoring stations in each mine) may well have underestimated personal exposures, but to what degree is unknown.[381]

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376 British HSE Phase 1 Report at 50.

377 *Id.* at 66.

378 *Id.* at 51.

379 Health Effects Review at 339.

380 Chen, W. *et al.* (2001). Exposure to silica and silicosis among tin miners in China: exposure-response analyses and risk assessment. Occup Environ Med 2001;58:31–37 at 32.

381 British HSE Phase 1 Report at 51.

The area sampling results expressed in terms of CTD then had to be converted to gravimetric levels of respirable crystalline silica. To do so, the authors conducted side-by-side sampling to develop a "few comparative workplace measurements in 1988–9."[382] They then proceeded as follows:

> The conversion factor at each facility [where side-by-side sampling was conducted] was obtained by averaging the ratios of the concentrations of respirable crystalline silica to that of the Chinese total dust over sampling sites within the facility, and the industry wide conversion factor was obtained by taking the same average over all sampling sites for facilities within the industry.[383]

This resulted in an RCS/CTD conversion factor of 3.6%, which the authors applied across the board to assign RCS exposures to all jobs in all facilities during all periods. But, as the British Health and Safety Executive observes, "how much mine-to mine or day-to day variation surrounds this figure is uncertain. The percentage is likely to be highly variable, depending for example, on whether workers encounter a sandstone seam or the metal ore itself."[384]

Furthermore, as the British Health and Safety Executive points out, "[n]o information was provided on the composition of the remaining ~96% of the total airborne mixed dust, but presumably much of it was tin oxides. Given the high cumulative CTD exposures, the radiographic findings in these workers possibly reflect a certain amount of mixed dust fibrosis, rather than opacites purely due to crystalline silica."[385] Beyond that: "No

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382 Chen, W. *et al.* (2001), *supra,* at 36.

383 *Id.* at 33.

384 British HSE Phase 1 Report at 51.

385 *Id.*

information on general hygiene conditions in the mines was given, but the possibility that workers lived all their lives in dusty mining areas, taking home workplace dust on their work clothes cannot be ignored. Such factors may lead to an apparent overestimate of the risks attributed to silica."[386] For all these reasons, to quote the British Health and Safety Executive: "The level of uncertainty in the cumulative exposure indices for respirable quartz is high, and how the data relate to personal inhalation exposures for the workers is unknown."[387] Indeed, OSHA itself apparently views the exposure assessment in the Chinese mining and pottery studies with considerable skepticism, stating that "Chen et al. (2001) and Chen et al. (2005) relied on short-term total dust samples, also with limited side-by-side sampling, to estimate exposures to respirable quartz dust."[388]

In a subsequent study that OSHA also references in its risk assessment for silicosis morbidity, Chen *et al.* (2005) examined silicosis risks in cohorts of Chinese tin miners, tungsten miners, and pottery workers.[389] This time, the silicosis risks were lower than what had been predicted in the 2001 study of tin miners. Moreover, in the 2005 study, tin miners had lower mean cumulative RCS exposures than tungsten miners (2.4 mg/m^3-years versus 3.2 mg/m^3-years) and a higher percentage of particle surface area occlusion than tungsten miners (18% versus 13%); yet, by OSHA's estimate, the silicosis risk for tin miners was

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386 *Id.* at 51-52.

387 *Id.* at 66.

388 Health Effects Review at 341.

389 Chen, W. *et al.* (2005). Risk of Silicosis in Cohorts of Chinese Tin and Tungsten Miners, and Pottery Workers (I): An Epidemiological Study. Am. J. of Indust. Medicine. 48:1-9.

almost three times higher than the risk for tungsten miners.[390] That is the opposite of what one would expect if the exposure assessments for the Chinese miner cohorts were correct and if other problems with the studies did not exist. To quote OSHA: "There is no apparent explanation for why tungsten miners appeared to have lower silicosis risk than tin miners . . . . It is possible that the difference in observed exposure-response relationships seen among tungsten and tin miners reflects exposure misclassification due to the need to estimate full-shift exposures to respirable quartz from 342 short-term total dust samples, or that the difference is the result of unidentified workplace-specific factors that influenced the relative toxicities of the quartz particles found in the tungsten and tin mines."[391] At the same time, OSHA projects more than a five-fold difference in silicosis morbidity risks for the same exposure across the tin mine, tungsten mine, and pottery industries examined by Chen *et al.* (2005).[392] As Dr. Cox observes, this "suggests that silicosis morbidity risk is not well explained by estimated cumulative exposure [in this study], but reflects effects of other variables."[393]

In sum, the studies by "Chen et al. (2001) and Chen et al. (2005) relied on short-term total dust samples, also with limited side-by-side sampling, to estimate exposures to respirable quartz dust."[394] In these studies, as in Hnizdo and Sluis-Cremer (1993) and Steenland and Brown (1995), "one of the principal sources of uncertainty," as OSHA notes,

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390 See Health Effects Review at 340.

391 *Id.* at 341-42.

392 See *id.* at 351-52, Table II-12.

393 Cox Comments at 99.

394 Health Effects Review at 341.

"is the estimation of respirable silica exposures of the cohorts."[395] The resulting exposure assessments used by Chen *et al.* (2001) and Chen *et al.* (2005) are unreliable, and the projections of silicosis risk are internally inconsistent. Like Hnizdo and Sluis-Cremer (1993) and Steenland and Brown (1995), these studies would not provide a reliable basis for estimating the risk of silicosis at the current general industry PEL of 100 $\mu g/m^3$ even if there were not an exposure concentration threshold for silicosis above that level.

The study of British coal workers by Buchanan *et al.* (2003)[396] is the study that OSHA views as providing "the most reliable basis for estimating silicosis morbidity risk due to the high quality of the underlying exposure data for the cohort, and the capability of the Buchanan et al. model to account for possible effects of exposures to very high concentrations of respirable silica."[397] That may be so, but the study does not provide a reliable basis for projecting silicosis morbidity risks at exposure concentrations of 100 $\mu g/m^3$ and below. In particular, as noted above, the data and modeling employed by Buchanan *et al.* (2003) do not rule out the existence of a concentration threshold above 100 $\mu g/m^3$ for silicosis morbidity.[398] To the contrary, the fact that the odds ratios for silicosis were statistically significant only for post-1964 cumulative exposures (which reflected much

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395 *Id.* at 357.

396 Buchanan, D. *et al.* (2003). Quantitative relations between exposure to respirable quartz and risk of silicosis. Occup Environ Med. 60:159–164.

397 Health Effects Review at 342. See also *id.* at 341 ("Of these studies, OSHA believes that the study of coalworkers by Miller et al. (1995, 1998) and Buchanan et al. (2003) is of the best overall quality, in large part due to the availability of respirable quartz measurements taken over several years that provided the basis for estimating exposures of individual cohort members.").

398 See pp. 91-92, 94-95, *supra.*

higher mean exposure concentrations than the pre-1964 exposures)[399] "suggests the potential practical importance of an exposure threshold in the range of historical exposures that were larger than those now permitted" under the current 100 μg/m^3 PEL for general industry.[400] Those post-1964 exposures included a substantial amount of work performed in the mine during a period between early 1971 and mid 1976, during which the miners "experienced 'unusually high concentrations of freshly cut quartz in mixed coalmine dust.'"[401] Indeed, "air levels of silica [were] such that quarterly mean exposures [of workers at the high-quartz seam] exceeded 1 mg/m^3 (10% of the quarterly measurements were over 10 mg/m^3)."[402] Those levels are 10 to 100 times as high as the current general industry PEL of 100 μg/m^3. In fact, the effective disparity is even greater than that, because long-term mean exposures must be maintained at a level significantly below the 100 μg/m^3 PEL in order for employers to be reasonably confident that they are in compliance with a PEL of 100 μg/m^3 95% of the time.[403] Particularly since the odds ratios for pre-1964 cumulative RCS exposures were not statistically significant (during a period when mean RCS exposures were lower than in the subsequent period), the high average quartz concentrations to which the workers were exposed for substantial amounts of time after 1964 (when the odds ratios for cumulative exposure were significant) suggests that a concentration threshold above 100 μg/m^3 may very well exist. Consistent with that notion, the British Health and Safety Executive noted:

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399 See Health Effects Review at 335.

400 Cox Comments at 98.

401 See Health Effects Review at 333.

402 *Id.* at 334.

403 See p. 22-24, *supra*.

"Another limitation with this study is the fact that there were few individuals with silicosis at low cumulative exposures, and therefore few data points near the beginning [*i.e.*, the lower end] of the exposure-response curve."[404] Hence, it would have been difficult to investigate and determine the existence of a threshold at the lower end of the estimated exposure-response curve – *e.g.*, in the region between the current PELs for general industry (100 $\mu g/m^3$) and construction (250 $\mu g/m^3$) – in this study.

Furthermore, even if the concentration threshold for radiological silicosis were below 100 $\mu g/m^3$, the British coal worker study by Buchanan *et al.* (2003) would produce overestimates of the risk of silicosis. Thus, as OSHA notes at 78 Fed. Reg. at 56336:

> Using medical and exposure data taken from a cohort of heavy clay workers first studied by Love et al. (1999), Miller and Soutar (2007) compared the silicosis prevalence within the cohort to that predicted by the exposure-response model derived by Buchanan et al. (2003) and used by OSHA to estimate the risk of radiologic silicosis with a classification of ILO 2+. They found that the model predicted about a 4-fold higher prevalence of workers having an abnormal x-ray than was actually seen in the clay cohort (31 cases predicted vs. 8 observed).[405]

For the foregoing reasons, the study by Buchanan *et al.* (2003) does not provide a basis for making reliable projections of silicosis morbidity risks at exposure levels of 100 $\mu g/m^3$ and below. This is particularly true if the study is used to estimate risks for workers outside the specific cohort on which the study was conducted – because, in addition to the exceptionally high mean RCS exposures experienced by many cohort members, the crystalline silica in the Buchanan *et al.* (2003) study was freshly fractured from massive sandstone, which would make it more biologically active than in settings where the silica has

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404 British HSE Phase 1 Report at 64.

405 78 Fed. Reg. at 56336.

had time to age.[406] Also, it is possible that the significant amounts of respirable coal dust to which the miners were exposed may have degraded their lung defense mechanisms, making them more susceptible to pneumoconioses like silicosis.

E. OSHA's Risk Assessment for Renal Disease Mortality Is Neither Well Supported Nor Robust and Cannot Support a Finding of Significant Risk.

OSHA projects that after 45 years of occupational exposure to crystalline silica at the current general industry PEL of 100 $\mu g/m^3$, a worker's excess risk of renal disease mortality is 39/1,000 and that at the proposed PEL of 50 $\mu g/m^3$, the risk would be 32/1,000.[407] This amounts to a reduction in estimated risk of 18 percent, which, given the exceptionally large uncertainty surrounding these estimates, is of questionable significance. The 95% confidence intervals for OSHA's risk estimates are enormous (2-200 for a PEL of 100 $\mu g/m^3$ and 1.7-147 for a PEL of 50 $\mu g/m^3$), and even OSHA concedes that its estimates of renal disease mortality (based on only 50 deaths) are "less robust" than its other risk estimates.[408] This modesty is well warranted. Indeed, in a report commissioned by OSHA, the lead author of the pooled analysis on which OSHA bases its risk estimate for renal disease mortality candidly states that the "amount of data [that he and his colleagues analyzed to evaluate the risk of renal disease mortality] is insufficient to provide robust estimates of risk."[409] That

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406 See Soutar C.A., *et al.* (2004). Dust concentrations and respiratory risks in coalminers: Key risk estimates from the British Pneumoconiosis Field Research. Occup Environ Med 61:477-481. OSHA-2010-0034-1122Soutar, *et al.* (2004).

407 See Health Effects Review at 316, 356-57.

408 *Id.* at 357.

409 Steenland, N.K. & Bartell, S.M. Silica Exposure: Risk Assessment for Lung Cancer, Silicosis and Other Diseases. Prepared under contract to OSHA by ToxaChemica International, Inc. (Draft Final, December 7, 2004) at 27. Docket ID: OSHA-2010-0034-0469.

acknowledgment, however, did not deter OSHA from including high estimates of excess renal disease mortality in its "significant risk" determination and in its calculation of expected benefits of the rule. Those actions are unwarranted.

1. There Is Serious Doubt as to the Association of Silica Exposure and Renal Disease Mortality.

To begin with, there is a serious question whether silica exposure causes renal disease mortality at all. Investigators have examined a possible association between silica exposure and renal disease mortality in a number of studies, with decidedly mixed results (most tending to the negative).

Thus, in an update of the study of North American industrial sand workers by J. McDonald, *et al.* (2005), there was no relation between chronic renal disease (nephritis/nephrosis) or renal cancer and cumulative silica exposure; in fact, the trends were opposite (*i.e.*, decreasing odds ratios with increasing cumulative exposure) for both diseases even though excess mortality from both diseases was found in the cohort.[410] Similarly, in a comprehensive mortality study of Vermont Granite workers by P. Vacek *et al.* (2011), SMRs for kidney cancer or non-malignant kidney disease (nephritis and nephrosis) were not significantly elevated.[411] The authors also conducted a nested case-control analysis in which conditional logistic regression was used to model the relationship between mortality and each of the exposure variables (cumulative exposure, exposure duration, and average exposure intensity). No statistically significant associations were observed between exposure to

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[410] McDonald, J. *et al.*, Mortality from Lung and Kidney Disease in a Cohort of North American Industrial Sand Workers: An Update. Ann Occup Hyg. 2005; 49(5): 367-73.

[411] Vacek, P. *et al.*, Mortality in Vermont granite workers and its association with silica exposure. Occup Environ Med. 2011; 68:312-318. Available on-line at http://dx.doi.org/10.1136/oem.2009.054452.

respirable crystalline silica and mortality from kidney cancer or non-malignant kidney disease. This was true of all three of the exposure metrics (cumulative, average, and duration), whether expressed as a continuous variable or a categorical variable divided into quintiles of the distribution of cases and controls combined. The authors concluded that the results of their study yielded no indication of an association between silica exposure and mortality from either kidney cancer or non-malignant kidney disease, even though their study had a substantially larger number of deaths from these diseases (34) than other studies.[412]

The negative findings of Vacek *et al.* (2011) are consistent with an earlier mortality study of Vermont granite workers by Davis *et al.* (1983), which found no relationship between mortality from genitourinary system diseases and cumulative silica exposure.[413] Koskela *et al.* (1987) reported a similar finding among Finnish granite workers.[414] And, as OSHA points out: "Both Carta et al. (1994) and Cocco et al. (1994) reported finding no increased mortality from urinary tract disease among workers in an Italian lead mine and a zinc mine."[415]

A recent update of the Stoke-on-Trent pottery workers cohort by N. Cherry *et al.* (2012) examined mortality risks by underlying cause for the full period 1985-2008 and

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412 OSHA has raised several objections to the Vacek *et al.* study's conclusions regarding the absence of an association between silica exposure and lung cancer in the cohort. Those objections, as shown above (see pp. 40-47, *supra*), are misguided and completely unfounded. In any event, OSHA has not suggested that its criticisms of Vacek *et al.* (2011) apply to the study's findings with regard to renal disease mortality.

413 See Davis, L. *et al.*, Mortality Experience of Vermont Granite Workers. American Journal of Industrial Medicine. 1983; 4: 705-723.

414 See Koskela R.S., *et* al. (1987). Mortality and disability among granite workers. Scand J Environ Health 13:18–25. OSHA-2010-0034-0363.

415 78 Fed. Reg. at 56309.

separately for the early period (1985-1992) and the later period (1993-2008) and by "any mentioned" cause for the later period. They also performed an internal dose-response analysis for the portion of the cohort for whom they had exposure information. Among the outcomes examined was mortality from chronic non-malignant renal disease (cNMRD). In an "any mention" mortality analysis for lung cancer, COPD, and cNMRD, the authors found that: "Overall there is little indication of increased risk with longer duration of exposure. . . ."[416] Moreover, no relation was seen, in the early or late time periods, between mean concentration and cNMRD. And, in additional analyses, cumulative exposure was unrelated to any of the outcomes of interest in either period. *Id.*

A large-scale mortality study of 17,644 medical surveillance participants in the German porcelain industry by T. Birk *et al.* also found that death from renal cancers and from non-malignant renal disease was not associated with employment or silica-exposure surrogates when the analysis used either the German population or the Bavarian population as referents.[417] In a subsequent analysis of this cohort, the authors found that cumulative exposure to respirable silica was not statistically significantly associated with mortality from kidney cancer, renal disease, or any other cause of death other than silicosis after adjusting for age, smoking history, and duration of employment.[418]

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416 Cherry, N. *et al.*, Mortality in a cohort of Staffordshire pottery workers: follow-up to December 2008. Occup. Environ. Med., published online October 26, 2012. Available on-line at http://oem.bmj.com/content/early/2012/10/25/oemed-2012-100782.full.html.

417 Birk, T. *et al.*, Mortality in the German Porcelain Industry 1985-2005: First Results of an Epidemiological Cohort Study. JOEM. 2009; 51, No. 3: 373-385.

418 Mundt, K. *et al.*, Respirable Crystalline Silica Exposure-Response Evaluation of Silicosis Morbidity and Lung Cancer Mortality in the German Porcelain Industry Cohort. JOEM 2011; 53(3): 282-289.

K. Steenland *et al.* (2002) conducted a study of silicosis patients in three states to determine whether they have an increased incidence of renal disease.[419] These patients had a mean of 48 years since first exposure to the end of follow-up, and their silica exposures appear to have been quite high, since a significant percentage of them had progressive massive fibrosis. Even so, the standardized incidence ratio (SIR) for end-stage renal disease (ESRD) was not elevated (SIR 0.77) in the analysis in which person-time began at the time of first exposure. Nor was there a significant trend for ESRD by duration of exposure.[420] The rate ratio for ESRD also was not significantly elevated when the analysis assumed that person-time began at the date of entry into the silicosis register. These results are not consistent with the hypothesis that silica exposure increases the risk of renal disease.

A similar finding was made by K. Rosenman *et al.* (2000) in a study of hospital-based silicosis surveillance records where cohort members were likely to have more advanced silicosis, as indicated by their hospitalization (29% had PMF), so the study would overestimate the prevalence of kidney disease among silicotics. Even so, although these silicotics were found to be more likely to have elevated serum creatinine levels than matched controls, there was no relationship between duration of exposure to silica or profusion of scarring on chest X-rays (surrogate measures of silica exposure) and prevalence of kidney disease or elevated creatinine levels.[421] And those with kidney disease or elevated creatinine levels were less likely to have performed sandblasting, which is associated with particularly

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[419] Steenland, K. *et al.*, Silicosis and end-stage renal disease. Scand J Work Environ Health. 2002; 28(6): 439-442.

[420] The rate ratio for ESRD also was not significantly elevated when the analysis assumed that person-time began at the date of entry into the silicosis register.

[421] Rosenman, K.D., *et al.*, Kidney Disease and Silicosis. Nephron. 2000; 85:14-19.

high levels of airborne silica concentrations. This study, the authors acknowledged, "does not support a direct dose-related nephrotoxic effect of silica" – even though over 95% of cohort members had silica-related X-ray abnormalities and over 70% had at least 20 years of exposure to silica.

In a NIOSH-sponsored study, G. Calvert *et al.* (2003) performed a case-control analysis in which cases were subjects whose death certificate mentioned the disease of interest (e.g., autoimmune or renal disease), and controls (5 for each case) were subjects whose death certificate did not mention the disease or any of several diseases reported to be associated with silica exposure.[422] Subjects were assigned to a qualitative silica exposure category based on the industry/occupation pairing shown on the death certificate. The authors found no increased risk of mortality from renal diseases when the combined results for the medium, high, and super high estimated exposure categories were compared to the low/no exposure category; nor was there an increasing trend for renal disease mortality with increasing exposure (indeed, the opposite seems to have been true).[423]

In sum, there is ample reason to question whether silica exposure causes (or is even associated with) an increased risk of renal disease mortality. Indeed, in a recent publication, the principal author of the study on which OSHA bases its estimate of renal disease mortality acknowledges that the evidence that silica exposure causes renal disease is only

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[422] This may have biased the study in favor of finding an association because selecting controls on this basis means that they would be less likely to have had silica exposure.

[423] Calvert, G.M., *et al.*, Occupational silica exposure and risk of various diseases: an analysis using death certificates from 27 states of the United States. Occup. Environ. Med. 2003; 60:122-129.

"suggestive."[424] That should have deterred OSHA from unjustifiably attributing a high risk of renal disease mortality to silica exposures at the current general industry PEL of 100 μg/m^3.

