

# Be Aware of Beryllium

*Understanding the risks to protect employees*

*By William R. Pioli*

**B**ERYLLIUM AND BERYLLIUM COMPOUNDS represent arguably the most toxic, nonradioactive chemical substances to which workers in general industry will ever be exposed [EPA(c); Newman(a); Rossman(b); Kreiss, et al 16+; ACGIH]. A single exposure to beryllium above one's sensitivity threshold may cause chronic beryllium disease (CBD), a progressive, irreversible, incurable, potentially fatal lung disease.

This little-known fact initially surprises even experienced SH&E professionals. Once it is understood that beryllium toxicity is not exclusively dose-response-related, SH&E professionals can more accurately assess current exposure hazards, evaluate past exposure risk and implement specific medical surveillance. This increased awareness can also help practitioners reduce the risk of CBD, thereby better protecting worker health and welfare.

## **Beryllium Chemistry & Metallurgy**

The element beryllium was discovered in 1798 and chemically isolated in 1828. Beryllium, atomic number 4, can be found in Group 2, alkaline earth metals, on the periodic table. The reactions, electronic configuration and physical properties of beryllium are all as expected for a Group 2 metal (Lide). Chemically, beryllium behaves similarly to other Group 2 elements; however, the other nonradioactive members of Group 2 (magnesium, calcium, strontium, barium) generally form compounds of low toxicity compared to those formed by beryllium. Table 1 presents a compendium of beryllium compounds [EPA(c); Lide].

Beryllium metal has several unique properties. It is the lightest of all solid and chemically stable substances with an unusually high melting point (1278°C) and low density, and high specific heat, heat of fusion, sound conductance and strength-to-weight ratio. Beryllium imparts several excellent properties to alloys even at quite low quantities (<5 percent) such as decreasing density-to-strength ratio, elevating the

melting point, increasing resistance to oxidation and the modulus of elasticity [EPA(c)]. Although high-percentage beryllium alloys are often too brittle for industrial application, they are used in the aerospace industry. Beryllium alloys can be found in ceramic applications, dental amalgam, electrical contacts and sporting equipment. They also are used in atomic energy applications as a heat shield, as nuclear reactor parts and as an excellent neutron window with which to control nuclear reactions. Thus, beryllium is highly important to the U.S. Dept. of Energy (DOE) and in other nuclear applications. Unfortunately, due to disease incidence among its employees and contractors, DOE has become the best source of beryllium data related to human exposure outcomes and statistical analysis (Viet, et al 245; Barnard, et al 804).

## **Beryllium Biology & Genetics**

Biologically, in sensitive individuals, beryllium incites a T-cell mediated immune system reaction that medical professionals do not completely understand [Newman(b) 197]. Apparently, one's body recognizes beryllium as foreign matter and develops a unique cellular memory in lymphocytes that only recognizes beryllium. When the memory cells detect the element, the lymphocytes vigorously attack it in an uncontrolled manner. Unfortunately, this progressive, immune-system attack causes severe lung damage leading to CBD in a published data range of 50 to 85 percent of sensitized individuals [Newman, et al; Rossman(b); Newman(a); DOE(c); Kreiss, et al].

Several scientists have concluded that a genetic link exists between contracting beryllium sensitization (SENS) and CBD [Newman(b) 197; Richeldi, et al(b) 337+; Wang, et al; Lombardi, et al]. The prime genetic marker is HLA-DPB1 Glutamate 69 (Glu69).

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Studies have shown that up to 97 percent of individuals with CBD have this marker while 30 to 45 percent of exposed workers have it (90 percent and 40 percent, respectively, are assumed in this article for calculation purposes). However, merely having Glu69 does not automatically ensure SENS or CBD after exposure to beryllium, nor does its absence indicate disease immunity [Newman(b) 198; Wang, et al]. Development of the disease appears to be much more complicated. Since one to six percent of exposed workers contract CBD and one to 16 percent contract SENS, the presence of Glu69 does not appear to be the only genetic factor in developing CBD after SENS (Wang, et al).

With no genetic screening, an exposed population of 100 workers can be expected to yield three CBD cases—a risk of approximately 1 in 30 (Figure 1). Using genetic screening, 90 percent or 2.7 of those three cases could theoretically be screened out and eliminated by not allowing these workers to be exposed.

This would leave 10 percent or less than one (0.3 CBD cases) of the anticipated three cases of CBD that would still occur. The risk then becomes 1 in 300—0.3 CBD cases/100 exposed population (Figure 2).

Suppose a company needs to employ 100 workers and that population will be exposed to airborne beryllium. It may be unrealistic to employ a reduction-in-force strategy that would screen out 40 percent of the workforce. To successfully employ genetic screening in this scenario, the sampled population should be 167 individuals, from which one can expect 67 (40 percent) to be Glu69(+) and subsequently screened out. An exposed population of 167 would have been expected to yield five CBD cases (3 percent x 167 = 5); however, by employing genetic screening, 90 percent of those cases can be anticipated to be prevented (90 percent x 5 = 4.5), leaving an occurrence rate of less than one case (0.5 CBD case/167 population ~ 1/333). This is the same principle depicted in Figure 3; it simply starts with 167 individuals instead of 100.

When one can afford to screen out 40 percent of the exposed population, two significant goals will be accomplished: 1) minimize the number of workers exposed to beryllium and; 2) reduce the anticipated incidence of SENS/CBD.

At this time, medical professionals are encouraging better industrial hygiene and improved workplace practices rather than genetic screening. SH&E professionals should note that genetic screening will help to identify those most “at-risk” as a proactive risk-reduction technique. However, before conducting genetic screening, one should consult an attorney to ensure that the program is managed legally.

