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MICHAELS AND MONFORTON RESPOND

We appreciate the opportunity to respond to the critique by Marc Kolanz of Brush Wellman Inc. of our article on the beryllium industry's public relations efforts.¹ Kolanz's letter is an excellent example of our article's primary message—that Brush has waged a concerted campaign over many years to refute the scientific evidence of the health hazards associated with beryllium exposure. The industry's efforts have impeded the replacement of an inadequate workplace beryllium exposure standard, thus placing workers at increased risk of developing beryllium-related diseases.

For the reader who may not have our article in front of them, we presented numerous previously unpublished documents illustrating Brush's efforts to counter the observations by independent scientists that workers exposed at levels below the standard were developing chronic beryllium disease (CBD), demonstrating that the Occupational Safety and Health Administration (OSHA) standard of 2 µg/m³ was (and continues to be) inadequate. In his letter, Kolanz does not dispute the existence nor the content of any of these documents.

Kolanz disagrees with our interpretation of various data points in the historical debate over the adequacy of the 2 µg/m³ standard. This debate has been resolved.

As we note, and as Kolanz acknowledges in his letter, it is now abundantly clear that CBD can and does occur among workers exposed to beryllium at levels far below the current OSHA standard, and that it is necessary to reduce exposure levels by at least an order of magnitude to prevent CBD.

Kolanz has taken the occasion of our article to advance Brush's interpretation of other scientific questions that are at best tangential to our article—in each case promoting what we believe are erroneous assertions that minimize the hazards associated with beryllium exposure. We address these assertions in this response and elsewhere.² Because many of the documents we use in this discussion are unpublished, we have posted them on the website of the Project on Scientific Knowledge and Public Policy (SKAPP),³ so that readers can decide for themselves how these historical records should be interpreted.

Compelling evidence that beryllium is a lung carcinogen

For more than 30 years, Brush Wellman has fought the labeling of beryllium as a carcinogen, motivated by fear that this would greatly reduce its ability to sell its products to downstream manufacturers.^{2,4-7} As one industry document stated: "If beryllium is determined to be a carcinogen and so labeled and so regulated it would only be a matter of time until its usage would shrink to a point where it would no longer be a viable industry."³ We believe this is the reason that Kolanz devotes a sizable portion of his letter to an attempt to show that beryllium is not a lung carcinogen, even though our article only touches briefly on beryllium's carcinogenicity.

Kolanz claims we distort the history of OSHA's efforts during the 1970s to develop a health standard to protect workers from beryllium exposure. We stand by our statement that the rulemaking was shelved following political pressure. This has been documented in a prize-winning series of newspaper articles by journalist Sam Roe⁸ and confirmed by the Secretary of Energy.⁹ To advance his own interpretation of OSHA's aborted beryllium rulemaking, Kolanz attempts to reopen a long-forgotten dispute from the late 1970s in which one National Institute of Safety and Health (NIOSH) researcher disagreed with two others. Kolanz cites only the position of the dissenting researcher, failing to note that in response to the dispute, OSHA's Assistant Secretary Eula Bingham asked Assistant Secretary for Health Julius B. Richmond to convene a panel of senior scientists to review all the epidemiologic, clinical, and experimental data to help assess whether beryllium

posed a carcinogenic risk for exposed workers.¹⁰ The reviewers concluded that “beryllium should be considered as a suspect carcinogen for exposed workers.”¹¹

In any case, a debate over what the literature showed three decades ago is primarily of historical interest because subsequent studies, with better methods and longer follow-up, have consistently found beryllium to be a lung carcinogen. This leads to Kolanz’s second erroneous assertion: that recent studies find that beryllium should not be classified as a human carcinogen. With one exception, the articles to which Kolanz refers^{12–14} are not actually studies but reanalyses of studies originally conducted by NIOSH scientists. The original NIOSH studies found increased lung cancer risk associated with beryllium exposure.^{15–17} Each of the reanalyses was paid for by Brush, clearly for the purpose of countering the positive NIOSH studies. Some of the same “product defense” scientists who performed the reanalyses for Brush have undertaken similar efforts for tobacco, alcoholic beverage, and coal-burning power companies.²

The single actual study (i.e., not a reanalysis) cited by Kolanz as supporting his assertion is a study of lung cancer risk among plutonium-exposed workers, which examined exposure to asbestos, hexavalent chromium, and beryllium as possible confounders.¹⁸ Given that the study found no association between exposure to either asbestos or chromium (two well-known lung carcinogens) and lung cancer, it is questionable at best to suggest this study provides evidence of no association between beryllium exposure and lung cancer.