2. The Studies OSHA Relies on Do Not Support its Risk Estimate for Renal Disease Mortality.

Acting on the questionable premise that silica exposure causes renal disease, OSHA projects that 45 years occupational exposure to crystalline silica at a concentration of 100 μg/m^3 will result in a 39/1,000 increased risk of renal disease mortality. It bases this estimate on an exposure-response coefficient that K. Steenland *et al.* (2002) developed by combining data from three cohorts – Homestake, North Dakota gold miners; U.S. industrial sand workers; and Vermont granite workers[425] – selected from the ten studies that were the subject of the pooled analysis of lung cancer risk by Steenland *et al.* (2001). As Dr. Peter Morfeld notes, the three cohort analysis for renal disease mortality "suffers from an unclear selection of cohorts. . . . There is no reason not to evaluate kidney disease mortality on the basis of all studies [from the ten cohort pooled analysis of lung cancer risk]."[426] These three studies purportedly were selected because they provided information on multiple cause mortality. But the listing of renal disease on an "any mention" basis, rather than as the underlying cause, cannot form a proper basis for estimating mortality risks from renal disease; hence, both OSHA and its contractor, ToxaChemica International, focus on underlying cause results

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424 Steenland, K. & Ward, E. Silica: A Lung Carcinogen. CA CANCER J CLIN 2013;00:00–00. Available on-line at http://dx.doi.org/10.3322/caac.21214 (first published on-line December 10, 2013).

425 Steenland, K., *et al.*, Pooled Analyses of Renal Disease Mortality and Crystalline Silica Exposure in Three Cohorts. Ann. Occup. Hyg. 2002; 46 (Supp. 1):4-9.

426 Morfeld Comment at 24.

in estimating mortality risk from renal disease. In these circumstances, as Dr. Morfeld observes, the selection of just three out of ten cohorts for the pooled analysis of renal disease mortality in Steenland *et al.* (2002) "raises a suspicion of study selection bias,"[427] and a systematic weight of evidence evaluation has not been provided to justify the selection of the three cohorts that were utilized.

Although there were only 50 renal disease deaths in the combined cohort from three studies – and although numerous studies, as noted above, have failed to find an association between silica exposure and renal disease – OSHA contends that the resulting risk estimates (though less than robust) are "credible given the large size of the pooled cohort study and quality of underlying exposure and job history information."[428] In fact, when one examines the three cohorts on which the Steenland *et al.* (2002) pooled analysis is based, one finds that OSHA's confidence in the "quality of underlying exposure and job history information" is misplaced and that the evidence for an excess risk of renal disease mortality at an average silica exposure level of 100 $\mu g/m^3$ is insufficient to support a finding of significant risk.

a. <u>Homestake Gold Miners</u>

As explained in Section II.D.3. above, the exposure assessment in the study of Homestake gold miners by Steenland and Brown (1995)[429] suffers from enormous

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427 *Id.* at 25.

428 Health Effects Review at 357. It should be noted that the study of Vermont granite workers by Vacek *et al.* (2011) had more deaths from renal disease than any of the three studies relied on by Steenland *et al.* (2002). Yet, as discussed above (see p. 89, *supra*), the authors found no indication of an association between silica exposure and mortality from either kidney cancer or non-malignant kidney disease in the cohort.

429 Steenland, K. and Brown, D., Mortality Study of Gold Miners Exposed to Silica and Nonasbestiform Amphibole Minerals: An Update With 14 More Years of Follow-Up. American Journal of Industrial Medicine. 1995; 27: 217-229; Steenland, K. and Brown, D.,

uncertainty and a high likelihood that exposures were underestimated. Having reviewed the issues in some detail at pages 123-130 above, there is no need to restate all the problems here. We would note, however, that the absence of any exposure measurements for the years prior to 1937 is likely to have had a particular impact on risk estimates for renal disease mortality – because, as OSHA notes, "most of the excess deaths [from renal disease] were concentrated among workers hired before 1930 when exposures were likely higher than in more recent years."[430] In fact, they likely were very much higher. As estimated by the study's authors, the median average exposure level of men hired prior to 1930 was more than seven times higher than the median average exposure level of men hired after 1950 (0.15 mg/m^3 versus 0.02 mg/m^3).[431] And only the men hired before 1930 showed a significantly elevated SMR for chronic renal disease – suggesting the existence of an average exposure threshold $\geq$0.15 mg/m^3 for any risk of silica-related renal disease mortality.

Steenland and Brown stated that for the cohort as a whole, the median intensity of exposure to silica was 0.15 mg/m^3 for men hired before 1930, 0.07 mg/m^3 for men hired between 1930 and 1950, and 0.02 mg/m^3 for men hired after 1950.[432] And they assumed zero exposure after 1975 even though 14-15% of cohort members continued to work at the

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Silicosis Among Gold Miners: Exposure-Response Analyses and Risk Assessment. Am J. Public Health. 1995; 85:1372-1377.

[430] 78 Fed. Reg. at 56309.

[431] *See* Steenland, K and Brown, D., Mortality Study of Gold Miners Exposed to Silica and Nonasbestiform Amphibole Minerals: An Update With 14 More Years of Follow-Up. American Journal of Industrial Medicine. 1995; 27: 217-229 at 221.

[432] *See* Steenland, K and Brown, D., Mortality Study of Gold Miners Exposed to Silica and Nonasbestiform Amphibole Minerals: An Update With 14 More Years of Follow-Up. American Journal of Industrial Medicine. 1995; 27: 217-229.

mine after that date. For the reasons discussed in Section II.C.3. above, these exposure values are not credible. OSHA's confidence in the "quality of underlying exposure and job history information"[433] in the study of Homestake gold miners is misplaced. The truth is that the exposure data underlying the analysis of renal disease mortality in this study is of questionable quality and almost certainly understates the silica exposures of cohort members. Just as "there are too many weaknesses associated with this study to permit any confident predictions of the risk of silicosis in relation to cumulative exposure,"[434] there are too many weaknesses to form credible estimates of renal disease mortality risks.

Furthermore, an elevation in mortality from chronic renal disease among Homestake gold miners was found only in men hired prior to 1930 when dust exposures were at their highest, while the SMR for chronic kidney disease was not statistically significantly elevated for the cohort as a whole or for men hired in either of the two later periods. Thus, if there is an association between silica exposure and renal disease mortality at all, the study of Homestake gold miners suggests there is a long-term average exposure threshold above 100 $\mu g/m^3$ separating the early highly exposed hires (whose median intensity of exposure to silica was estimated as 0.15 mg/m^3) from the rest of the cohort.

b. <u>North American Industrial Sand Workers</u>

The second study used in the Steenland *et al.* (2002) pooled analysis of renal disease mortality was a 2001 study of North American industrial sand workers by Steenland *et al.*

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433 Health Effects Review at 357.

434 British HSE Phase 1 Report at 50.

(2001)[435] While the authors of this study claimed to find an increased risk of renal disease mortality and end stage renal disease with increased cumulative exposure to silica, another contemporaneous study of North American industrial sand workers found no relation between end-stage renal disease or renal cancer and cumulative silica exposure; in fact, the trends were opposite.[436] In any event, the exposure estimates used in Steenland *et al.* (2001), as developed in Sanderson *et al.* (2000)[437], were highly uncertain and very likely understated – as illustrated by the fact that exposure estimates developed by Rando *et al.* for the largely contemporaneous study of North American industrial sand workers were considerably higher.[438] The limitations of the exposure estimates developed by Sanderson *et al.* (2000) are discussed in the attached report by Dr. Roy J. Rando.[439] They include the following:

- Sanderson *et al.* "collapsed the 18 plants into 4 categories and the 143 jobs into 10 categories, yielding 40 job/plant groupings in the exposure matrix. The collapsed job categories corresponded approximately to a department-based matrix and included quarrying, crushing, wet processing, drying, screening, milling, bagging and bulk loading jobs. Such a broad categorization of jobs certainly resulted in some exposure misclassification. This problem likely would

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435 Steenland, K. *et al.*, Kidney Disease and Arthritis in a Cohort Study of Workers Exposed to Silica. Epidemiology. 2001; 12:405-412.

436 See p. 88, *supra* (discussing McDonald, J. *et al.*, Mortality from Lung and Kidney Disease in a Cohort of North American Industrial Sand Workers: An Update. Ann Occup Hyg. 2005; 49(5): 367-73).

437 Sanderson, W. *et al.*, Historical Respirable Quartz Exposures of Industrial Sand Workers: 2000; 1946-1996. Am. J. Ind. Med. 38:389-398.

438 Rando RJ, R Shi, JM Hughes, H Weill, AD McDonald, and JC McDonald: Cohort Mortality study of North American industrial sand workers. III. Estimation of past and present exposure to respirable crystalline silica. Ann. Occ. Hyg. 45:209-216 (2001).

439 Rando, R.J., Estimates of Exposure to Crystalline Silica in Epidemiological Investigations of Industrial Sand Production Workers: Critical Review and Comparison of the Papers by Rando, et al. and Sanderson, et al., July 24, 2004 ("Rando Report"), submitted herewith as Attachment 7.

have been most significant in the milling, drying, and quarrying areas because of the inherently wide range in job tasks and resulting exposures. In the case of quarrying in particular, combining jobs in hard sandstone mining and wet sand dredging into the same category is clearly not warranted." Rando Report at 4-5.

- The only exposure data available to Sanderson *et al.* for the decades before 1974 came from a summary of particle count samples taken by Professor Theodore Hatch in a survey of 19 plants in 1946. But there were no individual plant identifiers in the survey, so plant-specific exposures could not be determined for the pre-1974 period. Rando Report at 5.

- Sanderson *et al.* converted the Hatch particle count data into gravimetric equivalents using a generic conversion factor of 1 mppcf = 100 μg respirable mass. As Dr. Rando points out, the applicability of this generic conversion factor across a broad range of industries is questionable "primarily due to the inherent assumption that airborne particle size distributions are the same, regardless of the industry and the processes used within it. This assumption must be regarded with considerable skepticism." Rando Report at 7. By contrast, Rando *et al.* "developed a specific conversion factor for [the industrial sand] . . . industry based on a set of respirable dust samples collected in a survey of several industrial sand plants in 1979." *Id.* Using that information and additional analyses, Rando *et al.* developed an industry-specific conversion factor of 1 mppcf = 276 $\mu g/m^3$ respirable mass for industrial sand facilities. "Clearly, because of potential differences in particle size distributions across differing industries and processes, an industry specific conversion factor such as that developed by Rando is preferable to a generic factor such as that utilized by Sanderson." Rando Report at 13.

- Rando *et al.* "conducted extensive research into the history of plant and process changes that may have altered dust emissions and worker exposure. . . . From these investigations, a compilation of common historical changes in each general process area of an industrial sand plant was developed. Specific dates or date ranges for such changes at each of the plants were then determined based on the interviews and documentary evidence." Rando Report at 8. Sanderson *et al.* conducted no such research. "Rather, they relied upon broad inferences of where and when changes might have occurred through statistical comparisons of modern and past exposure data. However, no specific information on what the putative change may have been or exactly when it occurred was available." *Id.*

- In the industrial sand industry, "the time period spanning approximately 1947 to 1974 saw the institution of many control measures aimed at reducing exposure to crystalline silica. Because of the lack of information on the history of implementation of such changes, Sanderson assumed a simple linear decrease in exposures over this time period. In contrast, Rando was able to use specific dates of implementation of such changes to decrease exposure estimates in a step-wise fashion, which is more likely to be representative of reality." Rando Report at 13.

- Exposures in the period before 1947 were considered to be constant – so that workers whose employment began prior to 1946 were deemed to be exposed at levels measured in 1946. And follow-up in the Steenland *et al.* (2001) study was through 1996, but work histories and exposure data collection ended in 1988, so late entries into the cohort may have had unaccounted for silica exposures during the eight years following 1988.

As can be seen, there are large uncertainties in the exposure assessment developed by Sanderson *et al.* (2000) and used in the analysis of renal disease mortality by Steenland *et al.* (2001). Moreover, in comparison to the more "hands-on" and plant-specific exposure assessment of Rando *et al.* (2001) – which OSHA appears to recognize is the superior exposure assessment[440] – the exposure values developed by Sanderson *et al.* for facilities in the same industry during comparable time periods were markedly lower, suggesting that Sanderson *et al.*'s exposure estimates and resulting Job Exposure Matrix not only were uncertain but also were understated.[441]

Furthermore, the SMRs for renal disease mortality in Steenland *et al.* (2001) were not impressively high. When evaluated in terms of *underlying cause*, the SMR for acute renal disease was not significant (95% CI: 0.70-9.86), and the SMR for chronic renal disease was just barely so (95% CI: 1.06-4.08). The study's authors, therefore, conducted no further analyses based on underlying cause mortality. Instead, their exposure-response analyses were based on *multiple-cause* mortality data, which encompassed all deaths with any

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440 See 78 Fed. Reg. at 56302 ("McDonald et al. (2001), Hughes et al. (2001), and Rando et al. (2001) had access to smoking histories, plant records, and exposure measurements that allowed for historical reconstruction and the development of a job exposure matrix. Steenland and Sanderson (2001) had limited access to plant facilities, less detailed historic exposure data, and used MSHA enforcement records for estimates of recent exposure.").

441 In Sanderson *et al.* (2000), the geometric mean of the samples for the period 1974 – 1996 was 25.9 ug/m^3; while in Rando *et al.*(2001), the geometric mean exposure for the period 1974 – 1998 was 42 ug/m^3. The actual differences were even greater than this, because Rando *et al.* took account of use of personal protective equipment after 1974, while Sanderson *et al.* did not.

mention of renal disease on the death certificate even where renal disease was not the underlying cause. There are a number of problems with use of multiple-cause data in mortality analyses, the most fundamental of which is that only the *underlying cause* data involve actual deaths from renal disease. As a matter of logic, then, only those data can be used to estimate the potential risk of renal disease mortality.[442] As noted above, based on the *underlying cause* data, the SMRs for renal disease in this cohort were either not significant (for acute renal disease) or only marginally so (for chronic renal disease). When this fact is considered along with the many uncertainties and the strong likelihood of underestimation in the exposure assessment, the Steenland *et al.* (2001) study of North American industrial sand workers clearly does not provide a sound basis for estimating the risk of silica-related renal disease mortality – particularly since the contemporaneous study of North American industrial sand workers by McDonald *et al.* (2005) found *decreasing* odds ratios for chronic non-malignant renal disease mortality with increasing cumulative exposure to silica.[443]

c. <u>Vermont Granite Workers</u>

The third study used in the Steenland *et al.* (2002) pooled analysis of renal disease mortality is difficult to analyze. It is referenced in Steenland *et al.* (2002) as "Attfield M.

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[442] OSHA appears to recognize this point, since its risk estimate for renal disease mortality reflects *underlying cause* data. See Health Effects Review at 316, 351-352, Table II-12 (basing its risk estimate for renal disease mortality on the exposure-response coefficient derived from underlying cause data). Another problem with using multiple-cause data is that the date of disease incidence is not known, so the decedent may have contracted renal disease even before being exposed to silica. Furthermore, in this cohort, the SMR for all causes of death (based on underlying cause analysis) exceeded unity (SMR = 1.23), which means there were more death certificates for the cohort than expected and, consequently, more opportunities for multiple cause listings.

[443] McDonald, J. *et al.*, Mortality from Lung and Kidney Disease in a Cohort of North American Industrial Sand Workers: An Update. Ann Occup Hyg. 2005; 49(5): 367-73.

Costello M. (2001) Quantitative exposure-response for silica dust and lung cancer in Vermont granite workers. Submitted for publication." Presumably, the published version is the study by the same name that OSHA has used to estimate lung cancer risks in the Vermont granite worker cohort, *viz.*, "Attfield, M.D. & Costello, J. (2004). Quantitative exposure-response for silica dust and lung cancer in Vermont granite workers. Am J Ind Med 45:129–138. OSHA-2010-0034-0543." Assuming that is correct, the study has a fundamental shortcoming for purposes of assessing renal disease mortality: it presents no data on the subject. Nor are such data for the Attfield and Costello study presented in Steenland *et al.* (2002) (apart from conclusory statements about rate ratios). So one is forced to speculate about renal disease mortality in the Attfield and Costello study. (Given the manner in which Attfield and Costello edited the lung cancer data in their study, this is no small concern.)

That said, certain things are known. For one thing, Attfield and Costello underestimated exposures by truncating work histories as of 1982 while extending follow-up through 1994. As one of OSHA's peer reviewers put the question: How is it possible that Attfield and Costello used unlagged exposures "if they did not update work histories beyond the original 1982 follow-up (p. 20, line 4) when they extended mortality follow-up to 1994?"[444] The answer is that they understated exposures, thereby overstating risk.

Most importantly, subsequent to publication of the Attfield and Costello study, P. Vacek and colleagues conducted a more comprehensive updated study of the Vermont granite worker cohort, which followed members of the cohort for ten additional years and

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[444] Peer review comment of Bruce Allen in Peer Review Comments, Silica Docket Item OSHA-2010-0034-1716, at 154.

had more complete mortality ascertainment and improved exposure assessment.[445] This updated study found no evidence of an association between silica exposure and either malignant or non-malignant kidney disease, even though the study had a substantially larger number of deaths from kidney cancer (28) and nephritis/nephrosis (34) than any other study. The SMR for chronic renal disease (nephritis/nephrosis) was below unity (SMR =0 .99; 95% CI: 0.68; 1.38). And, while cumulative silica exposure was significantly related to silicosis and other non-malignant respiratory disease in the case-control analysis, it "was not significantly related to mortality from kidney cancer (OR 0.96, 95% CI 0.84 to 1.09) or non-malignant kidney disease (OR 0.95, 95% CI 0.84 to 1.08)." Moreover, this lack of an association was found whether cumulative exposure was treated as a continuous or categorical variable, and whether it was analyzed on an untransformed or log-transformed basis.

In light of these findings in the updated study of the Vermont granite worker cohort, OSHA cannot reasonably rely on the superseded study by Attfield and Costello (2004) – which did not even present data on renal disease mortality – to estimate a risk of renal disease mortality for silica-exposed workers. As Dr. Peter Morfeld observes, "a pooled analysis of renal mortality risks based in large part on Attfield and Costello 2004 is unreliable for that reason alone."[446]

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445 Vacek, P., Verma, D., Graham, W. & Gibbs, G., Mortality in Vermont granite workers and its association with silica exposure. Occup Environ Med. 2011; 68: 312-318, available online at http://dx.doi.org/ 10.1136/oem.2009.054452. See pp. 36-41, *supra*.

446 Morfeld Comment at 24.

3. Because its Risk Assessment Rests on Such a Shaky and Uncertain Foundation, OSHA's Projections of Renal Disease Mortality Are Unsupported and Unreliable.

As shown above, OSHA is wrong to place confidence in the "quality of underlying exposure and job history information" used by Steenland *et al.* (2002) to estimate renal disease mortality risks for silica-exposed workers. In fact, the underlying exposure information in the studies relied on in the pooled analysis by Steenland *et al.* (2002) was highly uncertain and almost surely understated. Furthermore:

- The Homestake gold miner study – where a significantly elevated SMR for chronic renal disease was found only in men whose median average exposure level was estimated to be 150 $\mu g/m^3$ – suggests the existence of a threshold above 100 $\mu g/m^3$ for any increased risk of renal disease mortality that may be associated with silica exposure.

- The SMR for acute renal disease as the underlying cause of death in the industrial sand worker study by Steenland *et al.* (2001) was not significant while that for chronic renal disease was only marginally so. Moreover, the positive findings for renal disease mortality in that study are at odds with the findings of the contemporaneous study of North American industrial sand workers by McDonald *et al.* (2005), where *decreasing* odds ratios for chronic non-malignant renal disease mortality were observed with increasing cumulative exposure to silica.

- Whatever the Attfield and Costello study of Vermont granite workers may have found regarding renal disease mortality – and we do not know this, since data for renal disease mortality are not presented in the published version of the study – has been superseded by the updated study of that cohort by Vacek *et al.* (2011), which found no association between silica exposure and mortality from non-malignant kidney disease.

- The use of just three of ten studies in the pooled analysis, as noted above, raises the possibility of study selection bias.