**Table 1**

## Common Beryllium Compounds & Alloys

Material	CAS* Number	Formula
Beryllium metal	7440-41-7	Be
Beryllium oxide	1304-56-9	BeO
Beryllium hydroxide	13327-32-7	Be(OH) <sub>2</sub>
Beryllium chloride	7787-47-5	BeCl <sub>2</sub>
Beryllium fluoride	7787-49-7	BeF <sub>2</sub>
Beryl	1302-52-9	Be <sub>3</sub> Al <sub>2</sub> (SiO <sub>3</sub> ) <sub>6</sub>
Bertrandite	12161-82-9	Be <sub>4</sub> Si <sub>2</sub> O <sub>7</sub> (OH) <sub>2</sub>
Aluminum alloy (Al-Be)	12770-50-2	Various
Copper alloy (Cu-Be)	11133-98-5	Various
Nickel alloy (Ni-Be)	37227-61-5	Various

Source: EPA(c); Lide  
\*Chemical Abstract Service

**Table 2**

## Diseases Associated with Occupational Exposure to Beryllium

Acute	Chronic
Acute pneumonitis	Beryllium sensitization (SENS)
Nasopyaryngitis	Chronic beryllium disease (CBD)
Tracheitis	Subcutaneous, liver granulomas
Bronchitis	Cancer (lung, bone)
Dermatitis	
Conjunctivitis	
Upper and lower respiratory tract abnormalities	

### Beryllium Toxicity

Table 2 lists diseases associated with exposure to beryllium. Researchers generally agree that beryllium metal and all forms of beryllium (excluding mineral forms) can cause disease and should, therefore, be considered significant health risks (Sprince 385; Stokinger). Acute beryllium disease usually has a quick onset and resembles pneumonia or bronchitis (NJMRC). Thanks to recognition that high levels of exposure to beryllium dust and fume cause problems, occurrence of this disease is rare.

The initial sign of an adverse reaction to beryllium exposure is anticipated to be SENS. It precedes CBD and still occurs in exposed industrial populations at a significant rate—about 1 in 10—with CBD

occurring at about 1 in 30 in exposed populations. Granulomas most often occur in the lung and to a lesser extent in the skin and liver; however, due to the autoimmune nature of this disease, granulomas can occur anywhere in the body.

OSHA, American Conference of Governmental Industrial Hygienists (ACGIH) and NIOSH have all established occupational exposure levels for beryllium. While OSHA's permissible exposure limit (PEL) is the legal limit in the U.S., ACGIH and NIOSH have both recently updated their recommendations [threshold limit value (TLV) and recommended exposure limit (REL), respectively] to reflect current thought. Table 3 lists these groups' exposure levels.

OSHA and ACGIH govern beryllium exposure by means of a time-weighted average (TWA). According to ACGIH, TLV-TWA is "the time-weighted average concentration for a conventional eight-hour workday and a 40-hour workweek, to which it is believed that nearly all workers may be repeatedly exposed, day after day, without adverse effect." It currently has a TWA of 2.0  $\mu\text{g}/\text{m}^3$  and a short-term exposure level (STEL) of 10  $\mu\text{g}/\text{m}^3$ . The STEL limits worker excursion exposure to 10  $\mu\text{g}/\text{m}^3$  for 15 minutes four times per eight-hour shift in which the TWA is met.

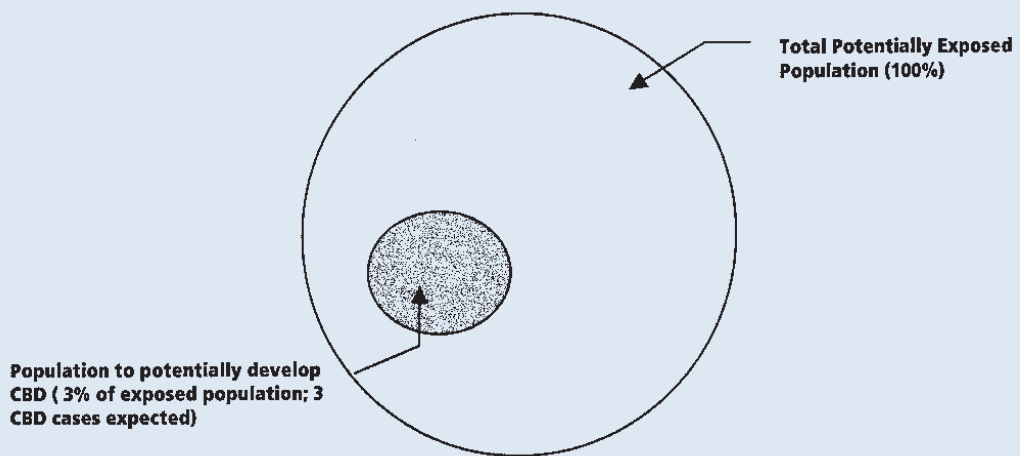
This means that ACGIH believes workers can be safely exposed to beryllium at a peak exposure of 10  $\mu\text{g}/\text{m}^3$ —which is 1,000 times the National Emission Standard for Hazardous Air Pollutants (NESHAP) of 0.01  $\mu\text{g}/\text{m}^3$ . In its "Notice of Intended Changes" for 2001, ACGIH proposed a new eight-hour TWA of 0.2  $\mu\text{g}/\text{m}^3$ , dropped the STEL, imposed a particle size selective criterion and added a sensitizer notation; under these changes, a worker could be exposed to peak 0.6  $\mu\text{g}/\text{m}^3$  exposure during an eight-hour workshift that meets the 0.2  $\mu\text{g}/\text{m}^3$  TWA; 0.6  $\mu\text{g}/\text{m}^3$  is 600 times the NESHAP.

