

RESEARCH ARTICLE

# Development of a health risk-based surface contamination cleanup standard for occupational exposure to beryllium

Paul Damian

SCS Engineers, 852 Northport Drive, Suite 5, West Sacramento, CA 95691, USA

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## Abstract

A health risk-based surface contamination cleanup standard (SCS) for beryllium (BE) was developed to facilitate the safe transfer of property (equipment and buildings) previously used in BE-related processes. Previous SCSs for BE were primarily based on Department of Energy (DOE) housekeeping criteria rather than health risks. Quantitative health risk assessment methods were used to develop an occupational SCS that explicitly considers the relevant exposure pathways and toxicity endpoints, including both cancer and non-cancer endpoints. For the cancer endpoint at the 1E-06 risk level, the analysis resulted in an SCS of 17  $\mu\text{g}/100\text{cm}^2$  based on resuspension of settled dust and subsequent inhalation exposure only (BE is regulated as a carcinogen by the inhalation route only). For the non-cancer endpoint, the analysis resulted in an SCS of 0.07  $\mu\text{g}/100\text{cm}^2$  based on dermal absorption, incidental ingestion following dermal contact, and inhalation. The non-cancer SCS was determined virtually entirely by the dermal absorption exposure pathway, with negligible contributions from the incidental ingestion and inhalation pathways. This analysis shows that application of the non-cancer SCS in BE monitoring and control programs will adequately protect workers from both the cancer and non-cancer health effects of BE when surface contamination is the primary source of BE exposure.

**Keywords:** Risk assessment; dermal absorption; contaminated buildings; resuspension factor

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## Introduction

Beryllium (BE) is a contaminant typically encountered at Department of Energy (DOE) sites and other nuclear-related facilities. The use of BE at these facilities over the years has resulted in various degrees of building contamination and presents challenges when the property is decommissioned for other non-nuclear related uses. An important issue is how best to evaluate whether a BE-contaminated building or piece of equipment has been adequately decontaminated so that the building or equipment is safe for the future intended use. Answering this question requires comparison of BE contamination levels on interior building or equipment surfaces with a surface contamination cleanup standard (SCS). In order to adequately protect the health of future occupants of the building, this standard should be based on health risk considerations. In particular, it should explicitly address the three exposure pathways by which workers may be exposed to contaminants on interior building surfaces or equipment. These exposure pathways include: direct dermal contact with the contaminated surface and subsequent dermal

absorption, incidental ingestion of contaminant transferred to the hands after direct dermal contact, and inhalation of contaminant resuspended into the air (for buildings). However, at the present time, although a SCS for BE has been established by DOE, this standard is not health risk-based, but rather is based on practical housekeeping criteria. Since the standard was not developed based primarily on health risk considerations, there is little justification for the current value if the purpose of the standard is to ensure the safety of future building occupants. The purpose of this paper is to propose a health risk-based SCS for BE based on standard risk assessment methods and available BE toxicity data. Such a standard is needed to facilitate transfer of property contaminated with BE while ensuring adequate protection of worker health.

BE is a naturally occurring metal that is non-corrosive and stiffer than steel (Tinkle et al. 2003). It is one of the lightest metals. The average concentration in US soils is 0.63 parts per million (ppm) with a range of < 1–15 ppm in the western US (United States Geological Survey [USGS] 1984). BE is used

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Address for Correspondence: Paul Damian, PhD, MPH, DABT, SCS ENGINEERS, 852 Northport Drive, Suite 5, West Sacramento, CA 95691, USA. Tel: 916-372-2014. Fax: 916-372-2033. Email: pdamian@scsengineers.com

as the pure metal in specialized nuclear and defense-related equipment and processes. It is used in metal alloys for the manufacture of electric parts, automobiles, computers, and some sports equipment, such as golf clubs (Agency for Toxic Substances and Disease Registry (ATSDR) 2007).

Because of its highly specialized uses, BE poses health risk issues primarily to workers in the BE mining and processing industry and at defense facilities. These health risk issues include both cancer and non-cancer health risks. BE has been designated as a Probable Human Carcinogen (Group B1 classification) by the US Environmental Protection Agency (USEPA) which states that 'inhaled beryllium would be characterized as a "likely" carcinogen in humans' based on increased incidences of lung cancer in BE workers (USEPA 2010). The National Toxicology Program (NTP) and the International Agency for Research on Cancer (IARC) also consider BE a human carcinogen. For the purposes of quantifying cancer risk related to inhalation exposure, USEPA has developed an air unit risk factor for BE of  $2.4E-03$  per  $\mu\text{g}/\text{m}^3$ . This unit risk factor is equivalent to a cancer potency factor (CPF) of 8.4 per  $\text{mg}/\text{kg}\text{-day}$ . However, with respect to ingestion exposure, USEPA (2010) states that 'the human carcinogenic potential of ingested beryllium cannot be determined'. The California Environmental Protection Agency (Cal-EPA) regulates BE the same way as the USEPA, treating it as carcinogenic only via the inhalation route of exposure (Cal-EPA 2003).