- The "underlying cause" results for renal disease mortality in Steenland *et al.* (2002) were model-dependent. As Dr. Peter Morfeld points out, "in the log-model the p-value for trend was significant (p=0.03) but not so in the linear model (p=0.21). The authors stated that the log-model fit better, but evidence was

not given (e.g., information criteria), and it is unclear whether the results are robust to other transformations (e.g., adding an offset > 0 before taking logs)."[447]

- Steenland *et al.* (2002) used a log-linear model with log cumulative exposure, which they say provided the best fit to the pooled cohort data, to develop a risk coefficient. But, as Dr. Cox notes, "choosing a single best-fitting model neglects model uncertainty and leads to excess findings of 'significant' results due to artificially narrow confidence intervals. The reported standard error for the coefficient is artificially small because the analysis by Steenland et al. (2002a) ignored model uncertainty (Viallefont et al., 2001) and failed to perform multiple imputation of uncertain exposure values (Donders et al., 2006). The use of a log-transform for estimated cumulative exposures introduces unknown and uncorrected biases and errors into the estimate of the coefficient, due to the fact that the log transformation is applied to (unknown and uncharacterized) errors in cumulative exposure estimates. The log-linear model used is misspecified (e.g., no terms for errors in estimated exposures) and thus . . . its conclusions may bear no resemblance to the truth."[448]

- Finally, as noted in section II.D.1. above and as the Vacek *et al.* (2011) study illustrates, there is a serious question whether silica exposure causes renal disease mortality at all.

For all these reasons, the pooled analysis by Steenland *et al.* (2002) cannot serve as the basis for developing a credible estimate of excess mortality from renal disease in silica-exposed workers. Indeed, even if the foregoing issues did not exist, OSHA's projection of 39 excess deaths from renal disease per 1,000 workers exposed at the current general industry PEL would rest on the shakiest of foundations – for, as Dr. Steenland himself candidly admits, the "amount of data [that he and his colleagues analyzed to evaluate the risk of renal disease mortality] is insufficient to provide robust estimates of risk."[449] Given the problems

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447 *Id.* at 25.

448 Cox Comments at 96.

449 Steenland, N.K. & Bartell, S.M. Silica Exposure: Risk Assessment for Lung Cancer, Silicosis and Other Diseases. Prepared under contract to OSHA by ToxaChemica International, Inc. (Draft Final, December 7, 2004) at 27. Docket ID: OSHA-2010-0034-0469. See also Testimony of Professor Kyle Steenland, January 24, 2014, (Docket Item No. OSHA-2010-0034-2162) at 3 (noting that the epidemiologic evidence that silica exposure causes kidney disease "is less conclusive, and there are fewer data permitting quantitative

identified above, one is forced to conclude that, far from being robust, OSHA's projections of renal disease mortality are unsupported and unreliable.[450] They cannot be used as the basis for making a finding of significant risk.

F. A Final Word on Significant Risk

As shown in sections II.A.-E. above, OSHA has not demonstrated the existence of a significant risk of material health impairment at the current general industry PEL of 100 $\mu g/m^3$ even on the assumption that 100 $\mu g/m^3$ is the *average* RCS concentration level to which employees are exposed for 45 years, so that their *cumulative* RCS exposure is 4.5 mg/m^3-years. That, however, is not the relevant exposure to examine – for two reasons.

- First, because of the change from the old ACGIH respirable dust criterion to the new ISO/CEN criterion, the current PEL value of 100 $\mu g/m^3$ and the proposed PEL value of 50 $\mu g/m^3$, which have been used as *average* exposure values in OSHA's risk assessment, will be equivalent to approximately 80 $\mu g/m^3$ and 40 $\mu g/m^3$, respectively. See pp. 21-22, *supra*.

- Second, to comply with a PEL of 80 $\mu g/m^3$, employers would have to maintain long-term average exposures at a level that is less than 50 percent of that value, *i.e.*, below 40 $\mu g/m^3$, while to comply with a PEL of 40 $\mu g/m^3$, employers would have to maintain long-term average exposures at a level that is less than 50 percent of that value, *i.e.*, below 20 $\mu g/m^3$. See pp. 21-22, *supra*.

Accordingly, OSHA's risk assessment really should have evaluated potential risks at average exposure levels of 40 $\mu g/m^3$ (50% of a PEL of 80 $\mu g/m^3$) and 20 $\mu g/m^3$ (50% of a PEL of 40 $\mu g /m^3$) – which, over 45 years, equate to cumulative exposures of 1.8 mg/m^3-years and 0.9 mg/m^3-years, respectively. Thus, even if OSHA's risk assessment were

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risk assessment"); Cox Comments at 95 (noting that "while the findings [of Steenland *et al.* (2002)] may have been based on a large number of workers, they were not based on a large number of deaths from renal disease (50 in total). Thus, apart from its other problems, the study's findings are not at all robust, with very large confidence intervals bracketing OSHA's resulting point estimates of risk.").

[450] See Morfeld Comment at 25.

beyond reproach in all other respects (which certainly is not the case), it would be subject to the criticism that it evaluated the wrong exposure levels and was even more likely to have missed an exposure threshold at a level higher than the *average* RCS concentration of 40 $\mu g/m^3$ to which workers would be exposed if their employers comply with the current general industry PEL as reformulated to meet the ISO/CEN respirable dust criterion.

III. OSHA Has Not Shown that the Proposed Standard with a PEL of 50 $\mu g/m^3$ Would Be Feasible Across the Range of Affected General Industry Sectors.

Like any Section 6(b)(5) standard designed to protect employees against a significant risk of material health impairment, the proposed Silica Standard must be shown to be "feasible."[451] From the outset, courts have interpreted the term "feasible" in Section 6(b)(5) to encompass both technological and economic feasibility,[452] and they have made clear that the burden of demonstrating the feasibility of its standards rests on OSHA.[453] To carry this burden, OSHA generally must demonstrate feasibility on a disaggregated industry-by-industry basis.[454] Moreover, while the feasibility inquiry focuses in part on the availability,

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[451] In addition, an OSHA standard must be cost effective. See *Cotton Dust,* 452 U.S. at 514 n. 32; *Int'l Union, UAW v. OSHA,* 37 F.3d 665, 668 (D.C. Cir. 1994); 76 Fed. Reg. 24576, 24578 (May 2, 2011).

[452] See *Industrial Union Dep't, AFL-CIO v. Hodgson*, 499 F.2d 467 (D.C. Cir. 1974); *AFL-CIO v. Brennan,* 530 F.2d 109 (3d Cir. 1975); *United Steelworkers of America v. Marshall*, 647 F.2d 1189, 1264 (D.C. Cir. 1980), *cert. denied*, 453 U.S. 913 (1981).

[453] *See United Steelworkers, supra,* 647 F.2d 1189 at 1265; *Forging Industry Ass'n v. Secretary of Labor*, 773 F.2d 1436, 1452 (4th Cir. 1985) (en banc).

[454] See *United Steelworkers, supra*, 647 F.2d at 1177 (evaluating feasibility on industry-by-industry basis); *Forging Industry Ass'n v. Secretary of Labor, supra*, 773 F.2d at 1452; *Building and Construction Trades Dep't, AFL-CIO v. Brock*, 838 F.2d 1258, 1272-73 (D.C. Cir. 1988); *International Union, UAW v. OSHA*, 938 F.2d 1310 (D.C. Cir. 1991) (feasibility test must be applied on a disaggregated industry-by-industry basis, except where a refusal to further disaggregate can be justified on grounds of administrative convenience).

practicality, and cost of controls, the ability (or inability) to measure exposures reliably also must be taken into account. As a practical matter, an OSHA standard would not be feasible if employee exposures at the PEL or action level cannot be measured with sufficient accuracy and precision to make reliable determinations of whether the PEL and action level are being exceeded and of when the proposed ancillary requirements of the standard apply.

As detailed here, OSHA's feasibility analysis is deeply flawed and cannot serve as the basis for concluding that the proposed standard is feasible across all affected industry sectors. Indeed, properly analyzed, the proposed standard would cost many times more than what OSHA has estimated, and, in many industries, would far exceed the revenue and/or profit thresholds that OSHA has long applied to assess economic feasibility. Moreover, the evidence strongly indicates that commercial laboratories (and OSHA itself) will not be able to sample and analyze crystalline silica accurately, precisely and reliably at airborne concentrations of 50 $\mu g/m^3$ and below. For these reasons, OSHA has not met its burden of demonstrating that the proposed standard with a PEL of 50 $\mu g/m^3$ is feasible, as required under Section 6(b)(5) of the Occupational Safety and Health Act of 1970.

A. OSHA Has Not Shown that the Proposed Standard Would Be Technologically Feasible in All Affected Industry Sectors.

In order to meet its burden of demonstrating that a standard is technologically feasible, "OSHA must prove [based on substantial evidence] a reasonable possibility that the typical firm [in the affected industry] will be able to develop and install engineering and work practice controls that can meet the PEL in most of its operations." *United Steelworkers*, *supra*, 647 F.2d at 1272-73. While OSHA may be able to make reasonable

predictions based on evidence in the record, it cannot rely on unsupported statements to assert that achieving the PEL is technologically feasible in particular industries.[455]

OSHA has not carried its burden of showing that the proposed PEL would meet the test of technological feasibility in all affected industry sectors. Despite the fact that the current PEL of 100 $\mu g/m^3$ has been in effect for over 40 years, OSHA's own data show that exposures in excess of that level are widespread.[456] Indeed, in many cases, exposures exceed the current PEL by factors of 2-3 or more.[457] This suggests strongly that complying with a revised PEL of 50 $\mu g/m^3$ would present significant, costly technological challenges in various industry sectors. This is particularly the case given OSHA's never-to-be-exceeded application of the PEL, which means that long-term average exposures will have to be maintained at a level substantially below the PEL (in many cases, below one-half of the PEL) in order for employers to be reasonably confident that the PEL will not be exceeded on any day on which exposure monitoring may be performed (see pp. 23-25, *supra*), a point which OSHA frankly acknowledges.[458] Yet OSHA has made no attempt to show the technological feasibility of reducing exposures to such low levels. Even apart from that consideration, analyses done by the foundry, hydraulic fracturing, and construction industries show that

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455 *See United Steelworkers*, *supra,* 647 F.2d 1189; *Color Pigments Mfrs. Ass'n v. OSHA*, 16 F.3d 1157 (11th Cir. 1994) (court finds no substantial evidence to support OSHA's industry-specific feasibility determination in the dry color formulator sector).

456 In the proposed rule, OSHA recognizes that over-exposures are widespread in both general industry and construction, with 81,000 workers being exposed above 100 $\mu g/m^3$ in general industry and 420,000 being exposed above that level in construction. 78 Fed. Reg. at 56349-56352, Table VII-5.

457 See 78 Fed. Reg. at 56349-52, Table VIII-5.

458 *Id.* at 56354.

complying with a PEL of 50 μg/m^3 would not be technologically feasible for many operations in those sectors.[459]

Another aspect of technological feasibility is the question whether RCS exposures at a level of 50 μg/m^3 and below can be measured accurately and precisely. As explained in section III.C. below, a large body of evidence indicates that RCS exposures at such low levels cannot be measured reliably with an acceptable degree of accuracy and precision. For that reason alone, the proposed PEL of 50 μg/m^3 is not technologically feasible.

B. OSHA Has Not Shown that the Proposed Standard Would Be Economically Feasible.

OSHA bears the burden to establish that its proposal is economically feasible across all affected industry sectors. To carry this burden, OSHA must "'construct a reasonable estimate of compliance costs and demonstrate a reasonable likelihood that these costs will not threaten the existence or competitive structure of an industry'" or imperil its long-term profitability.[460] As the D.C. Circuit explained, a standard would be economically infeasible "if compliance were likely to disable the industry from competing with substitute products, or markedly to increase concentration within the industry."[461] To make a complete economic feasibility determination, OSHA must engage in an in-depth analysis of each affected

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459 See Comments filed in this docket by the American Foundry Society, the American Petroleum Institute, and the Construction Industry Safety Coalition. The Panel incorporates by reference the portions of these comments that address technological feasibility.

460 *Hexavalent Chromium*, *supra,* 557 F.3d at 177 (3d Cir. 2009), quoting *United Steelworkers*, *supra,* 647 F.2d. at 1272.

461 *National Cottonseed Products Ass'n v. Brock*, 825 F.2d 482, 487 (D.C. Cir. 1987). While a strict cost-benefit analysis is not required to demonstrate the economic feasibility of an OSHA standard, the Supreme Court has suggested that that a cost-benefit test might be permissible under Section 6(b)(5), even if not mandatory. *See Entergy Corp. v. Riverkeeper, Inc.*, 556 US 208, 223, 129 S. Ct. 1498, 1508 (2009), 2009 U.S. LEXIS 2498 at *25.

industry sector and determine how the proposed standard may affect its economic and competitive structure.[462] However, as a threshold test of economic feasibility, OSHA typically applies a rule of thumb under which the competitive structure and long-term profitability of an industry are assumed not to be endangered as long as the annualized compliance costs for the industry are less than 1% of revenues and 10% of profits.[463] When either of those thresholds is exceeded, however, a more in-depth analysis is required. If the annualized compliance cost as a percentage of revenues or profits in an industry exceeds a high enough level, OSHA deems the standard economically infeasible for that industry.[464]

In analyzing economic feasibility for general industry, OSHA takes the position that it need not consider the costs that firms whose employees currently are exposed above 100 $\mu g/m^3$ would incur in reducing those exposures to the 100 $\mu g/m^3$ level.[465] At the same time, OSHA wrongly assumes that controls sufficient to reduce >100 $\mu g/m^3$ exposures to a level of 100 $\mu g/m^3$ will, in almost all cases, also be sufficient to reduce such exposures to a level of 50 $\mu g/m^3$, *i.e.*, that it costs as much to reduce exposures to a level of 100 $\mu g/m^3$ as to a level of 50 $\mu g/m^3$. The combination of this position and this incorrect assumption caused OSHA to ignore the costs that employers would incur to reduce RCS exposures of those employees whose current RCS exposure level exceeds 100 $\mu g/m^3$. That is a major omission – because

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462 *United Steelworkers, supra*, 647 F.2d at 1281; *Am. Textile Manufacturers Inst., Inc. v. Donovan*, 452 U.S. 490, 531 (1981).

463 See *Hexavalent Chromium*, *supra,* 557 F.3d at 181-182; 71 Fed. Reg. 10100, 10300-10301 (Feb. 28, 2006).

464 *See Hexavalent Chromium*, *supra*, 557 F.3d at 172 (OSHA finds that a standard is not economically feasible where the annualized costs exceeded 2.7% of revenues and 65% of profits).

465 *See* 78 Fed. Reg. at 56337.

two-thirds of the general industry employees who OSHA estimates are currently exposed above 50 μg/m^3 are exposed above 100 μg/m^3 (81,000 out of 122,500 employees).[466] Under OSHA's theory, the cost to reduce RCS exposures of those 81,000 employees is excluded in determining whether the proposed standard is economic feasible.

OSHA's position, however, runs counter to the test of economic feasibility enunciated by the courts, which focuses on the projected competitive structure of the industry after all firms have made whatever expenditures are necessary to move them from their current status into full compliance with the proposed standard. If mandating that all firms achieve full compliance with a PEL of 50 μg/m^3 would drive a large proportion of firms out of business or radically alter the competitive structure of an industry, the standard would not be economically feasible, even if – hypothetically – that result might have been driven in part by the costs needed to reduce exposures to the current PEL of 100 μg/m^3. As a matter of economics and logic, the impact on the industry's competitive structure will reflect what *transpires in the real world as it actually exists*, not in some hypothetical world of OSHA's own devising. The facts on the ground and the obligation to comply with the new requirements imposed by OSHA are what will determine the economic impact of a 50 μg/m^3 PEL. Analyzing economic impact on the basis of a situation that OSHA *knows* does not exist is a pointless exercise that bears no relation to what actually would occur. Instead, OSHA must focus on the competitive health of the industry as it would exist after the *full* costs necessary to achieve compliance with the proposed standard have been incurred.[467]

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466 *See id.* at 56347.

467 *See Forging Indus. Ass'n v. Sec'y of Labor*, 773 F.2d 1436, 1453 (4th Cir. 1985) (economic feasibility focused on "long-term profitability" of the industry); *Asarco, Inc. v.*

Nevertheless, in the analysis presented below, we will consider compliance costs on both an *actual full cost* basis and on a *hypothetical incremental or partial cost* basis. In either case, it is clear that OSHA has failed to carry its burden of showing that the proposed standard with a PEL of 50 $\mu g/m^3$ is economically feasible, because OSHA has not "construct[ed] a reasonable estimate of compliance costs" for its proposed standard. Rather, as detailed below, OSHA has vastly understated the costs, which – when properly analyzed – are found to substantially exceed OSHA's own benchmark thresholds of 1% of annual revenues and/or 10% of annual profits in many general industry sectors. For that reason alone, OSHA must reconsider the proposed PEL of 50 $\mu g/m^3$ and conduct further economic analyses to assess whether a standard with a PEL set at that level would "threaten the existence or competitive structure" of the affected industries.

1. OSHA's Estimate of Compliance Costs Is Deeply Flawed and Vastly Understates the True Costs of Compliance.

The two key inputs to OSHA's economic analysis are (1) the cost to comply with the proposed standard and (2) the revenues and profits for each affected industry sector. In the proposed rule, OSHA projects annualized compliance costs of only $132.5 million for all of general industry, 78 Fed. Reg. at 56358, and, after comparing those costs to outdated revenue and profit figures from 2006 and earlier, concludes that the proposed standard is economically feasible.

However, OSHA's cost estimates are fundamentally flawed and dramatically understate the economic impact of the proposed rule by as much as an order of magnitude or more. In fact, the full annualized cost for general industry to comply with the proposed

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OSHA, 746 F.2d 483, 501 (9th Cir.) (OSHA profit estimates should "reflect all regulatory costs").

standard exceeds *$6 billion*. Hence, once costs are properly estimated, it is plain that the proposed standard will have a serious economic impact on the vast majority of general industry sectors. Given the huge disparity between OSHA's cost estimates and the actual, real-world costs that will be imposed on employers, it would be arbitrary, capricious, and patently unreasonable for OSHA to use the cost estimates included in the proposed rule to conduct a feasibility analysis. Accordingly, at a minimum, OSHA must develop new and more realistic compliance cost estimates to evaluate the economic impact of the proposal on the various affected industry sectors.

a. <u>Summary of OSHA's Economic Feasibility Analysis</u>

To prepare its economic feasibility analysis, OSHA began by collecting summary statistics for industries with potential worker exposure to crystalline silica, including the number of affected entities and establishments, the number of at-risk workers, and the average revenue for affected entities and establishments. 78 Fed. Reg. at 56339. OSHA then prepared exposure profiles for at-risk workers based on sector and job category through workplace sampling. *Id.* at 56339, 56347. Based on that profile, OSHA estimated that 122,000 workers in general industry have silica exposures above 50 $\mu g/m^3$, of which 81,000 have silica exposures above 100 $\mu g/m^3$. *Id.* at 56347.

For the workers exposed above the proposed PEL of 50 $\mu g/m^3$, OSHA conducted a feasibility analysis to determine whether there was a combination of engineering controls and work practices that would reduce the exposures of those workers to below the proposed PEL. *Id.* at 56354-55. OSHA determined that a PEL of 50 $\mu g/m^3$ was technologically feasible for all of the general industry sectors. *Id.* at 56355. Having determined that the PEL of 50 $\mu g/m^3$ was technologically feasible, OSHA calculated the costs necessary to implement all of the engineering controls and work practice standards identified in the technological

feasibility analysis for each job category in each general industry sector. *Id.* at 56356-57 & Table VIII-9.