These proposed changes indicate that the group believes particle size is impor-

tant, but not peak exposure. Medical advice on the matter does not quantify exposure levels or dose. A comment such as "even small amounts of exposure to beryllium can cause disease in some people" (NJMRC) in conjunction with discussions related to disease initiation considering exposure above one's personal threshold [Rossmann(b)] lend credence to arguments that peak exposure may more important in SENS/CBD etiology than dose/response and

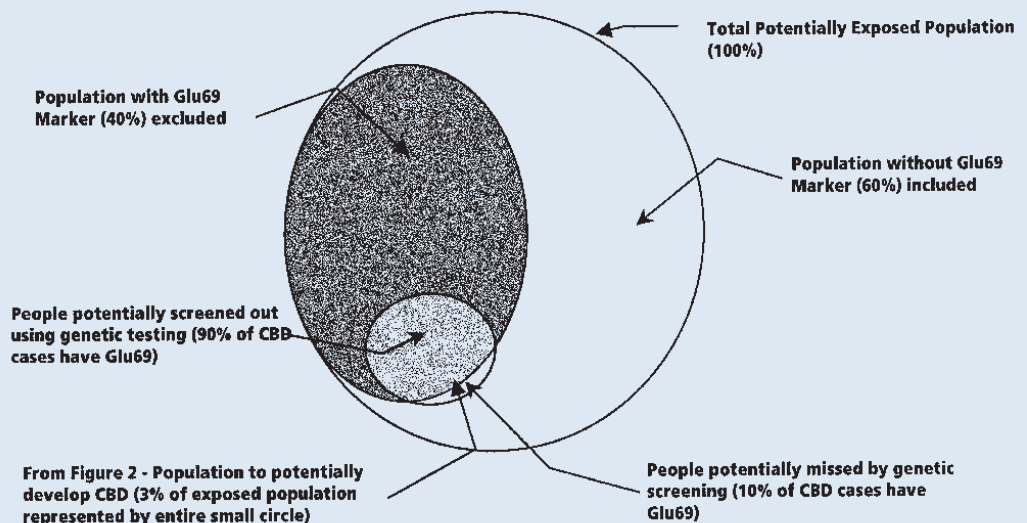
**Figure 1**

### Exposed Population at Risk



**Figure 2**

### Glu69 Genetic Screening Summary



**Table 3**

## Standards & Recommendations for Airborne Beryllium Levels

Agency/Group	Current Status	Exposure Limit
OSHA (PEL)	Law	2 µg/m <sup>3</sup> as an 8-hour TWA; 5 µg/m <sup>3</sup> as a ceiling not to be exceeded for more than 30 minutes; 25 µg/m <sup>3</sup> as a peak never to be exceeded.
ACGIH (TLV)	Recommendation	2 µg/m <sup>3</sup> as an 8-hour TWA; 10 µg/m <sup>3</sup> STEL.
NIOSH (REL)	Recommendation	0.5 µg/m <sup>3</sup> as a 10-hour TWA.
EPA (NESHAP)	Law	0.01 µg/m <sup>3</sup> for any 30-day period near a stationary source.

### What is 2µg/m<sup>3</sup>?

To gain perspective on what a 2µg/m<sup>3</sup> concentration is, consider this analogy: If one takes a grain of table salt, pulverizes it and evenly disperses it throughout a large house (2,200 sq. ft.), the concentration would be about 2µg/m<sup>3</sup>. Now, take the OSHA PEL (and the single grain of table salt), then divide it by 10 to arrive at the proposed ACGIH TLV of 0.2 µg/m<sup>3</sup>. This exposure level is, for all practical purposes, invisible to the naked eye.

TWA exposure. This is evidenced by individuals at beryllium processing plants who have contracted CBD despite no industrial exposure.

With medical and epidemiological evidence suggesting that peak beryllium exposures play a major role in disease etiology, ACGIH and OSHA remain locked in TWA mode. The literature suggests that OSHA's PEL of 2 µg/m<sup>3</sup> TWA is not protective [Viet, et al 245, 252; ACGIH; EPA(c); Strange, et al 416; Kreiss, et al 25]. In addition, DOE employees continue to experience CBD at exposure levels below this TWA. EPA conducted a comprehensive hazard assessment for beryllium sensitization and CBD by reviewing several reports on beryllium exposure cohorts from the U.S. nuclear industry. The study presents a range of lowest observable adverse effect levels (LOAEL) for industrial beryllium exposure from 0.036 µg/m<sup>3</sup> to 0.37 µg/m<sup>3</sup> [EPA(c) 12].

This suggests that merely complying with OSHA's PEL may expose a workforce to airborne levels of beryllium that are documented to cause disease. It should be noted that OSHA's actions are limited by federal procedural requirements. In fact, the beryllium PEL has remained unchanged since 1969. For well over a year, OSHA has been involved in reevaluating the beryllium PEL. As noted, ACGIH has listed beryllium in its "Notice of Intended

Changes" with a "sensitize" listing and a proposed reduction in the TLV from 2 µg/m<sup>3</sup> TWA to 0.2 µg/m<sup>3</sup>. Based on these findings, SH&E professionals should consider the benefits of imposing a ceiling value or immediately dangerous to life and health value on their workplace. This aggressive strategy would provide a mechanism for eliminating hazards associated with peak exposures permitted with a TWA.

Epidemiologists and medical researchers continue to discuss the importance of particle size as it relates to toxicity [EPA(c); ACGIH]. Currently, OSHA, ACGIH and NIOSH exposure levels contain no size-selective constraints—although ACGIH has proposed limiting the TLV for beryllium to inhalable particles to better control the mass of beryllium capable of entering the upper respiratory track (ACGIH 72). As

a result, the research focus may turn to the relationship between particle size and disease.

