OSHA'S PROPOSED BERYLLIUM RULE JUNE 27, 2017 for SHIPBUILDING AND CONSTRUCTION

AN EPIDEMIOLOGICAL / OCCUPATIONAL MEDICINE ASSESSMENT AND EVALUATION

FOR

THE ABRASIVE BLASTING MANUFACTURERS ALLIANCE (ABMA)

BY

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INTRODUCTION

On Tuesday, June 27, 2017, the U.S. Occupational Safety and Health Administration (OSHA) issued a request in the Federal Register (CFR Parts 1915 and 1926) for comments regarding their proposed rule "Occupational Exposure to Beryllium and Beryllium Compounds in Construction and Shipyard Sectors." This follows the promulgation of 29 CFR Part 1910, Occupational Exposure to Beryllium and Beryllium Compounds; Proposed Rule, Friday August 7, 2015. In the 2015 proposed rule, OSHA put forth the background analysis of the scientific literature from which the best available scientific evidence according to the agency purportedly showed a human causal association between occupational exposure to beryllium and its compounds and the development of lung cancer.

These comments offered to OSHA will focus on the human epidemiologic literature cited by the agency on which to support its conclusion of a beryllium exposure, lung cancer causal nexus in its proposed rule dated August 7, 2015, along with an assessment of more recently published studies that offer unambiguous evidence clearly demonstrating the lack of a causal association between current workplace beryllium exposure and lung cancer.

These comments to OSHA will also include evaluation of the scientific literature and the proposed inclusion of shipyards and construction regarding the potential for the development of chronic beryllium disease specifically related to trace mineral beryllium contaminant in abrasive blasting material employed in those sectors.

APPROACH

The scientific approach to develop comments to OSHA regarding their request for comments on the proposed June 27, beryllium rule relies primarily on:

- Electronic and "tree" literature searches for beryllium background, exposure studies and lung cancer, peer-reviewed epidemiological research articles, as well as published reviews of the literature and supporting evidence.

- Undertake study by study critique using various professionally-accepted, scientific criteria consisting of study type and methodology, study group size (power), disorder determination, statistical significance, dose-response, exposure determination, control groups, and confounding factor control.

- Select studies meeting criteria to aid in causal determination for lung cancer from exposure to beryllium and its compounds.

- Review and analyze the studies OSHA relied on in their August 7, 2015 proposed beryllium rule.

- Perform a weight of the evidence analysis for causal nexus inference. General causal determination is a formal scientific process, employing reliable scientific methodology to evaluate whether a chemical (Beryllium in this instance), biological, physical agent, biomechanical factor or stress can alone or in combination substantially contribute to the causation or permanent aggravation/acceleration of specific diseases or health conditions. Causal determination for a specific hazardous substance relies to the extent possible, on the results of well-constructed and conducted human studies or research, commonly referred to as epidemiology. The first step is to perform an extensive literature search for all potentially appropriate studies using both electronic and traditional, e.g., "tree" reference search approaches. Electronic searching typically includes the National Library of Medicine's MEDLINE, TOXLINE and PUBMED search engines. Epidemiological assessment requires that scientific literature is identified and studies are critiqued as to their design, exposure assessment, disorder determination and statistical treatment. For example, certain epidemiological study designs, such as case reports, case series and cross-sectional studies, do not provide a reliable scientific basis to determine and confirm causation. Additionally, other critical factors, such as employing a control group and controlling for confounding factors (other potential causes, e.g., smoking in lung cancer studies) and potential study biases must also be appropriately included and addressed. Next, a weight of the evidence analysis examining the data for all of the methodologically-sound studies should be performed using appropriate factors to evaluate whether an association is causal in nature. Such guidance factors (e.g., Hill, 1965) have been developed and used over a number of decades and are routinely employed by scientists especially epidemiologists and governmental agencies such as the National Institute For Occupational Safety and Health (NIOSH), US EPA, ACOEM, IARC, NTP, etc. These factors typically consist of:

  Strength of the association
  Size of the measured or estimated relative risk.

  Consistency of the association
  Similar findings by different persons, in different places, circumstances, and times.

  Specificity of the association
  The exposure can be demonstrated to be specifically related to the disorder and not a group of disorders or group of exposures.

  Temporality of the association
  Exposure to the suspected cause always precedes the onset of the disease or health-related characteristic.

  Biological gradient of the association
  A dose-response relationship between exposure to the suspected cause and increased risk or severity of the disease or health-related characteristic can be demonstrated.

Biological plausibility of the association
The observed association agrees with existing knowledge.

Coherence of the association
The observed association does not conflict with existing scientific knowledge.