Once OSHA calculated the control costs for a single exposed worker, it calculated an annualized cost for each sector by scaling up the cost estimates based on the number of overexposed workers in each sector and the degree to which OSHA believed that a particular set of controls was capable of protecting more than one worker. *Id.* at 56361-63. OSHA then compared its estimate of the annualized compliance cost for each general industry sector to its estimates of the sector's annualized revenue and profits to determine whether the estimated compliance cost exceeded 1% of revenue or and 10% of profits for the sector. Based on this analysis, OSHA concluded that the annualized costs of the proposed standard did not exceed the 1% or 10% revenue and profit thresholds for any general industry sector. *Id.* at 56369. However, as described below, OSHA has made very significant errors both in estimating the costs to comply with the proposed standard and in its comparisons to revenue and profit data. As a result, OSHA's reliance on this feasibility analysis is arbitrary and unreasonable.

b. OSHA Has Grossly Underestimated the Costs for General Industry to Comply with the Proposed Standard

OSHA's feasibility analysis grossly underestimates the costs of complying with the proposed standard due to a series of fundamental errors in its approach to estimating costs. Once those errors are corrected, the cost to comply with the proposed PEL and ancillary provisions of the standard is found to be dramatically higher than what OSHA has estimated. Even when only the hypothetical *incremental* costs of reducing exposures from 100 $\mu g/m^3$ to 50 $\mu g/m^3$ are considered, general industry's annualized costs to comply with the proposed standard would be more than *$4.7 billion*. That estimate, however, is far too low because it

assumes *hypothetically* that all firms have achieved compliance with the current PEL of 100 $\mu g/m^3$, which, of course, is not the case – since, by OSHA's reckoning, 81,000 workers (or 66% of all general industry workers whose current RCS exposures exceed the proposed PEL of 50 $\mu g/m^3$) are exposed above 100 $\mu g/m^3$. 78 Fed. Reg. at 56352. On a *full cost* basis, the *real* cost for general industry to comply with the proposed standard would exceed *$6 billion*. When construction and hydraulic fracturing are included, the full costs of compliance with the proposed standard rises to more than $8.6 billion.[468]

The Panel retained URS Corporation ("URS") to evaluate OSHA's technological feasibility analysis and cost estimates. URS' complete report is submitted with these Comments as Attachment 8[469] URS evaluated OSHA's feasibility analysis for the general industry sectors and identified a series of fundamental errors in OSHA's cost estimates for engineering controls and ancillary provisions that materially affect the projected compliance costs for the proposed rule. In most instances, URS was able to correct those errors and provide more accurate cost estimates. However, in other cases, URS elected to retain OSHA's cost estimates despite the fact that they significantly underestimate the true compliance costs. As a result of that decision, URS' own cost estimates remain conservative and are likely to underestimate the compliance costs that would actually be experienced by the general industry sectors.

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468 *See* Preliminary Letter Report of Environomics to the American Chemistry Council's Crystalline Silica Panel Regarding the Economic Impact of the Occupational Safety and Health Administration's Proposed Standard for Occupational Exposure to Respirable Crystalline Silica (February 7, 2014) ("Environomics Report"), Table 1.

469 URS Corporation, Critique of OSHA's Cost Models for the Proposed Crystalline Silica Standard and Explanation of the Modifications to Those Cost Models Made by URS Corporation (February 7, 2014) ("URS Feasibility Report").

After estimating the cost for each engineering control and calculating the number of control packages that would be needed in each of 19 general industry sectors, URS aggregated the costs across those sectors, with results shown in Tables 1 and 2 below.[470]

Table 1: Annualized Costs of Engineering Controls in 19 General Industry Sectors

Sector	OSHA Incremental Costs ($mm)	OSHA Full Costs ($mm)	URS Incremental Costs ($mm)	URS Full Costs ($mm)
Asphalt Paving Products	0.18	0.18	0.25	0.25
Asphalt Roofing Materials	2.19	4.39	116.12	173.24
Concrete Products	11.74	33.98	463.38	582.48
Costume Jewelry	0.35	0.12	0.15	0.44
Cut Stone	5.89	15.78	111.44	138.14
Fine Jewelry	0.31	1.10	1.26	3.83
Flat Glass	0.28	0.36	15.71	20.47
Iron Foundries	9.97	28.41	859.27	1,247.07
Mineral Processing	3.59	5.38	84.18	97.16
Mineral Wool	0.90	1.44	70.30	84.4
Nonferrous Sand Casting Foundries	3.43	9.77	345.31	480.99

[470] The URS Feasibility Report includes cost estimates for only 19 of the 29 general industry sectors. For the remaining 10 sectors, URS determined that the number of facilities included in each sector by OSHA was grossly overestimated, so that the cost per facility and the total compliance costs are highly uncertain in those sectors. *See* URS Feasibility Report at 2, n.1. For that reason, URS excluded them from its estimate of total general industry compliance costs, thereby understating the total compliance costs for general industry as a whole. The fact that OSHA grossly overestimated the number of facilities in these 10 sectors casts doubt on the Agency's general methodology for calculating compliance costs and makes its feasibility determination even more problematic.

Non-Sand Casting Foundries	5.72	16.29	526.25	749.43
Other Ferrous San Casting Foundries	2.98	8.50	272.41	391.95
Other Glass Products	1.53	2.48	38.85	54.16
Paint and Coatings	--	0.82	20.55	25.36
Pottery	4.09	10.20	344.33	472.81
Ready-Mix Concrete	7.03	10.78	344.96	356.46
Refractories	0.69	1.66	65.88	72.26
Structural Clay	11.45	33.48	264.36	402.64
Grand Total	**71.96**	**185.11**	**3,944.97**	**5,353.60**

Table 2: Total Annualized Costs of Proposed Standard (Including Ancillary Provisions)

Sector	OSHA Incremental Costs ($mm)	OSHA Full Costs ($mm)	URS Incremental Costs ($mm)	URS Full Costs ($mm)
Asphalt Paving Products	0.24	0.24	4.01	4.01
Asphalt Roofing Materials	3.16	5.35	123.51	180.63
Concrete Products	19.00	41.24	801.50	920.61
Costume Jewelry	0.13	0.21	1.97	2.26
Cut Stone	8.60	18.48	137.12	163.82
Fine Jewelry	1.88	2.67	17.35	19.93
Flat Glass	0.28	0.40	16.27	21.03
Iron Foundries	13.91	32.34	935.02	1,322.82
Mineral Processing	4.60	6.39	115.62	128.59
Mineral Wool	1.09	1.63	72.50	86.64
Nonferrous Sand Casting Foundries	4.84	11.18	379.94	515.62
Non-Sand Casting Foundries	8.00	18.58	576.61	799.79
Other Ferrous San Casting Foundries	4.18	9.70	296.57	416.11
Other Glass Products	1.84	2.79	42.28	57.58
Paint and Coatings	0.14	0.96	22.84	27.65
Pottery	6.01	12.12	394.50	522.98
Ready-Mix Concrete	16.51	20.26	401.55	413.04
Refractories	1.09	2.06	68.73	75.11
Structural Clay	12.91	34.94	314.55	452.84
Grand Total	**108.40**	**221.54**	**4,722.45**	**6,131.08**

As these data show, regardless of whether only the incremental compliance costs are used or whether the full compliance costs are properly considered, OSHA substantially underestimated the anticipated compliance costs for general industry. The comparisons are stark:

- URS' estimate of annualized *incremental engineering control costs* for 19 general industry sectors to reduce exposures from the level of the current PEL to the level of the proposed PEL is $3.945 billion.[471] In comparison, OSHA's *incremental engineering control cost* estimate for all of general industry was only $88.4 million.[472]
- When the *full engineering control costs* are considered, URS estimated that the annualized costs for 19 general industry sectors would be $5.354 billion. In contrast, URS found that the *full engineering control* costs under OSHA's estimate would be only $185.1 million for the same 19 general industry sectors.[473]
- URS' estimate of the annualized cost of the ancillary provisions of the standard was more than an order of magnitude higher than OSHA's ($777.4 million vs. $36.4 million) for the 19 general industry sectors reviewed by URS.[474]
- Combining the engineering control costs with the ancillary provision costs, URS estimated that:
    - The annualized *incremental cost* of the proposed standard for 19 general industry sectors would be $4.7 billion, while OSHA's estimate of the *incremental cost* is $108.4 million; and
    - The annualized *full cost* of the proposed standard for those 19 general industry sectors would be $6.13 billion, while OSHA's estimated *full cost* for these sectors is $221.5 million.

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471 See URS Feasibility Report, Table 3A.

472 See 78 Fed. Reg. 56358, Table VIII-8.

473 See URS Feasibility Report, Table 3A.

474 See URS Feasibility Report, Table 4A. As discussed below, approximately 60% of URS' ancillary cost estimate is for the cost of professional cleaning in certain industries. OSHA had expressly included professional cleaning in its description of measures necessary for certain industry sectors to meet the proposed PEL, but then mistakenly left those costs out of its own cost calculations.

- On a broader scale, the *full cost* of the proposed standard for general industry, construction, and hydraulic fracturing – as estimated by URS and Environomics – would be $8.6 billion,[475] as compared with OSHA's estimate of $672 million for the same industries.[476]

The remainder of this section describes in more detail the errors that URS identified in OSHA's cost analysis, the steps URS took to correct those errors, and the effect that URS' corrections had on the compliance costs for general industry.

(1) OSHA Has Grossly Underestimated the Engineering Control Costs for General Industry to Comply with the Proposed PEL.

The principal reasons why OSHA has underestimated engineering control costs in general industry are described below.

OSHA does not account for increased overexposure caused by the proposed adoption of the ISO/CEN definition of respirable dust. In an effort to harmonize OSHA's definition of respirable dust with current aerosol science, OSHA proposes to adopt the ISO/CEN definition of respirable dust in place of the "obsolete" 1968 ACGIH definition. 78 Fed. Reg. at 56444. The ISO/CEN definition increases the particle size "cut point" from 3.5 to 4 microns – with the result that in most workplaces, more respirable crystalline silica will be collected under the ISO/CEN definition than under the 1968 ACGIH definition at the same exposure concentration. See pp. 21-22, *supra*. While the difference between the two standards typically varies by as much as 20%, differences in excess of 30% have also been reported. PEA at IV-20. This difference has significant implications for OSHA's exposure profile and cost analysis because OSHA relied on exposure data reflecting the 1968 ACGIH

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475 Environomics Report, Table 1.

476 78 Fed. Reg. at 56358 (construction and general industry); PEA at A-61 (hydraulic fracturing).

method. *See* URS Feasibility Report at 9. As a result, OSHA has underestimated the number of workers currently exposed above the proposed PEL of 50 $\mu g/m^3$ as measured using the ISO/CEN definition. To correct this error, relying on data in the PEA, URS estimated the number of workers whose exposure would be below 50 $\mu g/m^3$ under the ACGIH definition, but above 50 $\mu g/m^3$ under the ISO/CEN definition by assuming that ISO/CEN measurements would be 20% higher than ACGIH measurements. *See* URS Feasibility Report at 9-10 (citing PEA at IV-18-21, applying statistical analysis to workers exposed between 25 and 50 $\mu g/m^3$ in OSHA's engineering costs spreadsheets). This materially increased the number of workers in general industry projected to be exposed above the proposed PEL.[477]

<u>OSHA underestimates the number of control packages needed to comply with the proposed PEL by adopting an employee-based approach.</u> In estimating the cost of complying with the proposed standard, OSHA applies an employee-based approach, which assumes multiple overexposed workers will be protected by each set of engineering controls. OSHA frequently assumed that four overexposed workers would be protected by each set of engineering controls and, in some cases, assumed six or even eight workers were protected. PEA at V-16. As URS explains, "*facilities* install engineering controls; thus the crucial factors in determining the nature and scope of engineering controls required in any given general industry sector are the number of facilities in that sector and the number of areas within a given facility where the employer would need to install the controls." URS Feasibility Report at 4. While URS believes that a facility-specific model would be the ideal

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[477] URS did not apply the same conversion to evaluate exposures above 100 $\mu g/m^3$ because the existing PEL is based on the ACGIH definition. As a result, the costs needed to achieve compliance with the existing PEL are not affected by the adoption of the ISO/CEN definition. *See* URS Feasibility Report at 10.

way to estimate compliance costs, the docket did not contain sufficient information for URS to utilize that procedure. *Id.* at 7. Instead, URS modified OSHA's employee-based approach to better approximate the efficiency with which engineering control packages would protect overexposed workers. In particular, URS found that OSHA's assumptions significantly overestimated the average number of workers protected by each engineering control package because many facilities—including virtually all small and very small entities—have fewer than four employees assigned to the job category and job site to which the engineering control package would apply. *Id.* at 5. Likewise, URS found that OSHA overestimated the number of facilities that operate two shifts per day. *Id.* To provide a more accurate estimate, URS created binomial distributions of the number of workers per facility and their work locations for large, small, and very small entities. *Id.* at 7. Using those distributions, URS recalculated the number of engineering control packages needed to protect all of the overexposed workers. *Id.* at 7-8. In addition, URS found that for some industries, overexposures above the proposed PEL would be sufficiently widespread that such facilities would be forced to implement engineering controls for all workers in a given job category, regardless of their individual exposure status. *Id.* at 8 ("[W]hen the number of overexposed workers exceeds a certain threshold, one is led to the practical conclusion that the existing control methods for a given job category at a facility are simply inadequate, and must be totally replaced or completely overhauled for all workers.").

<u>OSHA underestimated the unit costs of several engineering controls.</u> Based on discussions with personnel from companies in affected industries and vendors of control technologies, URS concluded that OSHA underestimated the unit costs of several proposed engineering controls. *See* URS Report at 12. (describing basis for revised costs). For

example, OSHA's proposed engineering controls included abrasive blasting cabinets that are much smaller than those typically used by foundries. As a result, OHSA greatly underestimated the maintenance costs associated with this control. *Id.* A complete list of URS' adjustments to OSHAs' unit cost assumptions can be found in the URS Feasibility Report and supporting data. *Id.*; URS Engineering Cost Model, Table 5, "Changes to Engineering Controls used in URS Alternative Engineering Costs Model."

OSHA's local exhaust ventilation ("LEV") costs do not include necessary costs for engineered designs and renovation work. In estimating the costs of upgrading or installing LEV systems, OSHA relied on outdated ACGIH data that was not intended to address crystalline silica, particularly at concentrations as low as the proposed PEL and Action Level. See URS Feasibility Report at 11. In particular, OSHA relied on sources which it asserted suggest a proposed PEL of 50 $\mu g/m^3$ could be achieved with minimal changes to existing LEV systems. *Id.*. However, plant operators interviewed by URS "stated that compliance could rarely be achieved by bolstering existing LEV equipment with stronger motors." *Id.* at 13. Instead, to achieve the proposed PEL, LEV systems would require additional design work and careful planning for mass balance of air flow. *Id.* ("[I]t is URS's experience, confirmed by industry plant operators, that old LEV systems would need to be removed, and a new system of ductwork, better shaped hoods, and reconstructed conveyor access points would need to be installed.") After accounting for these additional design, material, and installation costs, URS applied conservative assumptions and determined that LEV capital costs would likely increase from OSHA's estimate of $12.38 per cfm to $22.00 per cfm or more. *Id.*.

Several of OSHA's proposed engineering controls are insufficient or infeasible to achieve the proposed PEL. By consulting with other experts, URS determined that OSHA overestimated the effectiveness or feasibility of several engineering controls. *See* URS Feasibility Report at 10-12. As a result, the engineering controls included in OSHA's cost estimates would be insufficient to achieve the PEL in all circumstances. In some cases, URS was able to identify additional engineering controls and work practices that could be applied in order to achieve the PEL and incorporated the costs of those controls in its analysis. For example, URS found that OSHA frequently underestimated the length of conveyors that would need to be covered to achieve the proposed PEL, noting that "many mid-sized or larger foundries and structural clay facilities may have thousands of feet of conveyors used in their operations." *Id.* at 14. Therefore, URS increased the total amount of covered conveyors needed. *Id.* at 14-15. Likewise, URS determined that OSHA's proposal to replace compressed air with vacuum air was not a viable approach for industries which rely on compressed air in production, such as the foundry industry; accordingly, URS applied a flexible hooded duct LEV as an alternative control. *Id.* at 14. In other cases, additional engineering controls and work practices were not available. For example, URS determined that LEV was not a practical control for concrete mixing operations because wet concrete would set up on the filters. *Id.* In those cases, URS included the cost of respirators after concluding that alternative engineering controls were not available. These additional engineering controls and increased reliance on respirators add to the total compliance costs for all general industry sectors and are reflected in the URS estimates of full and incremental costs.

OSHA failed to support its assumptions regarding the LEV air capture velocity and filtration needed to achieve the revised PEL. Relying on outdated ACGIH manuals that were not intended to address crystalline silica at concentrations as low as 50 $\mu g/m^3$, OSHA assumed that only minimal changes in air capture velocity and filtration would be needed to reduce exposure from the existing PEL of 100 $\mu g/m^3$ to the proposed PEL of 50 $\mu g/m^3$. URS Feasibility Report at 12 ("[T]he ACGIH capture velocities used by OSHA were first developed and published many years ago, long before silica concentrations as low as the proposed 50 $\mu g/m^3$ PEL and 25 mg/m^3 AL were even contemplated by OSHA.").

More recent data establishes that increasingly higher capture velocities and CFM are required for each incremental reduction in exposure. *Id.* at 11, 14. To account for this non-linear relationship between LEV costs and exposure reductions, URS applied a conservative default assumption that halving the PEL would require doubling the CFM for each LEV system. *Id.* at 14. Based on available data and communication with focus industries, this assumption provided a reasonable and conservative estimate of the total CFM that would be needed to achieve a more stringent PEL. *Id.* at 14 ("The change [in CFM assumed by URS] was smaller than the upper ranges of the ACGIH recommendations and also smaller than most of the specific suggestions made by industry representatives."). Doubling the CFM estimates further added to the compliance costs for general industry; that additional cost is reflected in the URS cost model.

OSHA erroneously assumes that high-cost engineering controls would be used to achieve compliance with the existing PEL. In cases where firms are not in compliance with the existing PEL, OSHA frequently asserts in the PEA that a series of engineering controls and work practices would be needed in order to achieve the proposed PEL. The costs of the

engineering controls and work practices vary considerably, and in some cases, OSHA assumed (without supporting data) that non-compliant entities would apply high-cost engineering controls to meet the current PEL of 100 $\mu g/m^3$ and then apply additional low-cost controls to reduce exposures to the proposed PEL of 50 $\mu g.m^3$. URS Feasibility Report at 16 ("In some instances, OSHA has designated *the most expensive control* as the one that is necessary to meet the 100 $\mu g/m^3$ PEL." (emphasis in original)). For example, for sawyers and splitter/chippers in the stone cutting industry, OSHA assumed that high-cost pressurized water outlets, re-plumbing, floor grading, and drains would be installed to achieve the 100 $\mu g/m^3$ PEL, while low-cost options such as additional LEV and local wetting would be added to achieve a PEL of 50 $\mu g/m^3$. PEA at IV-106; URS Feasibility Report at 17.

This approach is fundamentally inconsistent with basic principles of economic decision-making, which dictate that the most cost-effective control measures would be applied first to achieve the existing PEL, with more costly alternatives being added only if the PEL is lowered. Thus, as URS explained, "[m]ore expensive controls would be added only after more cost-effective options have been exhausted." URS Feasibility Report at 16. By assigning these higher-cost controls to achieving the existing PEL of 100 $\mu g/m^3$, while assigning low-cost controls to further reducing exposures to 50 $\mu g/m^3$, OHSA has artificially lowered the incremental compliance costs of moving from 100 $\mu g/m^3$ to 50 $\mu g/m^3$, even though a rational plant operator would do just the opposite. *Id.* at 16 ("OSHA has used a sleight of hand maneuver to shift control costs to achieving the current PEL and distort the economic feasibility analysis for the proposed rule."). URS adjusted the assignment of engineering controls so that low-cost controls were applied first to achieving the current PEL,

while high-cost alternatives were reserved for achieving the proposed PEL. *Id.* at 18. This change properly reallocated substantial costs, as reflected in the URS cost estimates.

OSHA overestimates the number of workers that can be covered by certain engineering controls. As described above, OSHA assumes that most engineering controls are capable of protecting multiple overexposed workers. *See* PEA at V-16. In the abstract, that can be a reasonable assumption, but if and only if there are multiple overexposed workers in a given job at a specific facility. However, some assumptions OSHA made regarding the number of overexposed workers that a control would protect are physically impossible. For example, OSHA assumes that enclosing the cabs for forklifts and front end loaders would protect four overexposed workers. URS Feasibility Report at 5 (citing OSHA Model Workbook #7, Docket ID: OSHA-2010-0034-1781). However, that assumption is physically impossible. As URS explained, "[a]ssuming that there are two shifts, each enclosed cab would be capable of protecting only two workers, not four as OSHA suggests." *Id.* at 6. URS corrected this error by reducing the number of potentially protected workers per enclosed cab to two (assuming one worker per vehicle on each of two shifts). Corrections to these and other instances where OSHA made similar errors are reflected in the increased full and incremental compliance costs for general industry estimated by URS.

OSHA failed to account for trial-and-error inherent in achieving the revised standard. By simply listing a series of engineering controls and work practices that will be sufficient to achieve the proposed standard, OSHA ignores the fact that these are not "off-the-shelf" controls that can be implemented seamlessly. Instead, there are significant design requirements that often require modifications and fine-tuning through an iterative process, even after initial implementation. As URS explains, "facilities will engage in a trial and error

process, adding increasingly more costly controls and optimizing existing controls in an effort to reduce exposure below 50 μg/m^3." URS Feasibility Report at 19. This trial-and-error process can add significant costs to the implementation of engineering controls and may also require additional respirator use until facilities can ensure that the revised PEL can be achieved. Understandably, these costs are difficult to predict and URS did not attempt to do so. *Id.*. However, these unaccounted for costs will be imposed on the affected facilities. The exclusion of these costs from the URS cost model is another reason why URS's overall cost estimates are conservative.