The medical profession takes a conservative, albeit qualitative, approach by informing the public, "Anything that has some beryllium content may cause chronic beryllium disease. Even low levels of exposure in a sensitive person can lead to significant lung disease" (Balkissoon). To protect workers against SENS and CBD, SH&E professionals should heed this published, ominous warning. Beryllium is also a known human carcinogen according to the International Agency for Research on Cancer (IARC), OSHA and ACGIH (17); oddly enough, this fact is trivialized in light of the extreme severity related to CBD.

### Industrial Exposure Sources & Primary Routes of Entry

Does your facility manufacture beryllium alloys? Add beryllium to products? Smelt ores contaminated with any beryllium? Remelt any metals that contain beryllium? Machine any beryllium or beryllium-containing alloys? Burn coal or coke? If the answer to any of these questions is "yes," then a workforce is at risk of exposure to beryllium dust and fume and, therefore, of contracting SENS or CBD. Those who believe their facilities' exposure scenarios are not worth evaluating because there is little chance of beryllium exposure should consider this statement: "The potential health risk has been underestimated because the low level of beryllium content [in beryllium-copper alloys] has been trivialized" (Balkissoon). In clinical evaluations, medical professionals do not quantify industrial exposure to beryllium when estimating risk of CBD since any industrial exposure to airborne beryllium can result in CBD. If any beryllium can become airborne, then employees should be considered as at-risk of contracting CBD.

Molten metal operations, machining, finished product comminution and dust and fume collection systems are all potential sources of airborne beryllium.



Beryllium-contaminated dust-disturbing activities (e.g., sampling, cutting, burning, welding, cleaning) also represent a significant source of exposure. Disturbance of as little as 100 µg/100 cm<sup>2</sup> can cause an airborne exposure of greater than 0.2 µg/m<sup>3</sup> [DOE(c)]. The “personal cloud” is another source of airborne beryllium. A worker with contaminated clothing or shoes can be the source of exposure—both to him/herself and to others by releasing the beryllium trapped while walking, working or eating. This process is commonly referred to as “track out.” SH&E professionals should take precautions to restrict dust-disturbing activities and minimize track out.

Beryllium can enter the body through any of the three available mechanisms. The primary route of entry is the lungs, while skin absorption and ingestion are secondary routes of entry. Cuts in the skin provide a route of entry that results in subcutaneous granulomatous nodules. Ingestion represents a tertiary route of entry. Once in the body, beryllium is cleared slowly, with perhaps 50 percent clearing at six months post-exposure and the balance possibly never clearing [Rossman(b)]. Because beryllium body burden has such a long residence time, the risk is life-long that the body’s immune system will recognize and overreact to its presence. This fact is corroborated in published latency periods as long as 30 years for SENS and CBD (Rossman “Personal”; NJMRC; ATSDR). This fact also lends credibility to the assumption that increasing body burden above an undetermined cumulative dose may play a role in conversion to SENS/CBD.

### Beryllium Sensitization & Chronic Beryllium Disease

As noted, a worker’s sensitivity to beryllium—not the dose—determines disease occurrence. This

**Table 4**

## Symptoms of CBD

- Persistent coughing
- Fatigue
- Loss of appetite
- Chest and joint pain
- Blood in the sputum (sputum is saliva, mucus and other discharges that can be “coughed up” from the respiratory system)
- Rapid heart rate
- Shortness of breath with physical exertion
- Fevers and night sweat

explains why SENS is generally the first step the body takes on the path to CBD. Thus, understanding and preventing SENS is critical to reducing the risk of CBD. Reduction of SENS can best be accomplished by keeping industrial airborne beryllium levels as low as reasonably achievable (ALARA) and certainly no higher than the LOAEL range of 0.036 µg/m<sup>3</sup> to 0.37 µg/m<sup>3</sup> [EPA(c)]. Therefore, achieving these levels should be the first priority of any beryllium disease prevention plan.

The immune system’s memory, with a defined immunological response specific to beryllium, is generally referred to as beryllium sensitization or hypersensitivity (aka SENS). Even a single, brief exposure to a low concentration of beryllium above one’s personal threshold can engage the immune system, initiating SENS. At this point, the body’s immune system develops a permanent memory related to beryllium particles that allows the

**Table 5**

## Risk Assessment Assumptions

Risk	Range	Probability Estimate
Probability of becoming SENS after exposure to beryllium.	1 to 16 percent depending on job class.	Approx. 10 percent or 1/10.
Probability of contracting CBD after exposure to beryllium.	1 to 6 percent depending on job class	Approximately 3 percent or 1/30 (Lombardi)
Probability of progressing to CBD after becoming SENS.	50 to 85 percent	75 percent or 3/4 [Rossman(b)]
Probability one has the Glu69 genetic marker.	30 to 50 percent	Approximately 40 percent or 1/2.5 [Rossman(b)]
Probability of developing CBD after exposure to beryllium while possessing both Glu69 alleles.	Roughly a risk enhancement of 5 as double Glu69 (+) (Wang, et al; Richeldi, et al).	5 x 1/30 = 1/6
Probability of developing CBD if one does not have the Glu69 alleles.		1/333
EPA acceptable health risk	1/1 million to 1/100,000 [EPA(a) 1-17]	1/1 million for carcinogens

*Chronic beryllium disease is an immunological disease. It is progressive—even after exposure ceases—irreversible, disabling and potentially fatal.*

immune system to attack beryllium already in the body or any future inhaled, ingested or absorbed beryllium. SENS is a permanent health effect and is OSHA Form 300-recordable.

The latency period (average time from first beryllium exposure to development of symptoms) for SENS/CBD is highly variable, starting at approximately six months and remaining in place for the rest of one's life. It is

believed that the disease threshold can vary considerably within a workforce from individual to individual and over time. A disease-free, beryllium-exposed individual can become SENS later in life after a significant stressor reduces the immune system and correspondingly one's personal threshold later. Intercurrent life stressors such as pregnancy, birth, lactation or serious illness may also be related to initiation of SENS and subsequently CBD (Newman, et al 940).