Furthermore, two of the reanalyses cited by Kolanz audaciously claim to have discovered a previously unknown, serious bias in the case-control study design,^{13,14} a standard method that is widely used in contemporary epidemiology. A subsequent reanalysis and two commentaries by NIOSH and academic epidemiologists have refuted this claim.^{19–21}

Kolanz’s assertion that “recent studies find that beryllium should not be classified as a human carcinogen” is best understood as the latest installment in decades of effort by the beryllium industry to prevent the metal being labeled a carcinogen. Over the years, Brush Wellman has advanced many arguments in an unsuccessful effort to forestall imposition of the “cancer-causing label.” These arguments have been rejected by scientists who are not associated with the beryllium industry, including expert panels convened by the International Agency for Research on Cancer (IARC) and the U.S. National Toxicology Program (NTP).^{22,23}

To grasp the extent of this campaign, readers might be interested in other failed arguments used by Brush

in the past to assert that the evidence for beryllium’s carcinogenicity is flawed. For example, for many years, Brush’s representatives claimed the numerous animal studies demonstrating carcinogenicity were faulty^{5,24} and that the animal evidence was “highly debatable.”²⁵ This line of argument was finally dropped when the animal evidence became so powerful it could no longer be disputed.^{26,27}

Similarly, when IARC and NTP examined the scientific evidence on the carcinogenicity of beryllium, the industry attempted to claim that while the excess lung cancer risk observed in one epidemiologic study was real, the increased risk was not due to exposure to beryllium, but instead from exposure to sulfuric acid mist found in just one Brush Wellman facility.^{2,28–31} Kolanz promoted this position to the NTP in his unsuccessful attempt to convince the agency that beryllium was not a human carcinogen.³² Evidently, Brush has abandoned this argument, perhaps because it is inconsistent with its current position that the increase is spurious, caused by methodological errors.

There is no evidence for a safe level of beryllium exposure

As we document, for many years Brush held that there was no evidence that exposures lower than 2 µg/m³ were associated with disease. This position has been rejected by the scientific community, most recently by a National Academy of Sciences Panel (“The epidemiologic evidence shows that the long-standing limit of 2 µg/m³ is inadequate for preventing CBD”³³). Now, Brush reports that the firm’s comprehensive “worker protection model” in which exposures are held lower than 0.2 µg/m³ results in a sensitivity rate of 1%. This represents significant progress in preventing beryllium-related disease. In our view, however, the data from Brush’s facilities are not sufficiently robust to conclude that exposure levels lower than 0.2 µg/m³ are actually safe.

This has great relevance for public health protection and regulation. Other than Brush Wellman and the Department of Energy (DOE) contractors who manufacture nuclear weapons, we believe that many employers potentially involved in machining or recycling beryllium products do not have the capability of ensuring that workplace beryllium exposures are kept lower than 0.2 µg/m³.

Furthermore, there is little evidence to support the assertion that there is a 1% “background” rate of beryllium sensitivity (BeS) in non-occupationally exposed populations. Studies conducted by independent, academic scientists have reported no BeS among

individuals with no exposure to beryllium.^{34,35} This also has public health implications: the supposed existence of a 1% background rate might be seen by employers as a reason not to institute a beryllium disease surveillance program, fearing that a surveillance program would identify and force them to address non-occupational background BeS cases. This would be a most unfortunate development.

The BeLPT is a useful screening tool

Kolanz ignores the substantial literature on the beryllium lymphocyte proliferation test (BeLPT), citing only studies paid for by Brush.^{36–38} We discuss the use of the BeLPT as a tool for estimating prevalence of disease in beryllium-exposed populations. This approach is supported by a recent review by NIOSH scientists in the *Annual Review of Public Health*:

Despite limitations in test consistency and repeatability, beryllium lymphocyte proliferation testing has been an invaluable tool in the identification of workplace risks in population studies and of intervention effectiveness.³⁹

It is noteworthy that Kolanz uses a controversial article by Borak et al.³⁷ to support his assertion that the BeLPT is not reliable. This article was the subject of published erratum notice, explaining that the authors failed to disclose the article was actually based on a legal work product commissioned by Brush's attorneys.⁴⁰

We believe that Brush is eager to discredit the use of the BeLPT because it is widely used to identify new CBD and BeS cases; without the use of this test, far fewer individuals would be diagnosed with beryllium-related conditions, and beryllium would appear to be less harmful.

Subclinical beryllium disease is still beryllium disease

Physiologic changes caused by beryllium exposure, including lung granulomas, are signs of beryllium disease, whether or not the individual is symptomatic at the time. In fact, a portion of these patients actually has measurable physiologic abnormalities.⁴¹ Furthermore, many whose asymptomatic CBD is discovered through screening go on to develop fully recognizable symptomatic disease.⁴²

It is not true, as Kolanz asserts, that “older studies (pre-1989) refer solely to the identification of people with clinically evident disease (clinical/symptomatic CBD).” The existence of asymptomatic beryllium disease was known at least as far back as the 1950s. In one 1955 article, for example, Harriet Hardy describes a 33-year-old woman with beryllium-caused x-ray changes but no symptoms or lung function abnormalities:

The worker has no complaints. She leads her usual life, hindered only by anxiety, because she knows of the illness and subsequent death of several young women with whom she worked at jobs similar to her own. This represents a beryllium effect not productive of symptoms, because the process does not involve enough tissue to embarrass function.⁴³

Strangely, Kolanz asserts, “Material impairment of health, such as clinical CBD, is the appropriate regulatory basis to use when setting an exposure limit for beryllium.” This posits that asymptomatic physiologic damage is not important or relevant to the individual or to the policy maker. This theory is not supported by either common sense or OSHA's traditional approach to controlling chemical hazards (see, for example, OSHA's exposure standards protecting workers exposed to cadmium, formaldehyde, and lead).^{44–46}

There is no evidence that beryllium can be used safely in downstream facilities

Kolanz's report on the alleged mishandling of beryllium waste products by the Noranda Corporation underscores our contention that there is affirmative evidence that beryllium cannot be used safely in downstream facilities. While Brush may have instituted an adequate protective program in its own factories, it cannot control exposures in downstream facilities where beryllium is used in manufacturing processes or is eventually recycled. According to Kolanz, the managers of the Noranda facility were aware that beryllium was a constituent of the material being recycled, yet they still did not ensure it was handled properly. Thirty-two workers developed CBD. If this occurred at a multinational-owned facility with knowledgeable managers, it seems even less likely that workers will be protected at small facilities processing uncategorized metals or electronic products. As a society, we must consider if it is necessary to risk the health of workers so that beryllium can be used as a component of disk drives, golf clubs, fishing rods, pen clips, camera shutters, and other consumer goods.³⁹

In addition, Kolanz has made several errors that should be corrected. We certainly disagree with his interpretation of the history of the “toxic standard,” a label affixed to it by Merrill Eisenbud himself in his autobiography, in which he explains that it was so labeled “in recognition of the seemingly flimsy basis on which it was established.”⁴⁷

In its rulemaking process, DOE concluded that the current OSHA standard was inadequate, but recognized “it is difficult to determine from this scientific evidence the exposure level necessary to eliminate the risk of contracting CBD.”⁴⁸ DOE saw the necessity of moving

quickly to protect workers with an action level of 0.2 $\mu\text{g}/\text{m}^3$, while deferring to what was expected to be a full OSHA rulemaking that would conduct a more thorough review of the scientific literature. As we note in our article, the promise of prompt OSHA rulemaking has been unfulfilled.

Kolanz unequivocally asserts that Brush “did not hire Hill and Knowlton (H&K) nor implement their proposal.” The evidence we have for our reporting of Brush’s relationship with H&K is an invoice sent by the public relations firm to Brush (with accompanying note),⁴⁹ the H&K public relations program proposal,⁵⁰ an internal Brush memo talking about materials needed for the H&K initiative,⁵¹ a letter from Brush to H&K providing “supporting information for the PR program,”⁵² a series of letters developed by H&K for Brush to send to its customers reassuring them of the safety of beryllium,⁵³ and copies of letters sent by Brush Wellman that include much of the text provided by H&K (with copies sent to H&K).^{54,55}

Finally, Kolanz asserts that we have attempted to “manufacture certainty” because one of us (DM) served as an expert witness in one suit against Brush Wellman. As DOE noted in its rulemaking, protecting the health of workers exposed to beryllium does not require absolute certainty,⁴⁸ and we did not attempt to manufacture it. It is a basic public health principle that protections are implemented using the best evidence available at the time. Certainty is never required, and we do not claim that certainty was present decades ago. What we show is that independent scientists raised questions about the adequacy of the standard, and as more studies were done, it became clear these scientists were correct—workers were developing CBD at exposure levels permitted under the current standard. We document how Brush defended the inadequate standard, to the detriment of public health.

It is incorrect to suggest that the needs of litigation have somehow influenced our reporting. Lawsuits against Brush Wellman have dramatically decreased because the U.S. government has stepped in and provided compensation payments to most workers with beryllium disease, under the condition that they drop any lawsuits against the government or the beryllium industry. One of us (DM) was instrumental in developing this program, which has provided more than \$100 million in compensation payments to workers with CBD or BeS.² The program has undoubtedly saved the beryllium industry a large amount of money and has eliminated much litigation brought by sick workers against beryllium producers.

We clearly stated that Brush Wellman has collaborated with NIOSH in a useful research initiative, and we

recognize that Brush’s current efforts to protect its own employees appear to be exemplary. But we also present compelling evidence that Brush has waged a public relations campaign to defend an inadequate standard, impeding government efforts to strengthen workplace protections badly needed to protect beryllium-exposed workers from serious lung disease.

Kolanz’s letter is evidence that this unfortunate campaign continues. We hope that Brush has a change of heart and joins us in asking OSHA to move quickly to issue a truly protective workplace beryllium exposure standard.

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