OSHA failed to account for the inherent variability in sampling data. As explained more fully in Section III.C. below, exposure monitoring samples taken at or below the proposed PEL will exhibit significant variability. As a result, a single sampling result at, or just slightly below, the proposed PEL would not provide a high level of assurance that the workplace is in compliance with OSHA's never-to-be-exceeded exposure limit. URS Feasibility Report at 6. Instead, as URS explains, "facilities must take a conservative approach and apply engineering controls whenever there is a risk that an employee may be exposed above the PEL." *Id.* Thus, as a practical matter, each facility would have to target a mean silica concentration well below the PEL in order to attempt to ensure it is in compliance with the proposed standard. URS found, however, that where OSHA identified "only a few sampling results slightly below 50 μg/m^3," the Agency considered that result to be sufficient evidence of technological feasibility, "even if such results could not be consistently demonstrated." *Id.* at 10. Nonetheless, in most cases, URS did not enhance controls or make other adjustments to account for this anticipated sampling variability. As in the case of the "trial-and-error" costs, URS' decision to exclude these "exposure variability"

costs from its analysis contributed additional conservatism to URS' own estimates of overall compliance costs.

(2) OSHA Has Likewise Underestimated the Cost for General Industry to Comply with the Proposed Ancillary Provisions.

In the proposed rule, OSHA includes a number of ancillary provisions with which employers must comply, in addition to adopting engineering controls and work practices. These include exposure monitoring, medical surveillance, worker training, designation of regulated areas, and the use of respirators for workers exposed above the PEL. In addition, OSHA has indicated that facilities in various industry sectors likely will have to be professionally cleaned each year in order to maintain exposures below the proposed PEL.[478] URS identified a number of flaws in OSHA's assumptions and analysis that result in a gross underestimate of the costs of the ancillary provisions. Once all of the errors in OSHA's analysis are corrected, the costs of the ancillary provisions are found to be *approximately 20 times higher* than OSHA's estimate. *See* URS Feasibility Report Table 4A.

First, OSHA adopted an overly optimistic assumption about the number of overexposed workers who will require respirators because engineering controls and work practices are insufficient to achieve the proposed PEL on a consistent basis. More than 40 years after OSHA first established the current PEL of 100 $\mu g/m^3$, OSHA reports that 81,000 workers in general industry and maritime (27% of silica-exposed workers in those categories covered by the rule) are still exposed above the current PEL. PEA III-50. Despite this long history demonstrating the challenges associated with reducing workplace exposures to

[478] *See* PEA at IV-80, 83, 91, 92 (concrete products), IV-166, 168, 173 (foundries), IV-232 (mineral processing), IV-245, 246, 247 (porcelain enameling), IV-262, 267, 270, 271 (pottery), and IV-357, 365, 366, 367, 368, 369 (structural clay).

respirable crystalline silica, OSHA inexplicably assumes that if it cuts the current PEL in half under the proposed standard, only 10% of workers currently exposed above the proposed PEL of 50 $\mu g/m^3$ would remain overexposed, meaning that only 12,247 workers would need respirators. PEA V-32; 78 Fed. Reg. at 56352 (122,472 general industry workers currently exposed above 50 $\mu g/m^3$). OSHA provides no evidence or rationale to support this assumption. Given the history of overexposure to crystalline silica as measured against a PEL of 100 $\mu g/m^3$, it is arbitrary, capricious, and fundamentally unreasonable for OSHA to assume that the rate of overexposures will fall by 90% when the PEL is cut in half. In fact, the opposite seems more likely to be the case, since it will be much harder to comply with a PEL of 50 $\mu g/m^3$ than a PEL of 100 $\mu g/m^3$. Relying on this arbitrary and counter-intuitive assumption caused OSHA to significantly underestimate the number of overexposed workers who would be subject to continued monitoring, medical surveillance, and respirator use. URS Feasibility Report at 21 (continued monitoring), 22 (respirator use), 23 (medical surveillance).

The arbitrary nature of OSHA's assumptions is exacerbated by OSHA's position that the PEL is a never-to-be exceeded standard, so that an employer is deemed to be out of compliance if an employee's exposure exceeds the PEL on any day it happens to be measured. Thus, reducing *average* exposures to a level of 50 $\mu g/m^3$ or below would not assure that respirators need not be worn in the workplace.[479] Given the variability of exposure for most industrial facilities, the number of employees that would be required to comply with these ancillary provisions due to occasional overexposure would be significant.

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[479] To ensure compliance with a never-to-be exceeded standard and avoid the need for respirators, URS estimates that the 95th percentile sampling measurements—not the average measurements—must be below the PEL. URS Feasibility Report at 10.

For example, representatives from the foundry sectors believe that between the challenges associated with achieving the new PEL and OSHA's interpretation of the "never-to-be exceeded" standard, as many as 60% of all foundry workers would be required to wear respirators to ensure that there are no instances of exposure above 50 $\mu g/m^3$.[480]

Recognizing these points, URS rejected OSHA's assumption that there would be a 90% reduction in the overexposure rate when the PEL is reduced from 100 $\mu g/m^3$ to 50 $\mu g/m^3$. Instead, URS assumed that the percentage of overexposed workers would be cut in half in each general industry sector, an assumption that is far more reasonable than OSHA's, yet is still quite conservative. URS Feasibility Report at 21, 22. The assumed overexposure rate varies by industry sector in the URS analysis to reflect the actual current exposure data. *Id.* Under URS' approach, the overall rate of overexposures requiring the use of respirators across general industry is approximately 13.5% at the proposed PEL of 50 $\mu g/m^3$ (*i.e.* one-half of the 27% currently exposed over 100 $\mu g/m^3$, *see* PEA III-50.).

Second, URS identified significant ancillary costs that were excluded from OSHA's analysis. In particular, OSHA mistakenly left out of its cost estimates the costs for professional cleaning, despite the fact that OSHA indicated that professional cleaning was necessary for several general industry sectors, including, among others, concrete products, mineral processing, pottery, and all of the foundry sectors. URS Feasibility Report at 24. URS corrected this oversight by including professional cleaning costs for each general industry sector that was identified in the PEA as a candidate for professional cleaning. *Id.*, Table 4A, note 3; *see also* PEA at IV-80, 83, 91, 92 (concrete products), IV-166, 168, 173 (foundries), IV-232 (mineral processing), IV-245, 246, 247 (porcelain enameling), IV-262,

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[480] See Comments of the American Foundry Society in Docket No. OSHA-2010-0034.

267, 270, 271 (pottery), and IV-357, 365, 366, 367, 368, 369 (structural clay). Correcting this oversight on OSHA's part accounts for approximately 60% of the difference between OSHA's cost estimate for the ancillary provisions and the URS estimate. *See* URS Feasibility Report, Table 4A, note 3.

Third, OSHA underestimated the costs for initial monitoring by assuming, without any foundation in the record, that only one-quarter of workers in jobs where they are at risk of potential overexposure to silica would actually be monitored. However, the proposed standard requires that every at-risk job should have at least one employee tested per facility per shift. URS Feasibility Report at 20. As URS explains, "[m]any small and very small facilities do not have a total of four workers in any at-risk job category, so that the proportion of workers in at risk jobs who will have to be monitored will be greater than one in four." *Id.* After applying URS' exposure profile, the number of employees subject to initial monitoring was increased, particularly for very small and small facilities. *Id.* at 20-21.

Fourth, OSHA assumes a uniform 10-year amortization period for all ancillary costs, which URS determined was inappropriate for certain ancillary provisions. For example, because OSHA requires initial exposure monitoring "any time there is a major change in production or control equipment for a process," URS applied a 5-year amortization period to better reflect the frequency with which such changes are likely to occur. *Id.* at 21. Likewise, because medical surveillance must occur every three years, "URS shortened the time for annualizing the costs of medical surveillance from the ten years used by OSHA to three years." *Id.* at 23.

Fifth, while URS generally applied OSHA's per-unit costs for ancillary provisions, URS did make a few adjustments in cases where communications with industry

representatives indicated that OSHA had underestimated the actual, real-world costs of implementing the requirements. For example, URS increased the time requirements for certified industrial hygienists (CIHs) to perform initial monitoring because "OSHA made no allowance for the CIH to draw conclusions based on the sampling and to write reports." URS Feasibility Report at 20. Likewise, URS increased the training costs associated with small class sizes, *id.* at 23, and the number of visitors to regulated areas, *id.*, to better reflect real-world conditions.

After accounting for the flaws in OSHA's assumptions and analysis, URS determined that the expected costs for general industry to implement the ancillary provisions in the proposed rule *were 20 times higher* than OSHA's estimates. For the 19 primary general industry sectors, the total expected costs for general industry to implement the proposed ancillary provisions increased from $36.4 million by OSHA's estimate to $777 million under the URS cost model. *See* URS Feasibility Report, Table 4A.

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In sum, when the costs to comply with the proposed rule are estimated properly, they are found to total more than $6 billion annually for 19 general industry sectors, an amount that is more than 40 times higher than the compliance cost of $132.5 million that OSHA estimated for all of general industry. Because OSHA has not constructed a reasonable estimate of compliance costs for the proposed standard, it cannot make a supportable determination that the proposed standard would be economically feasible in general industry.

2. OSHA Has Not Produced a Supportable Assessment of the Impact that the Proposed Standard Would Have on Affected General Industry Sectors.

The URS analysis described above demonstrates that OSHA's estimate of compliance costs for 19 general industry sectors lacks any supportable basis. OSHA's economic impact analysis compounds the flaws in its compliance cost estimate because it ignores an economic crisis that materially reduced the revenues and profits of virtually every industrial sector covered by the proposed standard. As a result, OSHA's analysis systematically undervalues the economic impact that the proposed standard would have.

To assess the economic impact of the proposed standard, the Panel retained Environomics, Inc. to evaluate OSHA's economic analysis, work with URS in developing corrected cost information, and prepare an economic impact assessment of the proposal. As Environomics points out, OSHA's economic feasibility analysis arbitrarily relies on general industry sector revenues and profits in the years prior to 2007, immediately before the most devastating recession this country has faced since the Great Depression. Specifically, OSHA uses as its revenue baseline data for 2006, while its profits baseline reflects the seven-year period from 2000 to 2006 that is arguably among the most successful periods for both construction and general industry. Environomics Report at 8-9. By selecting a pre-recession baseline period, which was the apex of economic prosperity for many general industry sectors, OSHA has minimized the real economic impact that would result from the proposed standard. Data from those time periods are not representative of more recent revenues and profits for construction or for general industry. The financial crisis and recession that followed had a profound effect on both the construction and manufacturing sectors (particularly those sectors that support the construction industry). *See id.* at 8 ("Many of the regulated general industries produce largely construction materials and products . . . and

these industries have suffered from the economy-wide recession and the very sharp decline in construction demand for their products.") Further, the U.S. recession was not an isolated event; other economies, most notably in Europe, were affected in a comparable manner. Even in higher growth areas, such as China, the rate of growth slowed significantly over the same time span. Thus the economic recession and related events reduced worldwide demand for many manufacturing products, reducing opportunities for export and intensifying global competition. While the economy is beginning to recover it is nowhere near 2006 levels. *Id.* at 9 (construction spending in 2013 was more than 20% less than in 2006).

In essence, the recession set a new, lower baseline for revenues and profits, leaving general industry sectors much more vulnerable to regulations that would increase compliance costs. Thus, a more realistic analysis of the expected feasibility and economic impacts of the proposed standard must consider more recent data that includes the financial crisis and at least a portion of the recession that followed. Environomics has corrected part of this shortcoming in OSHA's analysis by updating the general industry profits baseline relied on by OSHA. Unfortunately, OSHA's refusal to grant the full extension of time we and others requested has made it impossible for Environomics to update general industry revenue data in time for inclusion in these Comments. The Panel hopes to be able to submit an economic feasibility analysis using updated general industry revenue data as time permits between now and the date of our appearance at the Public Hearing.[481]

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[481] Environomics Report at 16. Environomics also determined that OSHA's revenue estimates are inappropriate because OSHA makes inappropriate assumptions about the relationship between revenues and payroll. Environomics intends to address that issue more fully in a future submission as well. *Id.* at 15-16.

Meanwhile, as discussed below, even when the revised annualized cost estimates prepared by URS are compared to OSHA's inflated revenue and profit numbers, the compliance costs in many general industry sectors are found to exceed OSHA's revenue and profit thresholds, often by a wide margin. Moreover, when the URS cost estimates are compared to updated profit data, the comparisons are even more dire – as in sector after sector, the proposed rule would threaten to consume *all* or very significant shares of general industry's profits. This not only demonstrates the insufficiency of OSHA's economic analysis, but calls into question the economic feasibility of the proposed standard in general industry.

Environomics evaluated the economic impact of the proposed standard by making a number of different comparisons, including:

- Comparing URS' *incremental* cost estimate to OSHA's revenue data.
- Comparing URS' *incremental* cost estimate to OSHA's profit data.
- Comparing URS' *incremental* cost estimate to revised and updated profit data.
- Comparing URS' *full* cost estimate to OSHA's revenue data.
- Comparing URS' *full* cost estimate to OSHA's profit data.
- Comparing URS' *full* cost estimate to revised and updated profit data.
- Comparing OSHA' *incremental* cost estimate to OSHA's revenue data.
- Comparing OSHA' *incremental* cost estimate to OSHA's profit data.
- Comparing OSHA' *incremental* cost estimate to revised and updated profit data
- Comparing OSHA's *full* cost estimate to revised and updated profit data

The results of this analysis, which are described in the Environomics Report[482] and shown in Tables 3, 5, and 6 below, are markedly different from OSHA's conclusions in the proposed rule.

a. Using Only Hypothetical *Incremental* Costs, the Economic Impact of the Proposed Standard Would Exceed OSHA's Thresholds for Multiple General Industry Sectors.

Comparison of URS' *Incremental* Costs to OSHA's Revenue and Profitability Data. Relying on URS' revised cost estimates, Environomics first compared URS' *incremental* costs of the proposed standard to OSHA's own revenue and profit data for each industry sector. Most of the 19 general industry sectors evaluated by URS exceeded at least one of OSHA's thresholds. For 13 of the 19 general industry sectors evaluated by URS, annualized incremental compliance costs would exceed 1% of revenues, while for 15 of the 19 general industry sectors, annualized incremental costs would exceed 10% of profits. Data for industry sectors exceeding one or both of the thresholds are included in Table 3 below.

Thus, based on the URS *incremental* cost data, OSHA, at a minimum, must conduct a more detailed analysis of the competitive structure and vulnerabilities of each of the sectors that exceed the annualized revenue or profit thresholds before it can make a final determination regarding the economic feasibility of the proposed standard. *See* 78 Fed. Reg. at 56367. However, given the degree to which OSHA's thresholds are exceeded in some sectors, OSHA should conclude that, for those sectors, the proposed standard is not feasible, even without further analysis. In the *Hexavalent Chromium* case, OSHA determined that a standard was not economically feasible if the annualized costs exceeded 2.7% of revenues or 65% of profits. *See Hexavalent Chromium*, 557 F.3d at 172. Here, eight of the general

482 Environomics Report at 14-15 & Tables 4, 5.

industry sectors exceed both of those thresholds even on an *incremental* annualized cost basis, thereby indicating that the proposed standard is *prima facie* infeasible. *See* Table 3 (sectors exceeding *Hexavalent Chromium* thresholds noted in bold). Moreover, for six of those sectors, annualized costs exceed 100% of profits, suggesting quite directly that the proposed standard would surely "threaten the existence or competitive structure of … [the] industry" or imperil its long-term profitability.[483]

Table 3: Comparison of URS Incremental Costs to OSHA Revenue and Profit Estimates

Industry Sector	URS Incremental Costs as % of Revenue (OSHA estimate)	URS Incremental Costs as % of Profits (OSHA estimate)
Asphalt Paving Products	0.04%	0.56%
Asphalt Roofing Materials	1.62%	21.61%
Concrete Products	**3.61%**	55.62%
Costume Jewelry	0.25%	4.25%
Cut Stone	**3.68%**	67.01%
Fine Jewelry	0.23%	3.97%
Flat Glass	0.45%	13.08%
Iron Foundries	**9.02%**	**219.23%**
Mineral Processing	**4.93%**	**89.83%**
Mineral Wool	1.19%	21.67%
Nonferrous Sand Casting Foundries	**13.30%**	**323.24%**
Non-sand Casting Foundries	**11.34%**	**275.54%**
Other Ferrous Sand Casting Foundries	**7.66%**	**186.29%**
Other Glass Products	0.51%	14.98%
Paint and Coatings	0.27%	4.96%
Pottery	**14.29%**	**323.62%**
Ready Mix Concrete	1.35%	20.39%
Refractories	2.68%	60.79%
Structural Clay	**8.13%**	**184.10%**

[483] *Hexavalent Chromium*, *supra,* 557 F.3d at 177 (3d Cir. 2009), quoting *United Steelworkers*, *supra,* 647 F.2d. at 1272.

b. Using the *Full* Costs of Compliance, the Proposed Standard Clearly Is Not Economically Feasible.

As described above, the actual economic feasibility of the proposed standard should be measured by including *all* costs that actually must be incurred to achieve the proposed PEL. In cases such as this, where OSHA acknowledges widespread non-compliance with the existing PEL of 100 μg/m^3, there is no basis for ignoring the costs that firms will have to incur to reduce exposures to that level. In the PEA, OSHA specifically addresses the issue of non-compliance with the existing standard. *See* PEA Table III-5. The vast majority of job categories showed some exposure above the current PEL of 100 μg/m^3. *Id.* As Table 4 below shows, for 9 general industry job categories, at least half of the workers were exposed above the current PEL.

Table 4: Job Categories With Overexposures Exceeding 50%; Data excerpted from PEA, Table III-5.

Sector	Job Category	100-250 μg/m3	> 250 μg/m3	% over-exposure
Concrete Products	Abrasive Blasting Operator	26.7%	33.3%	60%
Flat Glass	Material Handler	33.3%	16.7%	50%
Mineral Wool	Material Handler	33.3%	16.7%	50%
Other Glass Products	Material Handler	33.3%	16.7%	50%
Pottery	Coatings Operator	32.4%	21.6%	54%
Pottery	Coatings Preparer	26.3%	31.6%	57.9%
Ready Mix	Truck Driver	0%	100%	100%
Structural Clay	Forming Line Operator	28.6%	42.9%	71.5%
Structural Clay	Grinding Operator	28.6%	21.4%	50%

In total, OSHA estimates that 81,000 employees in the general industry and maritime sectors are currently exposed above the existing general industry PEL of 100 $\mu g/m^3$. 78 Fed. Reg. at 56347; PEA at III-50. The costs to reduce exposures to 100 $\mu g/m^3$, therefore, undoubtedly would have a significant impact on the competitive structure and even economic viability of several general industry sectors. They should not be ignored.

Comparison of *Full* Compliance Costs to OSHA's Revenue and Profit Data. In light of the potentially significant economic impact of those costs, Environomics also prepared an economic impact analysis that included the *$6 billion* in *full* compliance costs that URS determined would be incurred across 19 general industry sectors to comply with the proposed standard. The results of that analysis, comparing the *full* costs of complying with the proposed standard in general industry to OSHA's own revenue and profit data for each industry sector, are shown in Table 5 below. In this comparison, 13 of the 19 general industry sectors exceeded OSHA's revenue threshold, and 16 of 19 exceeded OSHA's profit threshold. A more detailed analysis of these industries would be unnecessary here, given the magnitude of the full annualized costs the proposed rule would impose, even when compared to OSHA's inflated revenue and profit values – because nine of the 19 sectors exceed the thresholds for infeasibility applied in the *Hexavalent Chromium* case. *See* Table 5 (sectors exceeding 2.7% of revenues or 65% of profits marked in bold). Indeed, for several sectors, those thresholds are exceeded by more than an order of magnitude. (Nonferrous Sand Casting Foundries (18.05% of revenues and 438.67% of profits), Non-Sand Casting Foundries (15.72% of revenue and 382.19% of profits), and Pottery (18.94% of revenue and 429.02% of profits)). The sheer magnitude by which these general industry sectors exceed

OSHA's own thresholds leaves no question that the high costs of the proposed standard make it economically infeasible in general industry.