Chronic beryllium disease is an immunological disease. It is progressive—even after exposure ceases—irreversible, disabling and potentially fatal. CBD is the enduring damage caused by the immune system's overreaction to a beryllium lung burden. Its latency period can be as short as six months but the risk of disease occurrence is life-long; the typical latency period is 10 to 15 years [Rossman(b)]. Table 4 lists typical CBD symptoms (Sprince 391).

As noted, lymphocytes and macrophages attack beryllium in the body, generating granulomas and

scar tissue. The site of the attack (wherever it might be in the body) is scarred and disabled. The area of greatest concern is the lung; the liver and skin are secondary target organs. The progressive granulomas and the associated scar tissue in the lung reduce its elasticity, create obstructions and effectively reduce its ability to exchange O<sub>2</sub>-CO<sub>2</sub> in the gas exchange region (alveoli). Individuals with advanced CBD require a constant supply of oxygen and face death due to the disease. Essentially, one either suffocates to death from CBD or dies from heart failure (cor pulmonale). Because this attack on the body is due to hypersensitivity, CBD is often controllable with anti-inflammatory medication such as corticosteroids. However, these drugs have many deleterious side effects, including weight fluctuations, central nervous system disorders and fatigue. It is not difficult to understand why CBD is a feared industrial disease. Its prevention will only be realized when the disease etiology is well-understood and industrial exposure is significantly reduced or eliminated.

#### **Occurrence & Risk of SENS & CBD**

An important observation is that the general population does not experience CBD at a recognizable rate—it appears to nearly exclusively reside in the industrial domain. This leads one to a discussion of a so-called "safe level" of exposure or NOAEL (no observed adverse effect level). Outdoor, ambient concentrations of beryllium are generally below the NESHAP (0.01 µg/m<sup>3</sup>). Since the general public does not experience CBD and since industry still experiences CBD at exposures as low as 0.2 µg/m<sup>3</sup> TWA, one can conclude that the NOAEL is less than 0.2 µg/m<sup>3</sup> TWA but not lower than the NESHAP.

For empirical support, one need look no further than DOE facilities that handle beryllium. Many of these facilities continue to report SENS among new hires who are working under the federally required Chronic Beryllium Disease Prevention Program (CBDPP)—a program that must be managed by a CIH when workplace airborne exposure exceeds 0.2 µg/m<sup>3</sup> TWA [DOE(c)]. In addition, every published study of a cohort of U.S. workers exposed to beryllium and beryllium compounds (except mineral forms) has reported measurable rates of both SENS and CBD. Disease prevalence has varied, but the results are clear: If workers are exposed to beryllium and beryllium compounds (other than mineral forms) above LOAEL, beryllium-related diseases will occur in a significant portion of the workforce.

As a case in point, consider the the British Atomic Weapons Establishment in Cardiff, Wales, where employees handled beryllium metal from 1960 to 1997 (Weitzman). The site employed a generally stable workforce of 300 employees with no reported CBD cases over the 37-year

## **A Growing Need to Update PELs**

In a recent letter sent to Charles Norwood, Chair of the U.S. House of Representatives Subcommittee on Workforce Protections, ASSE commented on whether consensus can be reached in revising PELs. The Society voiced its concern that the "PELs currently promulgated by OSHA have not been revised since 1971" and said that the standards are grossly outdated for many of the covered toxic substances in light of the epidemiological evidence compiled over the past 30 years and other scientific information currently available. It also noted that although the National Toxicology Program and/or the International Agency for Research on Cancer have designated some of these substances as human carcinogens, the OSHA and PELs for them remain at levels injurious to human health.

"ASSE is convinced that enough credible scientific information is available to build consensus toward lowering the PELs for many substances and/or developing PELs for new chemicals that were not addressed in the rules of the 1970s. Many employers are already voluntarily protecting workers at levels below the PELs, but others, due largely to the lack of agency guidance, are unaware that workers are not sufficiently protected at the currently legal limits." The Society added, "Updating PELs is an achievable goal that can be accomplished in a timely manner based on information and methodologies that already exist or can be easily established. Furthermore, every time this issue has been raised in the past, a general consensus that updating PELs was necessary was readily achieved. There exists among most stakeholders the realization that revitalizing these standards will result in saved lives and improved health among American workers."

## Genetic Screening for Risk Reduction

Genetic screening is a powerful tool in reducing the risk of beryllium exposure. The presence of Glu69 has been associated with 85 to 95 percent (say 90 percent) of CBD cases. In an airborne beryllium-exposed population of 100 employees, 40 percent (40 people) have the Glu69 marker. Prevent this population from being exposed and that leaves 60 nongenetically predisposed people exposed to the risk of CBD. Screening out employees who are Glu69(+) will potentially eliminate 9 out of 10 anticipated CBD cases. Of the 60 exposed individuals, probability estimates indicate less than one CBD case (0.3 CBD cases; 1/333).