Experimental support for the association
Experimental or quasi-experimental evidence exists or can be demonstrated to support the association.

ASSESSMENT FOR LUNG CANCER

Twenty original and re-analysis studies were identified, critiqued and evaluated following the approach as defined previously. Six evaluations of the studies examining a causal inference were also assessed. It is noted that OSHA's 2015 regulatory lung cancer best available evidence assessment was confined to 15 studies and re-analyses and to date has not updated its lung cancer risk assessment for beryllium exposure as several, important studies have been published either right before or following the issuance of the standard. OSHA stated that it relied primarily on the 2011 article by Schubauer-Berigan M.K. et al. which was a follow up study of Ward et al. 1992 for its determination that occupational beryllium exposure produces lung cancer. However, the entire relevant literature base including their article was evaluated (Boffetta et al, 2012) and shown to have significant flaws including:

- Only two of six plants studied had statistical significantly elevated standardized mortality ratios (SMRs). The lack of consistency of the effect is noted in that study.
- The follow up study by Schubauer-Berigan of the Ward 1992 study showed decreased SMRs for only the Lorian and Reading plants as compared to the Ward 1992 study.
- The authors did not update the job histories from the original study 20 years prior.
- The potentially confounding effect of smoking, the primary cause of lung cancer in humans was inadequately controlled.
- Exposure determination from other workplace carcinogens was not adequately assessed including for asbestos exposure.
- Most of the studies cited by OSHA were additional analyses of the same underlying occupational study sites.

OSHA also failed to outline any "weight of the evidence" evaluation approach for determining a beryllium-lung cancer link from its 15 cited references (See Reference List) such as that performed by NIOSH in 1997 in its epidemiological literature critique for "ergonomic-related musculoskeletal disorders." Had it done so, the following should have been noted and considered in its beryllium carcinogenicity assessment:

- There was inadequate segregation of soluble and insoluble beryllium and beryllium compound exposed populations. This was not addressed despite the findings of Boffetta et al. in 2014.

- As noted in the evaluation for evidence of a beryllium lung cancer link by Boffetta et al. in 2012, there may be some evidence supporting such a link as noted in circa 1940's studies; however, this is not relevant to shipbuilding, welding, etc.

- Studies evaluated by OSHA for its Beryllium rule did not adequately separate the pre-1955 exposures which were significantly higher than subsequent and present exposures; nor did it segregate the data to fully examine the carcinogenic potential of soluble beryllium versus insoluble beryllium exposures.

- Only two factors: age at first hire and lagged duration of employment, showed small, elevated SMRs, while the other exposure factors such as length of employment failed to reveal significantly elevated SMRs as one would expect in dose-response carcinogenic risk determination (Rothman and Mosquin, 2011; Deubner and Roth, 2015). Virtually all of the SMR findings especially on reanalysis and evaluation do not show significant strengths of association as the SMRs are mostly well below a very limited strength of association of 1.5 or less; and some were not statistically significant.

- Use of a more appropriate smoking comparison population reduces the SMR to less than 1.0, meaning no correlation of beryllium exposure to development of lung cancer.

- Apparently OSHA did not consider the overall evaluations of the scientific evidence by MacMahon in 1994, Hollins et al. in 2009 and Levy et al. 2009, that failed to find sufficient evidence to establish a beryllium lung cancer causal association in humans, or two reanalyses of the original cohort, Levy et al. 2002, a re-analysis of a NIOSH cohort mortality study and Levy et al. 2007, a reexamination of findings from a nested, case-control study that also failed to find support for a causal association.

There are two well conducted epidemiological studies published just prior to or after the 2015 OSHA Beryllium rule that provide new evidence that does not support the conclusion that soluble and insoluble beryllium compound occupational exposure produces lung cancer. The key findings include:

- Boffetta et al. 2014 evaluated 4950 insoluble beryllium compound workers at four facilities. An SMR of 96 showing no potential causal association between insoluble beryllium compound exposures in workers and the development of lung cancer.

- Boffetta et al. 2016 conducted an historical cohort mortality study of 16,115 workers who had worked in 15 facilities; eight facilities with insoluble beryllium compound exposures and seven facilities with soluble beryllium compound. Exposures post 1955 to soluble beryllium showed a non-elevated SMR of 0.88 (CI: 0.75-1.03) for insoluble

beryllium compound workers; 1.09 (CI: 0.99-1.09) for soluble beryllium compound workers and 1.02 (CI: 0.94-1.10) for the entire cohort indicating no evidence for a causal association. Importantly, there was also no trend in duration of employment reflecting the lack of a dose-response effect.