Table 5: Comparison of URS Full Costs to OSHA Revenue and Profit Estimates

Industry Sector	URS Full Costs as % of Revenue	URS Full Costs as % of Profits (OSHA estimate)
Asphalt Paving Products	0.04%	0.56%
Asphalt Roofing Materials	2.37%	31.60%
Concrete Products	**4.15%**	63.88%
Costume Jewelry	0.28%	4.88%
Cut Stone	**4.39%**	**80.05%**
Fine Jewelry	0.26%	4.56%
Flat Glass	0.58%	16.90%
Iron Foundries	**12.76%**	**310.16%**
Mineral Processing	**5.48%**	**99.91%**
Mineral Wool	1.42%	25.90%
Nonferrous Sand Casting Foundries	**18.05%**	**438.67%**
Non-sand Casting Foundries	**15.72%**	**382.19%**
Other Ferrous Sand Casting Foundries	**10.75%**	**261.39%**
Other Glass Products	0.70%	20.40%
Paint and Coatings	0.32%	6.01%
Pottery	**18.94%**	**429.02%**
Ready Mix Concrete	1.39%	20.98%
Refractories	2.93%	66.43%
Structural Clay	**11.70%**	**265.04%**

c. Updating and Correcting OSHA's Profit Data Further Confirms that the Proposed PEL Clearly Is Infeasible.

In order to obtain a more realistic picture of the economic impact that the proposed standard would have on affected industries, OSHA's pre-2007 revenue and profit baselines need to be updated and revised. While Environomics has not yet had an opportunity to update the revenue data, it has been able to revise and update the profit data for general industry. And, when the URS incremental and full annualized cost estimates are compared to the revised profit data, almost every industry sector is found to materially exceed OSHA's

10% profit threshold. In fact, even when OSHA's own incremental cost estimates are compared to the revised profit data, three general industry sectors are found to exceed the 10% profit threshold. When OSHA's full cost estimates are compared to the revised profit data, seven of the general industry sectors exceed the 10% threshold.

Environomics developed the revised and updated profit data for the 19 general industry sectors by making the following two changes in the data used by OSHA.[484] First, Environomics updated OSHA's profit data (which covered calendar years 2000-2006) by adding data for calendar years 2007-2010, so that a total of 11 years of profit data (2000-2010) are considered in the Environomics analysis. This expanded baseline provides a far more robust view of the profitability of these general industry sectors over time, because the data set includes both the upswings *and* the downturns our economy has experienced in recent years. Second, Environomics' profit calculations include both profits *and* losses. In estimating industry profits, OSHA inexplicably excluded businesses that showed no profits or a deficit during a particular year. It is hard to understand how a full and accurate picture of the effect of a proposed rule on industry sectors can be evaluated if OSHA cherry-picks only those facilities from within a sector that happen to show a profit. Indeed, the firms that OSHA excluded from its analysis on this basis are precisely the ones that are most vulnerable to the financial stress that would result from having to comply with the proposed standard.

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[484] Environomics also noted that OSHA's reliance on the IRS *Corporate Source Book* was problematic because it organizes facilities by 4-digit NAICS codes that are much broader than the 6-digit NAICS codes OSHA uses in the Proposal. *See* Environomics Report at 9-11. Environomics intends to explore alternative sources that may provide profitability data at a more granular level, but there was insufficient time to complete that exercise before the filing deadline for these Comments.

By excluding the profit/loss results of those firms from its analysis, OSHA effectively has already altered the competitive structure of the industry.

Using these updated and adjusted data, Environomics has prepared further calculations that compare URS' Incremental Costs, URS' Full Costs, OSHA's Incremental Costs, and OSHA's Full Costs estimates to the more robust profit data. As detailed in the Environomics Report and shown in Table 6 below, these comparisons likewise establish that the proposed standard is economically infeasible for general industry as a whole. Specifically:

- Using URS' *incremental* cost estimates,15 of the 19 general industry sectors exceed OSHA's 10% profit threshold, and 9 of them exceed the 65% profit threshold applied in the *Hexavalent Chromium* case.
- Using URS' *full* cost estimates,15 of the 19 general industry sectors likewise exceed OSHA's 10% profit threshold, and 11 of them exceed the 65% profit threshold applied in the *Hexavalent Chromium* case.
- Using OSHA's *incremental* cost estimates, 3 of the 19 general industry sectors exceed OSHA's 10% profit threshold.
- Using OSHA's *full* cost estimates, 7 of the 19 general industry sectors exceed OSHA's 10% profit threshold, and one exceeds the 65% profit threshold applied in the *Hexavalent Chromium* case.

Table 6: Comparison URS and OSHA Cost Estimates to Environomics' Revised Profit Estimates

Industry Sector	URS Incremental Costs as a % of Profits (revised profit estimate)	URS Full Costs as a % of Profits (revised profit estimate)	OSHA Incremental Costs as a % of Profits (revised profit estimate)	OSHA Full Costs as a % of Profits (revised profit estimate)
Asphalt Paving Products	0.59%	0.59%	0.04%	0.04%
Asphalt Roofing Materials	22.76%	33.29%	0.58%	0.99%
Concrete Products	165.63%	190.24%	4.56%	9.15%
Costume Jewelry	6.75%	7.75%	0.81%	1.11%
Cut Stone	184.14%	219.99%	11.55%	24.82%
Fine Jewelry	6.31%	7.25%	0.68%	0.97%
Flat Glass	19.46%	25.15%	0.33%	0.48%
Iron Foundries	252.35%	357.01%	4.13%	10.03%
Mineral Processing	246.85%	274.55%	9.81%	13.64%

Mineral Wool	59.55%	71.16%	0.90%	1.34%
Nonferrous Sand Casting Foundries	372.06%	504.93%	5.20%	12.56%
Non-sand Casting Foundries	317.16%	439.92%	4.85%	11.74%
Other Ferrous Sand Casting Foundries	214.44%	300.87%	3.32%	8.05%
Other Glass Products	22.30%	30.37%	0.97%	1.47%
Paint and Coatings	6.38%	7.72%	0.04%	0.27%
Pottery	1512.30%	2004.83%	23.04%	46.46%
Ready Mix Concrete	62.04%	63.82%	2.55%	3.13%
Refractories	284.08%	310.45%	4.51%	8.51%
Structural Clay	860.31%	1238.54%	35.30%	95.56%

In an effort to provide further support for its conclusion that the proposed standard is economically feasible, OSHA compares its annualized cost estimates to yearly variations in producer prices charged by general industries[485] and their average profitability.[486] By making these comparisons, OSHA implies that even if there were increases in costs and/or reductions in profitability due to the proposed RCS standard, the general industry sectors could still absorb those changes without undue impact on their continued existence. These comparisons, however, are not sound economic analyses, and OSHA should not rely on them. The year-by-year changes in prices and profitability cited by OSHA include both increases and decreases and, therefore, say very little about the overall economic health of a particular industry or its ability to withstand a dramatic and sustained increase in compliance costs. As Environomics explains, "year-to-year fluctuations in an industry's profitability, or the lack of such fluctuations, are not particularly important to the industries' long-term economic health. What is important is the longer-term trend in profitability, notwithstanding whatever fluctuations occur." Environomics Report at 7. In contrast to these yearly

485 PEA at VI-27-51, Tables VI-2 & VI-3.

486 PEA at VI-52-72, Tables VI-4 & VI-5.

fluctuations, the costs to comply with the proposed standard are *annualized* and thus will have a consistent, permanent negative effect on each facility's revenues and profits year after year. Further, if facilities are faced with lower profits and revenues as a result of the proposed standard, they would have less capacity to tolerate large year-to-year swings in product prices and profitability. In sum, evidence of short-term volatility within an industry sector is of little value in projecting what will happen when a new regulation resets the baseline for profits and revenue.

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In conclusion, OSHA has not demonstrated that the proposed standard is economically feasible for the vast majority of general industry sectors. As discussed above, and as explained at greater length in the attached reports from URS and Environomics, OSHA's economic feasibility analysis grossly underestimates the cost of complying with the proposed standard and overestimates the revenues and profitability of the general industry sectors on which the compliance costs would be imposed. Due to these analytical errors, OSHA lacks a supportable basis for determining that the costs of the proposed standard "will not threaten the existence or competitive structure" of the affected general industry sectors or that those costs will not imperil the long-term profitability of those sectors.[487] At a minimum, OSHA must develop new and more realistic compliance cost estimates to evaluate the economic impact of the proposal on the various affected industry sectors. However, based on the analyses conducted by URS and Environomics, additional study by OSHA may be superfluous, because it is clear that the costs of the proposed standard would far exceed

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[487] *Hexavalent Chromium*, *supra,* 557 F.3d at 177 (3d Cir. 2009), quoting *United Steelworkers*, *supra,* 647 F.2d. at 1272.

the profitability and revenue thresholds that OSHA applied in the Hexavalent Chromium proceeding to determine that a PEL being considered in that rulemaking would not be economically feasible. This is true regardless of whether one uses the *full* costs of compliance in making the comparison to revenue and profits or uses only the hypothetical *incremental* costs in making the comparisons. In either case, the results lead to the conclusion that a respirable crystalline silica standard having a PEL of 50 mg/m^3 would not be economically feasible in most general industry sectors.

C. The Proposed Standard Also Is Infeasible Because of Measurability Problems at Silica Concentrations of 50 µg/m^3 and Below.

As OSHA acknowledges, for a standard to be technologically feasible, "available methods for measuring worker exposures [must] have sufficient sensitivity and precision to ensure that employers can reliably evaluate compliance with the standard and that workers have a reasonably accurate assessment of their exposure to hazardous chemicals."[488] Although OSHA would like to believe otherwise, there is in fact a serious question as to whether crystalline silica can be sampled and analyzed accurately, precisely and reliably at airborne concentrations below 100 µg/m^3. This question is of particular concern because, as Steve Edwards of U.S. OSHA's Salt Lake Technical Center observes: "Sampling and analysis of crystalline silica present unique problems to the industrial hygienist."[489] Those problems make it exceedingly difficult to measure reliably (with an acceptable degree of precision and accuracy) the small mass of silica that is collected by 8-hour personal sampling when airborne concentrations of respirable silica fall below 100 µg/m^3.

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488 PEA at IV-13.

489 Edwards, S.L., Crystalline Silica: Sampling and Analytical Issues. *The Synergist* (December 2000).

OSHA standards typically require that employers use a method of monitoring and analysis that has an accuracy of plus or minus 25 percent (+/- 25%) with a confidence level of 95 percent for measurements at airborne concentrations at or above the PEL.[490] This reflects the NIOSH Accuracy Criterion, which "requires that, over a specified concentration range, the method provide a result that differs no more than ±25% from the true value 95 times out of 100."[491] And, as NIOSH explains, the relevant concentration range for this purpose generally is "a range of concentrations bracketing the permissible exposure limit (PEL)" – so that accuracy within 25% of the true value in 95% of measurements can be "assured both at levels below the PEL for possible use in action level determinations and, more significantly, at the PEL itself, where method results must be legally defensible."[492] In fact, this NIOSH "accuracy criterion was devised as a goal for the development and acceptance of sampling and analytical methods capable of generating reliable exposure data for contaminants at or near the Occupational Safety and Health Administration's (OSHA) permissible exposure limits."[493]

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[490] See, *e.g.*, 29 CFR § 1910.1028(e)(6) (+/- 25% for Benzene); 29 CFR § 1910.1025(d)(9) (+/- 20% at three-fifths of the PEL for Lead); 29 CFR § 1910.1027(d)(6) (+/-25% for concentrations at or above the action level for Cadmium); 29 CFR § 1910.1026(d)(5) (+/-25% for concentrations at or above the action level for Hexavalent Chromium).

[491] NIOSH Manual of Analytical Methods (January 15, 1998) at 36.

[492] Key-Schwartz, R. *et al.*, "Determination of Airborne Crystalline Silica," in NIOSH Manual of Analytical Methods, 4th rev. ed. Cincinnati, OH, US Dep't of HHS, Public Health Service, Centers for Disease Control and Prevention, NIOSH, DHHS (NIOSH) Publication No. 03-127, at 273.

[493] See 61 Fed. Reg. 10012, 10013 (March 12, 1996).

To achieve 95 percent confidence that a measurement is accurate within a range of +/- 25%, the total coefficient of variation (CV_T) or relative standard deviation (RSD) for exposure sampling and analysis combined must be no greater than 12.8%.[494] Achieving that level of accuracy and precision "requires sensitive and accurate sampling and analytical methods to detect and quantify crystalline silica *in the presence of other types of dust*."[495] Using the Dorr-Oliver sampler with a flow rate of 1.7 liters/minute (as specified by OSHA Method ID-142), silica exposures below 100 $\mu g/m^3$ cannot be reliably measured with that level of accuracy and precision – because the mass of silica collected in 8 hours at a flow rate of 1.7 liters/minute (about 80 micrograms when the silica concentration is 100 $\mu g/m^3$, 40 micrograms when the silica concentration is 50 $\mu g/m^3$, and 20 micrograms when the silica concentration is 25 $\mu g/m^3$) is too small to perform accurate and precise measurements that meet the NIOSH accuracy criterion.

OSHA seems to have recognized this point implicitly when it developed the present proposal. Thus, the proposed standard – in contrast to OSHA's typical approach[496] – does not require the use of a method of monitoring and analysis that has an accuracy of plus or minus 25 percent with a confidence level of 95 percent for measurements in the range of the

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494 See Leidel, N.A. *et al.*, Occupational Exposure Sampling Strategy Manual. DHEW (NIOSH) Publication No. 77-173 (1977) (Docket Item # OSHA-2010-0034-1490) at 78. CV_T is calculated as the square root of the sum of the squares of the CV for sampling (generally assumed by OSHA to be 0.05 or 5%) plus the CV for the analytical method. See *id.* at 81. See also Leidel, N.A., Exposure Measurement Action Level and Occupational Environmental Variability. HEW Publication No. (NIOSH) 76-131 (1975) (Docket Item # OSHA-2010-0034-1501) at 3. The 5% assumed CV for sampling covers expected sampling pump error (flow rate variability). It does not encompass variability in the sampling process itself or inter-sampler variability.

495 PEA at IV-15 (emphasis supplied).

496 See 78 Fed. Reg. at 56448.

proposed PEL or action level. Instead, OSHA states that it is feasible to measure respirable crystalline silica exposures at the proposed PEL of 50 μg/m^3 "with a *reasonable degree of precision and accuracy*."[497] But it does not say what it considers to be "sufficient sensitivity and precision" for these purposes or what is required for a measurement to be deemed "reasonably accurate" and to reflect "a reasonable degree of precision and accuracy." Only inferentially does OSHA suggest that laboratories should be able to produce results that are within +/-25% of the reference value in order to be said to "achieve reasonably good agreement in their analytical results," but it seems to have abandoned the requirement that they be able to do so with a confidence level of 95 percent for measurements at airborne concentrations at or above the PEL.[498] Given the difficulties of sampling and analyzing crystalline silica accurately and precisely when exposure levels are in the range of 25 μg/m^3 - 50 μg/m^3, OSHA's failure to get specific on these points is not surprising.

The problems and limitations of sampling and analyzing such low airborne concentrations of crystalline silica are discussed at some length in a Report prepared by Sandra C. Wroblewski, CIH, of Computer Analytical Solutions. A copy of her Report, entitled *Silica Sampling and Analytical Concerns*, is submitted as Attachment 10 hereto. The Report shows that it is not possible to measure crystalline silica exposures below 0.1 mg/m^3 reliably using the 37 mm cassette sampling protocol (10-mm Dorr Oliver cyclone) that is typically employed for that purpose in North America and the existing analytical methods—notably, X-ray Diffraction ("XRD"), as exemplified by U.S. OSHA Method ID-

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497 *Id.* at 56446 (emphasis supplied).

498 See PEA at IV-38 (noting that labs in the PAT program came within +/-25% of the reference value just 80% of the time).

142 and NIOSH Method 7500, and Infrared Spectroscopy ("IR"). The highlights of the Wroblewski Report are as follows:

- OSHA Analytical Method ID-142 references a Precision and Accuracy Validation Range of 50-160 μg quartz per sample. For a Dorr Oliver sampler with a recommended flow rate of 1.7 L/min, 50-160 μg quartz per sample represents an air concentration range of 0.061 mg/m^3 - 0.196 mg/m^3. This may be an appropriate range to consider when evaluating method performance for an OEL of 100 μg/m^3. But even the lower end of this range is higher than the proposed PEL of 50 μg/m^3 and more than twice as high as the proposed action level, so Method ID-142 has not even been tested for validation in a range relevant to what OSHA has proposed here.

- OSHA's Inorganic Methods Protocol states that a validated method must have a pooled CV_1 (coefficient of variation) of 0.07 or less for data in the range of 0.5 x the OEL to 2 x the OEL. OSHA ID-142 references a CV_1 of 0.106 for loadings in the range of 50 μg to 160 μg of quartz. Thus, OSHA ID-142 does not appear to be acceptable even for an PEL of 100 μg/m^3, let alone for a PEL of 50 μg/m^3 and an action level of 25 μg/m^3 where the CV_1 presumably would be higher than 0.106.

- OSHA's Inorganic Methods Protocol states that the quantitative detection limit for an analytical method should be less than 0.1 times the PEL (or the mass equivalent of the PEL). For a PEL of 50 μg/m^3, the quantitative detection limit would have to be 4 micrograms of quartz (assuming an 8-hour sample is collected at a flow rate of 1.7 L/min). OSHA ID-142 lists a quantitative detection limit of 10 micrograms for quartz. That value is 2½ *times higher* than the detection limit that would be required for a PEL of 50 μg/m^3.

- NIOSH's goal for analyses of silica under the Proficiency Analytical Testing ("PAT") Program is a Relative Standard Deviation ("RSD") of <15%. The RSD for silica in PAT Rounds 71-138 ranges from 15.3% to 45.4%. For the more recent subset of these PAT rounds (Rounds 98-138), the range was 15.3% to 37%. Even in Rounds 130-133, the range of RSDs was 16% to 33%. All of these are above the target RSD of <15%. Moreover, the PAT sample weight range is 50-175 μg/filter. At a flow rate of 1.7 L/min, this represents a working range for airborne concentrations of 61 μg/m^3- 0.214 mg/m^3. So, even for silica samples that are well above the concentration range that would be relevant to a PEL of 50 μg/m^3, PAT Program results consistently show RSDs that are well above the level NIOSH considers acceptable.

- NIOSH researchers analyzed silica data from PAT Rounds 101-132 (1990-1998). Based on an analysis of reported measurements by the reference labs, the NIOSH researchers found that the overall <u>intra</u>-laboratory CV for XRD analytical methods in these rounds was 0.165, while for IR methods it was 0.166. These

CV values are based solely on *analytical* variance; they do not reflect *sampling* error, since the participating labs received pre-loaded filters from the generating lab. Assuming a 5% sampling error, and applying the formulas used by NIOSH to calculate method performance, the "Overall Precision" of XRD based on these data would be 17.2% and the "Accuracy" would be ±34%. The comparable values for IR would be 17.3% and ±34%. These are well above the values that OSHA would deem to reflect reliable and reproducible measurements of crystalline silica. Yet the mass of silica analyzed in the PAT studies was considerably greater than the mass that would be collected when airborne concentrations are at the level of 50 $\mu g/m^3$ (assuming an 8-hour sample is collected at a flow rate of 1.7 L/min).

- In their analysis of silica data from PAT Rounds 101-132 (1990-1998), NIOSH researchers found that all estimates of intra- and inter-laboratory variability tended to rise at low sample loadings, with the range of 60-80 μg silica per sample being a significant cut-point. At a flow rate of 1.7 L/min, 60-80 μg silica is equivalent to 8-hour exposure to a silica concentration of 74 $\mu g/m^3$ - 98 $\mu g/m^3$. Thus, the NIOSH analysis indicates there would be a significant increase in measurement variability for a PEL of 50 $\mu g/m^3$.

- For PAT Rounds 124-139, the average RSD (Relative Standard Deviation) with outliers excluded was 24.6 for XRD methods and 21.1 for IR methods. For silica loadings in the range of 50 μg - 69 μg (the lowest range analyzed), the average RSD with outliers excluded was 27.8 for XRD methods and 22.7 for IR. These RSD values reflect only analytical variability, since PAT Program participants are provided with pre-loaded filters. Even so, they clearly are unacceptably high. And, of course, they would be higher still if the mass of silica being analyzed reflected 8-hour sampling of respirable silica concentrations in the neighborhood of 50 $\mu g/m^3$ (where the resulting silica mass would be roughly 40 μg).