This scenario of potentially incurring less than 1 CBD case out of 100 exposed (or 1/333) is a marginal improvement on the 1 in 30 probability originally estimated (see pg. 32). However, genetic screening can add a risk-reduction factor of approximately 10. It should be noted that 1/330 remains a dangerously high risk when compared to the generally accepted levels of 1 in 100,000 to 1 in 1 million. Exposing even a genetically screened individual to airborne beryllium remains extremely risky and unsafe.

operating period. While it adopted a  $2 \mu\text{g}/\text{m}^3$  TWA of beryllium in air, typical airborne measurements were  $<0.1 \mu\text{g}/\text{m}^3$  TWA to  $0.2 \mu\text{g}/\text{m}^3$  TWA in the machine shop and  $0.5 \mu\text{g}/\text{m}^3$  TWA to  $1 \mu\text{g}/\text{m}^3$  TWA in the foundry. Airborne levels were rarely above  $2 \mu\text{g}/\text{m}^3$  TWA. To maintain these low exposure levels, the site:

- enforced meticulous housekeeping;
- imposed rigorous clean-up procedures;
- controlled access to process areas;
- curtailed track out;
- consistently gathered personal and area workplace exposure samples;
- implemented a state-of-the-art medical surveillance program as early as 1961;
- required PPE for all routes of entry; employees regularly wore respirators independent of exposure levels (reducing inhaled beryllium and lowering lung burden);
- carefully cleaned material of surface contamination before it was released.

Most significantly, the facility was originally designed (in 1960) to contain beryllium at its emission source through redundant containment systems that would resemble pharmaceutical controls in the U.S. The control measures used represent a significant (and prohibitively expensive) variation from standard practice in U.S. industry—both in the early 1960s and today. Contrast the Wales facility to DOE's Rocky Flats facility, where 122 CBD cases have been reported.

Even though machining beryllium may generate a particle size fraction suitable for deposition deep into the alveoli ( $<0.5$  micron aerodynamic diameter), the Wales facility's approach appears to have prevented CBD cases. This suggests that setting a protective occupational exposure level for beryllium is a major industrial hygiene challenge. That procedure must address limiting exposure for all routes

of entry, evaluating peak exposure and morphology, as well as considering chemical form. "Two to 17 percent of workers exposed to beryllium dust develop an allergy to beryllium, referred to as 'beryllium sensitization'" [Newman(a)], while one to six percent of workers exposed to beryllium dust will contract CBD (NJMRC). At the Rocky Flats facility, 9,484 workers have been medically screened; 193 are SENS (2.04 percent) and 122 have CBD (1.29 percent) [DOE(a)]—this despite implementing a sophisticated evaluation and exposure control system (CBDPP).

One quickly notices that whenever an industrial exposure to any beryllium occurs, there is a nonzero effect. If airborne beryllium above background levels (NESHAP) is present, all exposed workers are at considerable risk and there is a very high probability of quantifiable, permanent illnesses within the exposed population. Published information on CBD generally glosses over the fact that the percentages noted earlier reflect individual disease probabilities that, when clearly understood, would be considered unacceptable to most people. The following discussion examines this disquieting concept in more detail.

### What Is Considered "Safe"?

When SH&E professionals say it is "safe" to do something, they are referring to a risk assessment, either perceived or calculated. Regarding working with beryllium and compounds, many SH&E professionals have concluded (tacitly and in print) that it is "safe" to work with beryllium (Stalnaker 25). Unless one worked at the Cardiff facility, existing information contradicts that stance. It may be worth taking a closer look at the risks associated with exposure to beryllium, quantifying them where possible, comparing the calculated risk to acceptable levels, then presenting the findings through HazCom to the exposed workforce. While DOE facilities cannot ban or substitute for beryllium, they have recognized that the risk of contracting SENS and CBD is so high that the beryllium worker category is deemed volunteer status only.

A general, semiquantitative risk assessment can begin with the ranges provided by the medical and industrial hygiene communities. Table 6 presents a basis for quantitative risk assessment associated with exposure to beryllium. The results are not rigorously derived and may be perceived as somewhat conser-

*Business managers must make aggressive medical, technical and administrative choices in an effort to minimize the risk of exposure to airborne beryllium.*





vative. Also consider that the occurrence of CBD in the industrial population may be understated because 1) DOE facilities do not include CBD cases that may have occurred in temporarily exposed contractors; and 2) the disease is not well-recognized within the medical profession. The risk assessment can include safety factors to account for the lack of reliability in the probability estimate (high or low).

When an industrial operation exposes an individual employee to airborne beryllium, the probability of that person contracting CBD ranges from 1 in 16 to 1 in 100. EPA generally considers an upper-bound lifetime cancer risk to an individual of between 1 in 10,000 and 1 in 1 million as a safe range [EPA(a); (b)]. Even though these levels are for the general public based on 24 hour/day, 365 day/year exposure, the comparison is appropriate due to the fact that once a worker is exposed to beryllium, the risk of eventually developing the disease—after even a single exposure—is lifelong. Now, compare the 1-in-30 probability of contracting CBD after exposure to beryllium to the acceptable risk levels for exposure to EPA carcinogens (1 in 1 million). The initial conclusion must be that exposure to airborne beryllium carries an unacceptably high risk—or, said differently, is not safe. Certainly, one could argue the accuracy of the numbers selected for the “probability estimate” column in Table 6; they are not exact, are debatable and may be off by a factor of 10 depending on one’s reference. However, they are not off by a factor of 100,000, which is what is needed to place the risk of SENS and CBD into an acceptable domain.

Business managers regularly assume a risk of injury or illness on behalf of a workforce. In the case of beryllium exposure, the risk is so

**Machining, grinding, and dust- and fume-collection systems are potential sources of airborne beryllium. Beryllium-contaminated dust-disturbing activities (e.g., cutting, burning, welding, cleaning) also represent a significant source of exposure.**



high that this practice is not recommended. Business managers must make aggressive medical, technical and administrative choices in an effort to minimize the risk of exposure to airborne beryllium. Informed workers must also have a role in this decision-making process, as DOE has done. SH&E professionals should encourage general industry to follow that lead.