In addition to being carcinogenic by the inhalation route of exposure, BE also poses characteristic non-cancer health risks. In particular, chronic beryllium disease (CBD) and BE sensitization. CBD is an incurable, progressive, inflammatory lung disease observed most commonly in BE workers. The disease can be considered idiosyncratic since it only occurs in 2–5% of exposed workers (Kreiss et al 1993). It is characterized by granuloma formation and fibrosis. Symptoms include chest pain, coughing, fatigue, and shortness of breath. BE sensitization characteristically precedes the development of CBD. BE sensitization is symptomless and is therefore diagnosed using a blood test called the beryllium lymphocyte proliferation test (BeLPT). This test detects the elevated immune response to BE exposure.

For the purposes of assessing the non-cancer health risks of BE, USEPA has established a Reference Dose (RfD) for the ingestion route of exposure of  $2E-03$   $\text{mg}/\text{kg}$  body weight-day ( $\text{mg}/\text{kg}\text{-day}$ ) (USEPA 2010). An RfD is defined by USEPA (1989) as, '... an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level to the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime' page 72. Consistent with the definition of the RfD and RfD methodology, the BE RfD is based on the 'critical effect', or the adverse effect occurring at the lowest level of exposure documented in the toxicology literature. In the case of BE, USEPA has identified 'small intestinal lesions' as the critical effect. USEPA has also developed a Reference Concentration (RfC) of  $2E-05$   $\text{mg}/\text{m}^3$  for assessing the non-cancer risks of BE via inhalation exposure (USEPA 2010).

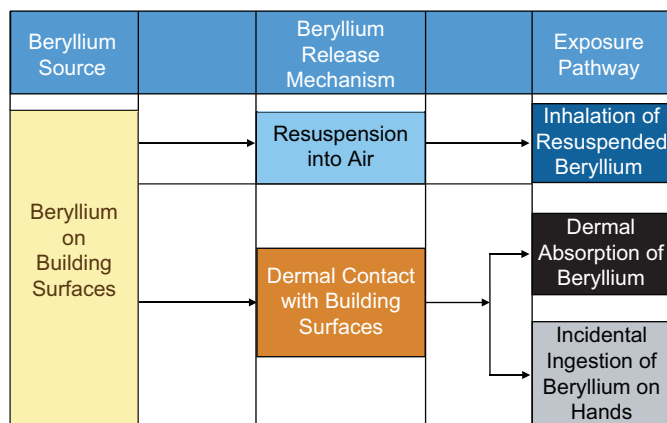
This RfC is equivalent to an inhalation RfD of  $5.72E-06$   $\text{mg}/\text{kg}\text{-day}$ . The inhalation RfC is based on BE sensitization and progression to CBD as the critical effect.

The current SCS, referred to as a 'release criteria', of  $0.2 \mu\text{g}/100 \text{cm}^2$  was established by DOE. The term 'release criteria' refers to the purpose of this standard as defining the maximum BE levels allowed on equipment before it can be released for other uses or to the general public. Note that this criterion was designed to address equipment rather than building surfaces. Most importantly, this criterion is not based on health risks but rather a 'housekeeping' standard of  $0.1 \mu\text{g}/100 \text{cm}^2$  'believed to be in effect at various DOE facilities and the AWE [Atomic Weapons Establishment] facility in the United Kingdom' (Federal Register 1999, page 68886). The initial proposed value of  $0.1 \mu\text{g}/100 \text{cm}^2$  was later modified to  $0.2 \mu\text{g}/100 \text{cm}^2$  based on the excessive cost of achieving the lower standard and the demonstrated feasibility of achieving the higher standard at Rocky Flats (Federal Register 1999). DOE also noted that Rocky Flats personnel and/or contractors conducted an analysis which showed that implementation of the higher standard of  $0.2 \mu\text{g}/100 \text{cm}^2$  would not result in any exceedance of USEPA's National Emission Standards for Hazardous Air Pollutants limit of  $0.01 \mu\text{g}/\text{m}^3$  and therefore, on that basis, would be a safe standard. This analysis, which apparently uses new resuspension factors that can be used to evaluate inhalation exposure, appears to be unpublished, and could not be found for review or incorporation in this paper. There is no indication that this analysis considered the other main exposure pathways associated with contaminated building surfaces, specifically dermal absorption or incidental ingestion.

In summary, the current DOE release criteria of  $0.2 \mu\text{g}/100 \text{cm}^2$  was primarily based on a housekeeping standard, supported to some degree by an unpublished analysis that apparently showed that this standard would prevent exceedance of the NESHAP for BE. The objective in developing the proposed standard in this paper is to provide a transparent (peer reviewable) health risk-based rationale that explicitly addresses the three main exposure pathways for worker contact with building surface contamination.