As the Wroblewski Report shows, the PAT Program results and the performance data that U.S. OSHA provides for Method ID-142 indicate that current sampling and analytical methodologies do not provide a basis for reliably measuring airborne silica concentrations at a level of 50 $\mu g/m^3$ with an acceptable degree of accuracy and precision. Accordingly, compliance (or non-compliance) with the proposed PEL of 50 $\mu g/m^3$ and with requirements triggered at the action level of 25 $\mu g/m^3$ could not be determined with a reasonable level of confidence.

Experts in crystalline silica analysis at NIOSH explain some of the reasons for this. They note that "[t]he measurement of airborne crystalline silica can be challenging" – in part because of the difficulties associated with sample preparation (including complex procedures to reduce mineral interferences and re-depositing the sample on an analytical filter), the need for appropriate calibration, and the choice of standard reference materials.[499] "Redeposition of the sample [which occurs in NIOSH 7500; OSHA ID-142; MSHA P-2] is difficult to perform at low sample loadings."[500] And it is important to match particle size and phase purity of calibration standards with field samples in order to minimize analytical bias (*e.g.*, the infrared absorption response is particle size dependent, increasing as particle size decreases).[501] In short, as NIOSH points out: "Accurate and sensitive measurement of crystalline silica is complex. A high degree of attention is required throughout the analysis."[502] Moreover, "[a] high level of analyst expertise is required to optimize instrument parameters and correct for matrix interferences either during the sample preparation phase or the data analysis and interpretation phase."[503] For these reasons, among others, the NIOSH experts conclude that "current analysis methods [NIOSH 7500; OSHA ID-142; MSHA P-2] do not have sufficient accuracy to monitor below current exposure standards" (which are an

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499 Key-Schwartz, R. *et al.*, "Determination of Airborne Crystalline Silica," in NIOSH Manual of Analytical Methods, 4th rev. ed. Cincinnati, OH, US Dep't of HHS, Public Health Service, Centers for Disease Control and Prevention, NIOSH, DHHS (NIOSH) Publication No. 03-127, at 266.

500 *Id.*

501 See *id.*

502 *Id.* at 275.

503 *Id.* at 270.

OSHA PEL of 100 μg/m^3 and a NIOSH REL of 50 μg/m^3) using a 1.7 L/min Dorr-Oliver cyclone sampler.[504]

NIOSH's concern about the ability to measure silica concentrations accurately and reliably at levels in the neighborhood of 50 μg/m^3 was echoed by a Working Group of the International Organization for Standardization (ISO), which noted that it is becoming increasingly difficult for current methods and instruments to accurately and reliably measure new, lower exposure limits where the mass measured on the filter is <50 micrograms.[505] As the Working Group observes, analysis of silica mass at that level (which is about the mass collected in 8-hour sampling at 2 liters/minute where the air concentration is 50 μg/m^3) results in relatively large measurement errors (>25% at 2 sigma).[506] Thus, the sensitivity of some measurements on samples of air with a concentration <50 μg/m^3 using current sampling apparatus at about 2 L/minute is poor.[507] Furthermore, the Working Group points out, the analysis also becomes more problematic at that level because the analyst cannot be confident about the presence of silica unless additional confirmatory evidence is available.[508] No wonder the British Health and Safety Commission observed: "Due to the limitations of

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504 *Id.* at 265.

505 See Stacey, P. *et al.* (ISO Working Group ISO/TC146/SC2/WG7), An International Comparison of the Crystallinity of Calibration Materials for the Analysis of Respirable alpha-Quartz Using X-Ray Diffraction and a Comparison with Results from the Infrared KBr Disc Method. Ann. Occup. Hyg. 2009; 53: 639-649.

506 See *id.*

507 See *id.*

508 See *id.*

current methods for airborne measurement, it may be difficult . . . to enforce a WEL [workplace exposure limit] of 0.05 mg/m^3."[509]

To justify its contention that RCS exposures can be measured reliably and accurately with an acceptable degree of precision at an exposure level of 50 μg/m^3, OSHA makes a number of arguments, none of which withstands analysis. To begin, OSHA contends that analytical methods have sufficient sensitivity to measure the mass of respirable silica that would be collected by a cyclone sampler in 8-hour sampling at a flow rate of 1.7 liters/minute when the RCS exposure is 50 μg/m^3, *i.e.*, a silica mass of approximately 40 μg, and when the RCS exposure is 25 μg/m^3, *i.e.*, a silica mass of approximately 20 μg. OSHA's Method ID-142 has a stated Limit of Quantification (LOQ) of 10 μg for quartz and 30 μg for cristobalite. Since these LOQs are less than 40 μg (and, in the case of quartz, less than 20 μg), OSHA contends "that the XRD and IR methods of analysis are both sufficiently sensitive to quantify levels of quartz that would be collected on air samples taken from concentrations at the proposed PEL and action level."[510] But, according to OSHA: "The LOQ is the lowest amount of analyte that can be reliably measured in a sample *with acceptable analytical precision and recovery*,"[511] – and OSHA has provided no data to indicate that analytical precision and recovery are acceptable when the mass of silica analyte is 10 μg. The fact that silica can be detected (and perhaps even quantified to a limited extent) when the mass is 10 μg does not mean it is being *reliably measured with acceptable analytical precision and*

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509 British Health and Safety Commission, Control of Substances Hazardous to Health Regulations 2002 (as amended 2005) - Proposal for a Workplace Exposure Limit for Respirable Crystalline Silica: Consultative Document at 15.

510 See PEA at IV-32.

511 *Id.* (emphasis supplied).

recovery. Furthermore, while the theoretical LOQ or limit of detection (LOD) may be at the level stated in the Method when a pure silica standard is being analyzed under ideal conditions of method development "where substances that interfere with the analysis are not present," the actual LOQ and LOD in the real world is likely to be higher – as "the presence of interferences increases the potential error because additional measurements have to be made to compensate for changes to the background under the measurement peak or changes to the peak profile because of coinciding peaks (Stacey, 2007)."[512] A recent laboratory performance study discussed at pages 219-223 below also calls into question the putative LOQ and LOD values referenced by OSHA. In that study, the laboratories reported non-detected levels of silica for 34% of the filters having silica loadings of 20 micrograms or more.

OSHA next attempts to show that the sampling and analytical methods have acceptable precision when measuring the 40 μg and 20 μg mass equivalents of 8-hour exposure at the proposed PEL of 50 $\mu g/m^3$ and the proposed action level of 25 $\mu g/m^3$. But its efforts to make such a showing fall well short of success. For one thing, there is confusion as to what the relevant metric should be. OSHA begins with the following statement:

> The term precision refers to the amount of random error or variation in replicate measurements of the same sample, and is often expressed as a standard deviation about the mean of the measurements (denoted as S_T). When random errors are normally distributed, a 95-percent confidence interval can be calculated [as the mean ± (1.96 x the standard deviation)]. . . . The relative standard deviation (RSD), calculated by dividing the standard deviation by the mean for a data set, is often used to estimate error for analytical methods. The RSD is also known as the coefficient of variation (CV).[513]

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512 See Comments of Cardno ChemRisk on OSHA's Discussion of the Adequacy of Sampling and Analytical Methods for Measuring Respirable Crystalline Silica at Exposure Levels of 25 and 50 $\mu g/m^3$ ("Cardno Comments"), January 27, 2014, at 4, 5. The Cardno Comments are submitted herewith as Attachment 11.

513 PEA at IV-33.

OSHA then goes on to refer to another "statistic called the Sampling and Analytical Error (SAE) [which is used] to estimate the precision of air sampling and analytical methods *to assist compliance safety and health officers (CSHOs) in determining compliance with an exposure limit*."[514] In contrast to the term "precision" – which is calculated as a *two-sided* 95-percent confidence interval – the SAE is calculated as a *one-sided* 95 percent confidence limit. Use of a *one-sided* 95 percent confidence limit in calculating the SAE may be understandable, since its purpose is "*to assist compliance safety and health officers (CSHOs) in determining compliance with an exposure limit*," a situation in which precision is relevant in only one direction – *i.e.*, to determine whether the exposure limit has been exceeded and to do so on a statistical basis that can be supported if it is challenged before the Occupational Safety and Health Review Commission or in court. But in determining the precision of an analytical method, variability in *both directions* is relevant, because a reported analytical measurement may be either high or low – so a *two-sided* 95-percent confidence interval is appropriate. The same is true in the case of exposure measurements being made by an employer – *i.e.*, it is just as important for the employer to have confidence that the reported measurement is not inaccurately low as to know that it is not inaccurately high; otherwise, the employer could be overexposing his workers without knowing it. The NIOSH Accuracy Criterion reflects this point, specifying that "accuracy and precision is ±25% at a 95% confidence, which indicates the need for a two-sided confidence limit."[515] Accordingly, OSHA should not use the SAE "when making statements about the existing

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514 See *id.* (emphasis supplied).

515 Cardno Comments at 5.

sampling and analysis methods being sufficiently sensitive and precise," because, as OSHA calculates that statistic, it is not an appropriate measure of precision.[516]

To support its contention that accuracy and precision remain acceptable down to silica filter loadings of 20 μg (equivalent to exposure concentrations of 25 μg/m^3), OSHA relies almost entirely on a single study conducted at its Salt Lake Technical Center (SLTC) lab in March 2013. OSHA describes the study as follows:

> For quartz, two sets of 10 replicate filters were prepared with loadings of 21.0 and 40.6 μg using NIST standard quartz reference material SRM 1878a. For cristobalite, filter loadings of 20.0 and 40.0 were prepared using NIST SRM 1879a. The spiked filters were prepared and analyzed at SLTC using a Rigaku ultraX 18-kilowatt (kW) rotating-anode X-ray diffractometer. The mass of crystalline silica detected on the filter was quantified based on the area of the primary peak (i.e., the most sensitive peak) as compared with a standard calibration curve. The results for this test are shown in Table IV.B-6. The RSD (CV_1) for the filters with 40 μg of quartz is 0.073, and the RSD for filters with the nominal 20 μg loading of quartz is 0.086.[517]

The associated precision values at the 95th percentile confidence level were 17% for the 40 μg quartz loadings and 19% for the 20 μg quartz loadings. OSHA's reliance on this single study at the SLTC lab to support its claim of acceptable accuracy and precision down to exposure concentrations of 25 μg/m^3 is misplaced for several reasons.

First, the March 2013 SLTC study was conducted on pure NIST standard quartz and cristobalite reference materials deposited directly on filters which were then prepared for analysis by an analyst who was aware that he was conducting a laboratory performance study with the aim of achieving the least variability and the best possible precision value. No

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516 See Cardno Comments at 3, 5-6; Comments of URS Corporation on the Analytical Methods Discussion in OSHA's Notice of Proposed Rulemaking for a Crystalline Silica Standard and in the Associated Preliminary Economic Analysis (PEA) Document, February 7, 2014 ("URS Measurability Comments") at 7-8. The URS Measurability Comments are submitted herewith as Attachment 12.

517 PEA at IV-34.

interfering materials that simulate actual samples were added, and no actual sampling was involved. Moreover, no other processes or analytical procedures were applied to any of the test samples such as those that would be used to remove interferences from sample matrices. Thus, there was no acid washing and no use of secondary or tertiary XRD angles to eliminate interferences. In addition, all of the samples for this study appear to have been analyzed together, or within a short time of each other (in March, 2013). Samples analyzed on the same day or within a few days of each other likely would be analyzed against the same calibration curve, likely would be prepared from the same stock standards as used for the calibration of the instrument, and likely would be analyzed by the same analyst. These features of the March 2013 SLTC study mean that the results of the study are not representative of the variability that can be expected in the analysis of real industrial samples in which the silica is embedded in a matrix of interfering minerals and is not analyzed as part of a lab performance study.[518] Indeed, OSHA itself seems to recognize the distinction between this single lab performance study and the additional issues that characterize the analysis of real world samples. "Special handling procedures are required during the collection, preparation, and analysis of samples to avoid or to correct for interferences that can result in either an overestimation or underestimation of the quantity of crystalline silica present on the sample filter."[519] NIOSH, as noted above, makes this point as well,

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[518] See Cardno Comments at 11-12; URS Measurability Comments at 1, 4-8.

[519] PEA at IV-25. See also *id.* at IV-29 ("Interferences from silicates and other minerals can affect the accuracy of IR results. The electromagnetic radiation absorbed by silica in the infrared wavelengths consists of broad bands. In theory, no two compounds have the same absorption bands; however, in actuality, the IR spectra of silicate minerals contain silica tetrahedra and have absorption bands that will overlap. This can be a serious limitation because 90 percent of the minerals in the Earth's crust contain silica tetrahedra that will interfere with the analyses of crystalline silica.").

emphasizing that complex procedures must be followed to reduce mineral interferences and that a high level of analyst expertise is required.[520]

Second, the March 2013 SLTC study "fails to account for several sources of analytical error, including:

- Effect of differences in particle sizes on the analysis of silica by XRD and IR methods (Bhaskar et al., 1994; Kauffer et al., 2002; Ferg et al., 2008; Stacey et al., 2009);
- Effect of potential interferences on the XRD and IR analysis methods (Eller et al., 1999; Stacey, 2007);
- Effect of inter -laboratory differences in sample preparation, calibration standards, and implementation of the XRD and IR methods (NIOSH, 1995; Eller et al., 1999; Stacey et al., 2003; Stacey, 2007; Stacey et al., 2009), and;
- Effect of intra-laboratory differences in sample preparation and analysis caused by differences between analysts and variability in analysis runs (NIOSH, 1995; Eller et al., 1999; Stacey et al., 2003)."[521]

Indeed, OSHA itself acknowledges that the SLTC study does not capture inter-laboratory variability, which is "[a]nother source of error that affects the reliability of results obtained from sampling and analytical methods."[522]

Furthermore, the results of this one special study involving just ten samples at each loading level appear to be an aberration even for the SLTC lab, as indicated by the results of

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[520] See Key-Schwartz, R. *et al.*, "Determination of Airborne Crystalline Silica," in NIOSH Manual of Analytical Methods, 4th rev. ed. Cincinnati, OH, US Dep't of HHS, Public Health Service, Centers for Disease Control and Prevention, NIOSH, DHHS (NIOSH) Publication No. 03-127, at 270.

[521] Cardno Comments at 9-10. In addition, "because only the XRD method was used, the SLTC evaluation fails to account for the analytical error associated with differences between the two methods [XRD and IR], which has been identified in the scientific literature (Bhaskar et al., 1994; Eller et al., 1999; Kauffer et al., 2002; Kauffer et al., 2005; Ferg et al., 2008)." *Id.* at 10.

[522] See PEA at IV-35.

longer term quality control tests at the SLTC and a performance study conducted in 2010.[523] Thus, while the CV_1 for quartz was 0.073 for the 40 µg silica loadings and 0.086 for the 20 µg silica loadings in the March 2013 performance study, the SLTC lab's average CV_1 for quartz analysis over a range of 50-300 µg per sample from February 2007 through July 2010 was 0.129, while at loadings of 50-60 µg per sample, it was 0.144 over a comparable period.[524] If one assumes just 5% pump flow rate variability, neither of these CV_1 values produces a result meeting the NIOSH Accuracy Criterion, which effectively requires that the CV_T for exposure sampling and analysis combined must be no greater than 12.8%.[525] OSHA's formula for calculating precision at the 95% confidence level is as follows:[526]

$$Precision = 1.96 \times \sqrt{CV_1^2 + CV_2^2}$$

Applying that formula to the average CV_1 of 0.144 that the SLTC lab reported for silica loadings in the range of 50-60 µg per sample and using a conservative CV_2 of 0.05, the precision of the SLTC lab is found to be 30%, which surely is not acceptable. Yet the variability reflected here clearly is significantly lower than what would be expected in the analysis of real world samples containing interferences, because these CV_1 values are all based on analysis of quality control samples consisting solely of NIST-certified quartz standards. Moreover, the sampling range is above the range that is relevant at a PEL of 50

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523 See URS Measurability Comments at 8-10.

524 See PEA at IV-34.

525 See Leidel, N.A. *et al.*, Occupational Exposure Sampling Strategy Manual. DHEW (NIOSH) Publication No. 77-173 (1977) (Docket Item # OSHA-2010-0034-1490) at 78. CV_T is calculated as the square root of the sum of the squares of the CV for sampling (generally assumed by OSHA to be 0.05 or 5%) plus the CV for the analytical method. See *id.* at 81.

526 See PEA at IV-35.

$\mu g/m^3$ and an action level of 25 $\mu g/m^3$, where the associated silica mass values are in the range of 20-40 μg.

Even higher CV_1 (or RSD) values were obtained in a performance study conducted by the SLTC lab in 2010. In that study, the RSD values for the 40 μg silica loadings ranged from 0.128 to 0.162 on one instrument and from 0.134 to 0.226 on the other.[527] For the 20 μg loadings, the RSD values ranged from 0.161 to 0.174 on one instrument and from 0.216 to 0.287 on the other.[528] At the 40 μg loading, using a CV_2 value of 5% with the RSD for the primary analytical line of the Rigaku XRD system yields a precision value of 27%; the comparable precision value at the 20 μg loading level is 33%.[529] Again, this was a laboratory performance study using NIST-certified standard material without any interferences, so the results should reflect better precision than what can be expected when real world industrial samples are being analyzed. Even so, the precision reflected in this study clearly is not acceptable.

But the precision is even worse than this – because, as pointed out in the Cardno ChemRisk Comments, the 5% value that OSHA has used "to account for variability in sampling pump flow rates accounts for only a portion of the potential sampling error. Sampling error can occur from multiple sources other than just pump flow rate variability, including:

- Variability in the performance of different cyclones (Gautam and Sreenath, 1997; Gorner et al., 2001; Verpaele and Jouret, 2012);

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527 See Silica Precision Data attached to a cover note from Warren Hendricks to Bill Perry, Docket item # OSHA-2010-0034-1670.

528 See *id.*

529 See Cardno Comments at 9, Table 1.

- Performance of the cyclone with different dust particle sizes for a single dust species, with different dust species, and with a real world multispecies environment (Gautam and Sreenath, 1997; Vincent, 2007; Kulkarni et al., 2001; Verpaele and Jouret, 2012);

- Effect of loading/cleaning on cyclone performance (Lodge, 1988; Vincent, 2007), and;

- Effect of the electrostatic properties of dust (Lodge, 1988; Vincent, 2007)."[530]

As explained by Cardno ChemRisk, the combined sampling variability (CV_2) attributable to the various sampling factors can be estimated using the following equation, which accounts for pump flow rate variability, intersampler variability, and sampler type variability:[531]

$$CV_2 = \sqrt{(5\%)^2 + (6\%)^2 + (5\%)^2} = \sqrt{25 + 36 + 25} = 9.3\%$$

When this more realistic value of 9.3% (0.093) is used for CV_2, the precision values of all the studies referenced by OSHA increase even further[532] – making it even more obvious that sampling and analytical precision at exposure levels of 50 μg/m^3 and below is unacceptable and that measurements of worker exposures at those levels will be unreliable.

The foregoing points all relate to variability within a single lab, analyzing a known loading of pure silica reference standard material without any interfering minerals. In this sense, the precision results represent the absolute best case of intra-laboratory variability. Even so, the precision, as discussed above, is not acceptable; yet it represents only part of the variability that is expected when – as in the real world – samples are analyzed by different

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530 Cardno Comments at 7.

531 See *id.* at 8.

532 See *id.* at 8-9 & Table 1.

laboratories. To quote OSHA: "Another source of error that affects the reliability of results obtained from sampling and analytical methods is inter-laboratory variability, which describes the extent to which laboratories would obtain disparate results from analyzing the same sample."[533] So, even OSHA acknowledges that the results of sampling at its SLTC lab do not reflect the full extent of variability in the sampling and analysis of silica. To get a more complete picture, one must look elsewhere – and, as OSHA points out: "The best available source of data for characterizing total variability (which includes an interlaboratory variability component) of crystalline silica analytical methods is the American Industrial Hygiene Association (AIHA) PAT program."[534]

Results from earlier rounds in the PAT program were discussed above in the context of the Wroblewski Report, which showed that precision in these earlier rounds was unacceptable for measuring RCS exposures reliably even at concentrations well in excess of the proposed PEL. In its analysis of more recent PAT rounds 156-165, encompassing the time period from April 2004 to June 2006, OSHA found that the pooled RSD for participating laboratories was 19.5% – and even that value is something of an understatement because for most of the period, AIHA artificially limited the maximum individual laboratory RSD values to 20%.[535] Even so, using OSHA's formula $Precision = 1.96 \times \sqrt{CV_1^2 + CV_2^2}$ and assuming just 5% sampling variability for CV_2, precision at the 95% confidence level based on the RSD of 19.5% is 39.5%. When precision is that poor, measurements of silica exposure will not be reliable. And, of course, the silica filter loadings in these PAT rounds

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533 PEA at IV-35. See also Cardno Comments at 11.