### Medical Surveillance

If SH&E professionals detect the presence of beryllium through hazard evaluation and hazard assessment techniques, they should institute a medical surveillance program directed toward early detection of SENS and CBD. The earlier these conditions are detected, the better the medical professional can manage the course of the disease. Medical and industrial hygiene professionals agree that the best available medical screening tool is the beryllium lymphocyte proliferation test (BeLPT) [NJMRC; Rossman (a); (b) 945+; DOE(c)]. Including chest x-ray and pulmonary function test (PFT) in medical screening programs are of lesser value in identifying SENS, as positive chest Xray and PFT are more likely associated with symptomatic CBD; they will aid diagnosis but not provide early detection.

The use of genetic testing as a screening tool should also be strongly considered. It has been used in a clinical setting for research purposes (Richeldi, et al(b) 337; Richeldi, et al(a) 242; Wang, et al; Lombardi, et al). To date, however, the benefits of this research have been limited due to the legal implications involved. The medical professional can establish test protocol wherein results are provided confidentially to the employee. The employee can then factor those results into his/her decision regarding acceptance of the risk related to beryllium exposure. This process can only work in a voluntary-based program. The medical professional must also be prepared to handle the notification process, as well as a range of possible employee responses. A plan for consistently managing affected employees could include additional disease information, workers' compensation information, assurance regarding job security and third-party counseling (Tan-Wilheim).

### Management Approach

A general business adage states, "If you can measure it, you can manage it." A corollary might be, "If you ignore it, it will bite you"—and it applies to management of exposure to beryllium. If SH&E professionals wish to exclude CBD from their HazCom program, they must make a significant attempt to detect and measure its presence and take all feasible actions to prevent SENS and CBD. A routine hazard assessment will indicate whether beryllium is present. An inventory will establish its locations. A statistically valid air sampling program will determine its concentration in the workplace. SH&E professionals must use the standard tools of industrial hygiene, and occupational health and safety to address beryllium exposure:

- Minimize the number of workers exposed to

### References

- American Conference of Governmental Industrial Hygienists (ACGIH).** 2001 *TLVs and BEIs for Chemical Substances and Physical Agents*. Cincinnati: ACGIH, 2001.
- American Institute of Chemical Engineers (AIChE).** *Guidelines for Hazard Evaluation Procedures*. 2nd ed. New York: AIChE, 1992.
- Balkissoon, R.** "Chronic Beryllium Disease." Denver: NJMRC, Aug. 9, 2001. <<http://www.nationaljewish.org/news/balkissoonbercop.html>>.
- Barnard, A., et al.** "Retrospective Beryllium Exposure Assessment at the Rocky Flats Environmental Technology Site." *AIHA Journal*. 57(1996): 804-808.
- Dept. of Energy (DOE)(a).** "Chronic Beryllium Disease Prevention Program." 10 CFR 850. *Federal Register*. Dec. 8, 1999: 68854-68914.
- DOE(b).** *Implementation Guide for Use With 10 CFR 850: Chronic Beryllium Disease Prevention Program*. DOE G 440.1-7A. Washington, DC: DOE, 2001.
- DOE(c).** *Implementation Workshop for 10 CFR 850 Regulations Related to Chronic Beryllium Disease*. Washington, DC, March 5-8, 2001.
- Dept. of Health & Human Services (DHHS).** "Case Studies in Environmental Medicine: Beryllium Toxicity." Washington, DC: DHHS, Agency for Toxic Substances and Disease Registry, July 1992.
- EPA(a).** "Guidelines for Carcinogen Risk Assessment." EPA Risk Assessment Forum, NCEA-F-0644 Review Draft. Washington, DC: EPA, July 1999.
- EPA(b).** "National Oil and Hazardous Substances Pollution Contingency." *Federal Register*. March 5, 1996.
- EPA(c).** "Toxicological Review of Beryllium and Compounds in Support of Summary Information on the Integrated Risk Information System." Washington, DC: EPA, April 1998.
- Kreiss, K., et al.** "Machining Risk of Beryllium Disease and Sensitization with Median Exposure Below 2 µg/m<sup>3</sup>." *American Journal of Industrial Medicine*. 30(1996):16-25.
- Lide, D., ed.** *Chemical Rubber Company Handbook of Chemistry and Physics*. 77th ed. Boca Raton, FL: CRC Press, 1996.
- Lombardi, G., et al.** "HLA-DP Allele-Specific T-Cell Response to Beryllium Account for DP-Associated Susceptibility to Chronic Beryllium Disease." *The Journal of Immunology*. 166(2001): 3549-3555.
- Manuele, F. and B. Main.** "On Acceptable Risk." *Occupational Hazards*. 64(2002): 57-60.
- National Jewish Medical and Research Center (NJMRC).** MedFacts: Facts About Beryllium Disease, Aug. 1, 2001. <[http://www.nationaljewish.org/medfacts/beryllium\\_medfact.html](http://www.nationaljewish.org/medfacts/beryllium_medfact.html)>.
- National Safety Council.** "What Are the Odds of Dying? Odds of Death Due to Injury, U.S., 1998." <<http://www.nsc.org/lrs/statinfo/odds.html>>.
- Newman, L.(a).** "Hearing on Compensation for Beryllium-Related Illness." Testimony before the U.S. House of Representatives Subcommittee on Immigration and Claims, Sept. 14, 2000.
- Newman, L.(b).** "To Be+2 or Not to Be+2: Immunogenetics and Occupational Exposure." *Science*. 262(1993): 197-198.
- Newman, L., et al.** "The Natural History of Beryllium Sensitization and Chronic Beryllium Disease." *Environmental Health Perspectives*. 104S(5): 937-943.
- Richeldi, L., et al(a)** "HLA-DPB1 Glutamate 69: A Genetic Marker of Beryllium Disease." *Science*. 262(1993): 242-244.
- Richeldi, L., et al(b).** "Interaction of Genetic and Exposure Factor in the Prevalence of Berylliosis." *American Journal of Industrial Medicine*. 32(1997): 337-340.
- Rossman, M.(a).** "Chronic Beryllium Disease: Diagnosis and Management." *Environmental Health Perspectives*. 104S:5(1996): 945-947.