Additionally, the scientific/medical literature search and assessment failed to identify any epidemiological studies performed using appropriate scientific methodology for discerning a risk for lung cancer from trace mineral beryllium abrasive blasting exposure with possible development of lung cancer that showed a materially increased lung cancer risk in shipyard and construction workplaces.

ASSESSMENT FOR CHRONIC BERYLLIUM DISEASE FOR CONSTRUCTION AND SHIPYARD ABRASIVE BLASTING

OSHA in its proposed rulemaking for possible beryllium exposure in shipyards and construction reported that it has evidence that beryllium exposure in these sectors is limited to the following operations: abrasive blasting in construction, abrasive blasting in shipyards, and welding in shipyards. An additional scientific literature search and evaluation was performed using the same methodology outlined above for the beryllium lung cancer assessment. The following is noted:

- None of the seven (7) case-control and longitudinal epidemiological studies cited by OSHA in its August 9, 2015 health risk analysis (see references) and evaluated in this assessment along with 19 case series and cross-sectional articles (see OSHA 2015 health risk analysis) were performed using abrasive blasting or welding exposed populations in shipyards and construction.

- The independent literature search and evaluation in this assessment failed to identify any epidemiological studies correlating the development of CBD with performing abrasive blasting work in shipyards and construction sites.

- The literature analysis also failed to identify any studies performed according to appropriate methodology for identifying a causal nexus from exposure to mineral beryllium especially in those two workplace settings and the development of CBD.

- While OSHA in its proposed beryllium rule stated that it has evidence of beryllium exposure in the shipyard and construction industry sectors, none were provided for review and assessment.

- OSHA did not provide evidence of an increased risk for the development of CBD from exposure to abrasive blasting materials potentially containing trace amounts of mineral beryllium at shipyards and construction sites.

- A recent study by NIOSH investigators (Mugford et al., 2017) reported the results of bulk and personal airborne sampling at two coal slag processing facilities. Coal slag is a major component of non-silica abrasive blasting materials used in construction and shipyard activities. The researchers reported bulk samples containing beryllium below the levels of detection employed (LOD - 0.096 mg/kg) at one facility. At the other facility, the beryllium bulk sample analyses were either below the LOD or at most, 4.1 ug/kg or 4.1 parts per million. It is critical to note that the bulk sampling performed on the finished product was reported to be below the LOD at both facilities.

- Mugford et al, 2017 from NIOSH also reported coal slag processing workers airborne beryllium exposures at below the level of detection at one facility (LOD - 0.01 ug/m3) or below the LOD or 0.11 ug/m3 in the other facility.

- There are no published epidemiological studies capable of establishing causation performed according to accepted causal association methodology indicating beryllium exposure as the result of welding activities in construction and shipyard sites producing chronic beryllium disease in workers in those industries.

CONCLUSIONS

Including the more recent cohort studies along with other evaluations and re-analyses, scientific assessment of a potential causal association between occupational beryllium exposure and the development of lung cancer clearly shows a lack of consistent evidence of a significant risk for the development of lung cancer in either soluble or insoluble beryllium exposed occupational groups who worked after 1955. It is highly recommended that OSHA update its initial 2015 occupational health hazard determination incorporating other scientific studies published prior to 2015 and more recent studies. It is stressed that the 2016 by Boffetta et al. casts significant doubt on earlier studies and re-analyses due to its large cohort size and use of significantly more facilities as compared to earlier research; as well as appropriate use of reference groups by state, consistency checks of cohort information and proportional hazard Cox regression models.

There is also no scientific basis for the inclusion of the construction and shipyard industries in the OSHA Beryllium rule due to beryllium exposure from abrasive blasting and welding. As noted above, there is no epidemiological evidence that mineral beryllium workplace exposures causes CBD as a result of abrasive blasting or welding in those industries. Further, a recent NIOSH study (Mugford et al., 2017) at two coal slag processing facilities clearly documents that beryllium is only found at trace contamination levels well below amounts that could generate an exposure above the proposed OSHA Beryllium PEL of 0.2 ug/m3. The researchers findings from actual personal airborne beryllium samples show that the proposed OSHA Beryllium PEL would be well below the proposed 0.2 ug/m3 level. It is also noted that OSHA regulations for welding are not scientifically based on possible beryllium exposure resulting in adverse health effects.

Thus, there is no scientific support for inclusion of shipyards or construction sites from either a lung cancer or chronic beryllium disease risk concern. Further, medical monitoring and surveillance activities are not indicated for this group of workers including the lack of scientific support for the use of chest computerized tomography (CT) in beryllium containing working environments at the beryllium exposure levels indicated for shipyards and construction sites (Woolf et al., 2014; Moyer 2014; and de Koning et al., 2014).

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