534 PEA at IV-35.

535 *Id.* at IV-37.

were far higher than the silica loadings that would have to be analyzed for exposures of 50 $\mu g/m^3$ and below. Recently, Cardno ChemRisk analyzed the results for silica sample loadings in the range of 40-70 μg in PAT rounds 156-180 and found that the precision values ranged from 37% to 40%, depending on whether CV_2 was assumed to be 5% or 9.3%.[536] So there has been relatively little improvement in these more recent rounds. And in all PAT rounds, the laboratories are aware that they are participating in a certification performance test, so every precaution will be taken to assure that the results reported are the very best that the laboratory is capable of achieving.

OSHA claims that the results for PAT rounds 156-165 show that precision was as good at the lower range of filter loadings (49-70 μg) as at the higher range.[537] In fact, however, the data show no such thing. OSHA made a computational error. While over the full range of filter loadings, 80% of the labs reported results within ±25% of the applicable reference value, the breakdown between results for higher and lower silica loadings was not what OSHA claims. What the results actually show is that 83% of the labs reported results within ±25% of the reference value when silica filter loadings were >70 μg, while only 73% of the labs reported results within ±25% of the reference value when silica filter loadings were <70 μg.[538] This finding is perfectly consistent with the finding of NIOSH researchers (related in the Wroblewski Report discussed above) that the estimates of intra- and inter-laboratory variability in earlier PAT rounds tended to rise at low sample loadings, with the range of 60-80 μg silica per sample being a significant cut-point. See page 203, *supra*. (As

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536 See Cardno Comments at 9, Table 1, 13.

537 See PEA at IV-38, IV-39 & Table IV.B-8, IV-43 to IV-44.

538 See URS Measurability Comments at 10-13; Cardno Comments at 16.

an aside, OSHA has not explained why it apparently considers RCS measurements to be reliable when only 80% of the reported results are within ±25% of the true value. After all, the NIOSH Accuracy Criterion "requires that, over a specified concentration range, the method provide a result that differs no more than ±25% from the true value 95 times out of 100,"[539] and the relevant concentration range for this purpose generally is "a range of concentrations bracketing the permissible exposure limit (PEL)."[540] While the NIOSH Accuracy Criterion may not apply directly to PAT program results, it certainly suggests that the 80% "success rate" cited by OSHA for measuring concentrations well above the proposed PEL is hardly reassuring.)

A particularly revealing aspect of the PAT program analysis is the data relating to OSHA's own SLTC laboratory in PAT rounds 160-180, covering a period from June 2005 through February 2010. Over these rounds, where the silica filter loadings ranged from 55 to 165 μg, the SLTC lab's RSD was 19 percent (which was almost precisely the same as the 19.5% pooled RSD for participating laboratories in PAT rounds 156-165), and just 81% of the SLTC's reported results were within ±25% of the reference mean.[541] Moreover, these results were obtained on samples that range from 38% higher to 200% higher than the silica mass that would be collected at the proposed PEL. Assuming sampling variability of just 5% and applying OSHA's formula $Precision = 1.96 \times \sqrt{CV_1^2 + CV_2^2}$, the precision of the SLTC

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539 NIOSH Manual of Analytical Methods (January 15, 1998) at 36.

540 See Key-Schwartz, R. *et al.*, "Determination of Airborne Crystalline Silica," in NIOSH Manual of Analytical Methods, 4th rev. ed. Cincinnati, OH, US Dep't of HHS, Public Health Service, Centers for Disease Control and Prevention, NIOSH, DHHS (NIOSH) Publication No. 03-127, at 273.

541 See PEA at IV-40.

lab at the 95% confidence level in these PAT rounds is 39%, which clearly is not acceptable and which comes nowhere near meeting the NIOSH Accuracy Criterion.

As OSHA frankly admits: "The overall RSD of 19 percent for this set of samples is substantially greater than the CV_1 of 10.6 percent cited in OSHA Method ID-142 (revised December 1996), and it is higher than the various CV_1s that were obtained from the analysis of quality control samples analyzed at SLTC."[542] According to OSHA, this is no surprise: "Based on OSHA's experience, estimates of the RSD from the PAT data are consistently higher than the precision that is achievable by individual laboratories."[543] Except that the SLTC *is* an "individual laboratory" – so that cannot explain why the SLTC's RSD in the PAT program is so much worse than the RSDs "obtained from the analysis of quality control samples analyzed at SLTC." After all, inter-laboratory variability was not involved in either situation. Rather, the principal difference between OSHA's in-house quality control studies and its PAT program efforts is that the former involve analysis of pure silica reference standard material with no interfering minerals, while PAT program filters are made up to simulate real-world samples in which the silica is contained in a matrix that might be produced by industries that would likely need to be monitored for silica exposure.[544] As explained by URS, "the four matrices currently in use are coal dust (mining industry), calcite (present in concrete), talc dust (a soft, clay-like mineral that could roughly simulate the non-silica portion of bricks, tiles, or many other construction or industrial materials), and lastly a

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542 *Id.* at IV-41.

543 *Id.*

544 See URS Measurability Comments at 10, 13-15; Cardno Comments at 12.

mixture of coal dust and talc."[545] It undoubtedly is the presence of these interfering matrix materials that causes the SLTC's RSD values to deteriorate so substantially in the PAT program compared to the RSD that the SLTC lab reports for quality control samples.[546] And it is the PAT program results that should be the focus of attention in determining whether silica exposures at levels of 50 $\mu g/m^3$ and below can be reliably measured with acceptable accuracy and precision in real-world samples.

In an apparent self-contradiction, OSHA contends that PAT program results are not appropriate for this purpose, while at the same time, it acknowledges that "[t]he best available source of data for characterizing total variability (which includes an inter-laboratory variability component) of crystalline silica analytical methods is the American Industrial Hygiene Association (AIHA) PAT program."[547] The PAT program results are, indeed, the "best available source of data" to characterize variability in the analysis of RCS samples that simulate silica-containing respirable dust to which workers are exposed in the real world. As shown by Cardno ChemRisk and URS, OSHA's self-contradictory arguments to the contrary do not withstand analysis.[548]

Further evidence of the high variability and poor precision of RCS analyses when silica filter loadings are at the levels resulting from 8-hour sampling at the proposed PEL and action level is provided by the results of a recent commercial laboratory performance study sponsored by the ACC Crystalline Silica Panel. The study was designed to assess the

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545 URS Measurability Comments at 14; Cardno Comments at 12.

546 See URS Measurability Comments at 10.

547 PEA at IV-35.

548 See Cardno Comments at 15-16; URS Measurability Comments at 13-15.

accuracy, precision, and reliability of analytical results that might be expected from AIHA-accredited commercial laboratories analyzing filters with respirable crystalline silica (RCS) dust loadings corresponding to RCS exposure concentrations of 100, 50 and 25 $\mu g/m^3$ collected at a sampling rate of 1.7 liters/minute over an 8-hour work shift. In this totally blinded performance study, filters containing three different levels of respirable quartz dust loadings were sent over a period of several months to five different AIHA-accredited commercial laboratories for analysis. The labs were not informed that they were participating in a performance testing study; instead, they were sent filters that appeared to have been collected during ordinary workplace monitoring of crystalline silica exposures by commercial customers.[549]

The study included three replicate rounds of testing. For each round, "reference levels" of 20, 40 and 80 μg of respirable quartz dust (corresponding to 8-hour exposures of 25, 50 and 100 $\mu g/m^3$, respectively) were deposited onto new polyvinyl chloride (PVC) filters by the RJ Lee Group. Some of the filters contained quartz only, while others contained quartz mixed with kaolin or soda-feldspar. A more complete description of the study's design and implementation is contained in Attachment 13 hereto (Letter of May 1, 2012 from Drew R. Van Orden of RJ Lee Group to Jackson Morrill of the American Chemistry Council). The results of the performance study were analyzed statistically by Dr. Cox whose report entitled "Statistical Assessment of Performance Tests for the Analysis of Respirable Crystalline Silica (Quartz) by Commercial Laboratories Using XRD" ("Cox Performance Test Report") is submitted herewith as Attachment 14.

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549 See Cardno Comments at 14.

Dr. Cox found that, even when non-detect values were excluded, none of the mean reported values for the three reference levels of RCS came within 30% of the applicable reference value, "indicating that the accuracy of the analyses was problematic even when the non-detects were excluded."[550] The exclusion of the non-detects was not insignificant – because for 34% of the filters with loadings of 20 or more micrograms of silica, *i.e.*, 36 out of 105 non-blank filters in the test program, the laboratories reported non-detected levels of silica. Had these non-detects been included, Dr. Cox points out, "the mean reported values would have fallen further below the respective reference levels, making the accuracy of the results even more problematic."[551]

Dr. Cox also found the following

> [T]he silica mass reported by the labs does not sharply discriminate among different reference levels of actual silica loadings. For example, a reported silica mass in the highest quartile (all of which would be from the 80 μg reference level set if there were perfect agreement between higher reference levels and higher reported silica mass) has about a 50% (9/18) chance of coming from the 40 or 0 μg reference level sets.[552]

His further analysis showed "that under the conditions of this testing protocol, the laboratory results were not sufficiently accurate to reliably distinguish between concentrations that differ by a factor of 2 (i.e., 80 μg v. 40 μg)."[553]

Inter-laboratory variability also was quite significant. Some of the labs reported higher RCS loadings than other labs at every reference level. To quote Dr. Cox: "A filter

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550 Cox Performance Test Report at 5.

551 *Id.*

552 *Id.* at 8.

553 *Id.*

with a given load of silica particles could easily yield a reported silica mass that varied by a factor of approximately two, depending on which labs provided the analyses."[554] In addition, Dr. Cox noted:

> [W]ithin individual laboratories, there was substantial overlap among the 95% confidence intervals around mean reported silica mass values for different reference levels. Thus, variability of reported results within individual laboratories was such that reference levels of 20, 40, and 80 μg could not be distinguished reliably from each other.[555]

This reflects the fact that the intra-laboratory coefficients of variation (or relative standard deviation values) for replicate analyses of filters having the same reference level loadings of RCS were quite high. The relative standard deviations for all but one of the labs ranged from 20% to 66% at the various reference levels, suggesting that intra-laboratory precision in these analyses of silica dust was poor.[556] Cardno ChemRisk performed a subsequent analysis of the data – calculating the CV_1, SAE, and precision values for silica loadings at the 20 μg and 40 μg levels – and reported the following: "The estimate of CV_1 at a loading of 20 μg was 37%, and the SAE and precision values ranged from 61% – 62% and from 72% – 74%, respectively, depending on whether a value of 5% or 9.3% was used for CV_2. At a loading of 40 μg, the estimate of CV_1 was 32%, and the SAE and precision values ranged from 53% – 54% and from 63% – 65%, respectively, depending on the value assumed for CV_2."[557] Obviously, even when the lower CV_2 value is used, the resulting precision values of 63% for the 40 μg silica loading and 72% for the 20 μg silica loading indicate that

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554 *Id.* at 11.

555 *Id.*

556 See *id.* at 12-13.

557 Cardno Comments at 15.

RCS measurements at exposure concentrations of 50 μg/m^3 and 25 μg/m^3 are simply not reliable.

As noted above, OSHA typically requires that employers use a method of monitoring and analysis that has an accuracy of plus or minus 25 percent (+/- 25%) with a confidence level of 95 percent for measurements at airborne concentrations at or above the PEL – which, in turn, means that the total coefficient of variation (CV_T) or relative standard deviation (RSD) for exposure sampling and analysis combined must be no greater than 12.8%.[558] Yet in this study, accuracy was outside the +/- 25% range even when viewed simply as mean reported results, and intra-laboratory RSDs were much greater than 12.8%.

Dr. Cox concluded his report with the following observation:

> Inter-laboratory variability in this performance test program was so high that the reported results could not be used to reliably discriminate among filters prepared to reflect 8-hour exposures to respirable quartz concentrations of 25, 50 and 100 μg/m^3. Moreover, even within a single laboratory, there was enough variability in the reported results so that 2-fold variations in exposure concentrations could not be reliably distinguished.
>
> While the specific conditions of this blinded performance test program may limit the general applicability of these findings, the results point to significant potential shortcomings in the accuracy and precision of analytical results reported for quartz loadings in the neighborhood of 80 μg and below – and they indicate that for a PEL of 50 μg/m^3 and an action level of 25 μg/m^3, measurability problems could make determinations of compliance or non-compliance unreliable.[559]

Both the studies OSHA relies on and the laboratory performance study sponsored by the ACC Crystalline Silica Panel focus on the amount of RCS that would be collected in eight hours when exposure monitoring is performed with a sampler having a flow rate of approximately 1.7 L/minute, as is typical in North America. At page IV-43 of the PEA

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558 See pp. 199-200 & nn. 490, 494, *supra.*

559 Cox Performance Test Report at 14.

OSHA notes that the BGI GK 2.69 cyclone has a higher flow rate of 4.2 L/minute – the implication being that by processing a larger volume of air, a high flow-rate sampler will capture a larger amount of silica than a Dorr-Oliver sampler in the same period of time and that, as a result, greater accuracy and precision of measurements will be possible. However, as Cardno ChemRisk and URS explain, there are several reasons why one cannot assume that the use of high volume samplers will result in adequate precision at RCS exposure levels of 50 $\mu g/m^3$.

- First, as Cardno ChemRisk points out, "the accuracy and precision of the high flow rate samplers for measuring respirable crystalline silica have not been evaluated."[560] While there have been studies of "the sampling efficiencies of these samplers relative to the ISO/CEN particle size convention" and comparisons of mass collection volumes of high flow and low flow rate samplers, "[n]one of these studies evaluated the accuracy and precision of the [high flow rate] samplers using the methods recommended in NIOSH (1995) for sampling method development."[561]

- Second, "studies by Lee et al. (2010; 2012) indicate that high flow rate samplers tend to collect a higher proportion of larger size particles than the lower flow rate samplers currently used."[562] Since they display a higher sampling efficiency for particles at the 10 µm boundary of the respirable range, high flow rate samplers like the BGI GK 2.69 cyclone "tended to have a substantial bias towards collecting more respirable particulates than the low flow samplers, collecting between 12% to 31% more mass than the low flow samplers" in the Lee *et al.* (2010) study.[563] In a follow-up study, Lee *et al.* (2012) again "found that the high flow samplers tended to collect a greater mass of respirable particles, between 2.3% to 18.7% more compared to the lower flow rate 10 mm Dorr Oliver sampler."[564] Importantly, "[w]hile the high flow samplers collected more quartz mass than the low flow samplers, the standard deviations associated with

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560 Cardno Comments at 16.

561 *Id.* at 18.

562 *Id.*

563 *Id.*

564 *Id.* at 18-19.

the mass ratios and net mass ratios were high, indicating a potential increase in sampling and analysis error (Lee et al., 2012)."[565]

- Third, while RCS filter loadings will increase when a high-volume sampler is used, "so will loadings of potential interferences, with the result that detection limits for RCS may remain unchanged and precision will not improve. Because there will be a larger mass of interferences, additional sample handling procedures such as acid washing will be required, resulting in reduced precision. The samples also may require analysis using alternative secondary or tertiary peaks, or the overall X-ray intensity may be diminished due to increased filter loading."[566] Moreover, "[b]ecause respirable silica in occupational settings tends to have a greater proportion of smaller particle sizes, while the high flow samplers tend to oversample larger size particles compared to low flow rate samplers, it seems likely that the high flow samplers will collect a greater proportion of non-silica particles that can interfere with the analysis of respirable silica using the XRD or IR methods."[567]

- Fourth, if the use of high volume cyclones were allowed in addition to the traditional Dorr-Oliver sampler, "interlaboratory precision would suffer due to the use of multiple sampling devices."[568]

Thus, use of a high flow rate sampler like the BGI GK 2.69 cyclone provides no assurance that precision in measurements of RCS exposures at levels of 50 $\mu g/m^3$ and below will be improved. At the same time, instituting the practice of sampling with high flow rate cyclones, *in lieu* of the Dorr-Oliver sampler, would give rise to a number of other complications and potentially troubling issues.

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565 *Id.* at 19.

566 See URS Measurability Comments at 3, 16.

567 Cardno Comments at 19.

568 Key-Schwartz, R. *et al.*, "Determination of Airborne Crystalline Silica," in NIOSH Manual of Analytical Methods, 4th rev. ed. Cincinnati, OH, US Dep't of HHS, Public Health Service, Centers for Disease Control and Prevention, NIOSH, DHHS (NIOSH) Publication No. 03-127, at 266. See also NIOSH Hazard Review: Health Effects of Occupational Exposure to Respirable Crystalline Silica (2002) at 12 ("Because each type of cyclone exhibits specific particle collection characteristics, the use of a single cyclone type for each application would be advisable until evidence becomes available indicating that bias among cyclone types will not increase laboratory-to-laboratory variability.").

- Use of the Dorr-Oliver sampler with a recommended flow rate of 1.7 L/min is specified in OSHA Method ID-142. NIOSH Method 7500 specifies use of a sampler with a flow rate varying from 1.7 L/min (nylon cyclone) to 2.5 L/min (aluminum cyclone) but notes that regulatory agencies currently use a 1.7 L/min flow rate with the Dorr-Oliver cyclone in the United States. So these Methods would have to undergo revision before high volume samplers could be used.

- The Dorr-Oliver sampler has been used since the 1960s. Hence, exposure data for many of the epidemiological studies on which silica risk assessments (including OSHA's) are based were collected using the Dorr-Oliver sampler (or were converted from particle count to a gravimetric basis using a Dorr-Oliver sampler).[569] So most risk assessments for silica are based directly or indirectly on Dorr-Oliver sampling and may not be applicable to measurements made with other samplers.

- If new samplers are used in the future, comparability with past measurements made with Dorr-Oliver samplers would be compromised.

- Finally, because the Dorr-Oliver sampler is (and, for many years, has been) widely used in North America, switching to other samplers would involve significant costs, training time, and verification testing.

For all these reasons, as NIOSH cautions: "At this time, silica sampling should be done with a 1.7 L/min Dorr-Oliver nylon cyclone to meet the ISO/CEN/ACGIH respirable sampling convention within the United States."[570]

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In sum, as Michael A. Pannell, Senior Industrial Hygienist in OSHA's Office of Health Enforcement recently observed, exposure assessment of crystalline silica remains a problem because "the means to collect a representative sample is difficult" and "the

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[569] See Key-Schwartz, R. *et al.*, "Determination of Airborne Crystalline Silica," in NIOSH Manual of Analytical Methods, 4th rev. ed. Cincinnati, OH, US Dep't of HHS, Public Health Service, Centers for Disease Control and Prevention, NIOSH, DHHS (NIOSH) Publication No. 03-127, at 266.

[570] *Id.*

analytical variations are wide."[571] At a PEL of 50 μg/m^3 and an action level of 25 μg/m^3, reliable measurements of worker exposure will not be possible, determinations of compliance/non-compliance will be suspect, and employers will be left to speculate as to whether various ancillary requirements of the Standard apply to their work sites. This alone makes the proposed PEL technologically infeasible. And that is particularly the case during the two-year period following the effective date of the Standard when commercial laboratories will not yet have been required to meet the quality assurance provisions of paragraph (d)(5)(ii).

Conclusion

OSHA has not shown that silica exposures associated with a PEL of 100 μg/m^3 present a significant risk of material health impairment or that reducing the PEL to 50 μg/m^3 would substantially reduce any such risk that might exist. Nor has OSHA made a supportable showing that the proposed PEL of 50 μg/m^3 would be economically feasible across the range of industry sectors to which it would apply or that RCS exposures at a level of 50 μg/m^3 and below can be reliably measured with an acceptable degree of accuracy and precision in real-world samples containing interfering matrices. For all these reasons, OSHA should not set the PEL for respirable crystalline silica at a level lower than 100 μg/m^3.

Instead, to assure that the incidence of silica-related disease continues its decades-long decline to negligible levels, OSHA should change the formulaic PELs for RCS exposure in general industry to a simple value of 100 μg/m^3 and should work with employers to

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571 Pannell, M.A., Senior Industrial Hygienist, OSHA Office of Health Enforcement, *Impediments to Developing a Viable SiO_2 Exposure Assessment Program*: Slide Presentation at the 2013 American Industrial Hygiene Conference & Exposition, May 18-23, 2013, Montreal, Canada.

improve compliance with the PEL. At the same time, all employers of silica-exposed workers should implement effective programs of reasonable and appropriate monitoring (or other exposure assessments) and medical surveillance for those employees who are potentially exposed to significant levels of crystalline silica.