*The risks of becoming SENS or contracting CBD are unacceptably high, making even short-duration, low-dose industrial exposure to beryllium dangerous.*

(References continued on page 40)

## What is Sarcoidosis?

CBD is often misdiagnosed as sarcoidosis, which possesses similar symptoms. Sarcoidosis is a disease that occurs when areas of inflammation develop in different organs of the body. Granulomas are also seen with this disease. They may occur in the lungs, lymph nodes, eyes, skin or any area of the body. The granulomas may clear up on their own or cause permanent scarring. The cause of sarcoidosis is unknown. About 10 to 40 out of every 100,000 people develop the condition; it is most common in people between age 20 and age 40. Like SENS/CBD, sarcoidosis is not contagious.

Source: NJMRC

beryllium. The fewer workers exposed, the fewer cases of SENS and CBD.

- Conduct hazard assessments and hazard evaluations (AICHE).

- Take airborne samples (personal and area). Look for peak exposures as well as TWA values as even a single exposure above one's sensitivity threshold can initiate disease.

- Conduct surface wipe tests. (Take care, however, as even the act of taking wipe samples can expose the industrial hygienist to harmful levels of airborne beryllium) [DOE(c)].

- Discuss the hazards of beryllium exposure in HazCom training.

- Operate a comprehensive medical surveillance program.

- Conduct epidemiological research (past disease incidence, IH records, workers' compensation data, death certificate review).

- Implement a dedicated management system to clearly and confidently address SENS and CBD prevention.

DOE's Chronic Beryllium Disease Prevention Plan is the best available management system. DOE has also issued a guidance document to help its facilities implement the program. Even though not

required in general industry, this program provides a well-developed, systematic method of managing beryllium exposure and its associated health risks, including SENS and CBD.

### Conclusion

The hazards associated with occupational exposure to beryllium cannot be eliminated. It is legal to use or produce beryllium and beryllium compounds—and to expose the workforce to airborne beryllium. Utilizing published medical epidemiological data, SH&E professionals should draw their own conclusions about the risk of SENS/CBD occurrence within their workforce. They must then factor in the risk-reduction evidence associated with a world-class CBDPP in order to judge whether the residual risk is tolerable and acceptable (Manuele and Main 60). As the information and assumptions in this article attest, the author believes that the residual risk is unacceptable, making industrial exposure above LOAEL to airborne beryllium unsafe. If beryllium exposure were managed like it was at the Cardiff, Wales, facility, one could argue that beryllium can be handled safely. Unfortunately, standard industrial practice excludes many of those control measures.

To comprehensively evaluate your site regarding the impact beryllium:

- Involve management, SH&E staff, medical providers and the affected workforce early in the process.

- Ask SH&E staff to review MSDS for evidence of

beryllium content (since beryllium is a carcinogen, it will show up on the MSDS down to 0.1 percent). Contact suppliers of suspect materials for more details on their beryllium content.

- Ask SH&E staff to monitor for beryllium (airborne and surface).

- Ask medical providers to review past medical records for evidence of SENS, CBD and any other specific or idiopathic lung disorders that may be related to beryllium exposure.

- Consider genetic screening for the workforce.

- Screen all newly purchased materials for beryllium content.

- Update the HazCom program to review hazards associated with beryllium exposure.

- Implement a CBDPP—and take no short-cuts.

- Make exposure to beryllium voluntary.

SH&E professionals often focus on acute injury resolution. Certainly, a disease such as CBD with its long latency period, complicated medical diagnosis and low profile in industrial medicine can remain below the radar. It should not. The documented risks of becoming SENS or contracting CBD are unacceptably high, making even short-duration, low-dose industrial exposure to beryllium dangerous. Basic risk assessment indicates that industrial exposure to beryllium is unsafe. Furthermore, the personal and emotional consequences associated with SENS and CBD are tremendous. Until beryllium use is restricted, SH&E professionals must raise awareness among management and employees of the extreme toxicity associated with airborne beryllium exposure so that informed decisions can be made. ■

### References (continued from page 39)

- Rossmann, M.(b). Personal conversation. Feb. 2, 2000.
- Sprince, N. "Beryllium Disease." In *Occupational Respiratory Diseases*, J.A. Marchant, ed. DHHS (NIOSH) Publication No. 86-102. Washington, DC: DHHS, NIOSH, 1986. 385-399.
- Stalnaker, C.K. "Understanding and Controlling Beryllium Hazards." *Professional Safety*. November 1999: 22-25.
- Stokinger, H. *Beryllium: Its Industrial Hygiene Aspects*. New York: Academic Press, 1966.
- Strange, A., et al. "Beryllium Sensitization and Chronic Beryllium Disease at a Former Nuclear Weapons Facility." *Applied Occupational and Environmental Hygiene*. 16:3(2001): 405-417.
- Tan-Wilheim, D., et al. "Impact of a Worker Notification Program: Assessment of Attitudinal and Behavioral Outcomes." *American Journal of Industrial Medicine*. 37(2000): 205-213.
- Viet, S., et al. "Chronic Beryllium Disease and Beryllium Sensitization at Rocky Flats: A Control Study." *AIHA Journal*. 61(2000): 244-254.
- Wang, Z., et al. "Differential Susceptibilities to Chronic Beryllium Disease Contributed by Different Glu69 HLA-DPB1 and DPA1 Alleles." *The Journal of Immunology*. 163:3(1999): 1647-1653.
- Weitzman, D. "Beryllium Control Model Atomic Weapons Establishment Cardiff, Wales." Prepared by Atomic Weapons Establishment and U.S. DOE, June 25, 1997.

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