## Methods

Development of any cleanup standard for a chemical requires first determining what exposure pathways are relevant. This is often best illustrated with a conceptual model, as shown in Figure 1. Figure 1 summarizes the relationships between the original source of the contaminant (the building surface), the release mechanisms (how the contaminant becomes available for uptake by the individual), and the exposure pathways (the mechanisms by which individuals contact the contaminant). Figure 1 shows that BE on building or equipment surfaces is released by two mechanisms: removal by direct dermal contact and resuspension into air (by mechanical disturbances such as sweeping, dusting, etc.). BE is then taken up by the exposed individual through the following three exposure pathways, as shown in Figure 1:



**Figure 1.** Conceptual Model for the Development of a Health Risk-Based Beryllium Surface Contamination Standard.

- Direct dermal contact and subsequent dermal absorption;
- Direct dermal contact and subsequent incidental ingestion from hand-to-mouth transfer; and
- Inhalation of contaminant resuspended into room air from the building or equipment surface.

The next step in developing a SCS is determining which toxic endpoint of BE is relevant for each exposure route. Some chemicals are carcinogenic by one route of exposure and not by others, and BE is such a chemical. As noted above, BE is currently regulated by both USEPA and Cal-EPA as carcinogenic by the inhalation route and non-carcinogenic via all other routes of exposure (ingestion and dermal). Development of health-risk based standards for such chemicals normally entails calculating the standard based on both cancer risk and non-cancer health risks and selecting the most conservative (lowest) of the resulting two standards as the final standard. For the cancer risk-based standard only the inhalation route is relevant and included in the SCS calculation. For the non-cancer-based standard all exposure routes are relevant.

#### Calculation of the cancer risk-based standard

Since BE is not a volatile compound, it becomes airborne only when BE-containing dust is resuspended from building surfaces through mechanical disturbance such as sweeping, dusting, moving furniture, etc. A daily exposure level (Chronic Daily Intake or CDI) associated with this pathway was calculated as follows:

$$CDI_{inh} = \frac{SCS \times RF \times InhR \times CF \times EF \times ED}{BW \times AT} \quad (1)$$

where:  $CDI_{inh}$  = Chronic Daily Intake from inhalation (mg/kg-day); SCS = Surface contamination cleanup standard (mg/cm<sup>2</sup>); RF = Resuspension factor (cm<sup>-1</sup>); InhR = Inhalation rate (m<sup>3</sup>/day); CF = Conversion factor (cm<sup>3</sup>/m<sup>3</sup>); EF = Exposure frequency (days/year); ED = Exposure duration for adult (years); BW = Body weight (kg); and AT = Averaging time (days).

The inhalation rate, InhR, of 20 m<sup>3</sup>/day, is a standard risk assessment assumption for workers (USEPA 1989). The

exposure frequency, EF, corresponds to the number of days per year an individual would be expected to contact building surfaces. A value of 250 days per year is the standard assumption for commercial workers (USEPA 2004). The exposure duration, ED, is the total number of years an individual would be expected to work in the building. A value of 25 years is a standard assumption for this value (USEPA 2004). The body weight, BW, is the average body weight for an adult. The value of this parameter is 70 kg (USEPA 2004). The averaging time, AT, is the total number of days over which the exposure is averaged in the life of the individual. For carcinogens, the standard assumption is always 70 years or 25,550 days (USEPA 2004). An RF value of 1E-06 m<sup>-1</sup> (1E-08 cm<sup>-1</sup>) was obtained from Abu-Eid et al. (2002).

Cancer risk is calculated from the CDI as follows:

$$Cancer\ risk = CPF \times CDI \quad (2)$$

Setting cancer risk equal to the negligible cancer risk level of 1E-06 (one in a million), combining equations (1) and (2), and rearranging to solve for SCS results in the following equation:

$$SCS = \frac{1 \times 10^{-6} \times BW \times AT}{CPF \times RF \times InhR \times CF \times EF \times ED} \quad (3)$$

Solving the above equation using the parameter values described in detail above results in a cancer risk-based SCS of 17 µg/100 cm<sup>2</sup>.

#### Calculation of the non-cancer-based standard

As discussed above, all exposure pathways are relevant for the non-cancer risk standard. Direct dermal contact with contaminated building surfaces is the most obvious exposure pathway to be considered.

#### Dermal absorption

Some fraction of the BE on building surfaces may be transferred to the skin after contact and subsequently absorbed through the skin. The CDI for the dermal absorption pathway was calculated as follows:

$$CDI_{DA} = \frac{SA_d \times SCS \times EVF \times TE \times ABS \times EF \times ED}{BW \times AT} \quad (4)$$

where:  $CDI_{DA}$  = Chronic Daily Intake from dermal absorption (mg/kg-day);  $SA_d$  = Skin surface area assumed to contact building surface per event (cm<sup>2</sup>/event); SCS = Surface contamination cleanup standard (mg/cm<sup>2</sup>); EVF = Event frequency (dermal contact events/day); TE = Contaminant transfer efficiency (unitless); ABS = Fraction of chemical dermally absorbed (unitless); EF = Exposure frequency (days/year); ED = Exposure duration (years); BW = Body weight (kg); and AT = Averaging time (days).

The skin surface,  $SA_d$ , refers to the expected amount of an individual's skin surface assumed available for direct contact with building surfaces. For this analysis the entire palmar surface area of both hands was assumed to be the skin surface

contacting the building or equipment surface. An  $SA_D$  value of  $320\text{ cm}^2$  was assumed as the average area of the entire palmar area of both hands of an adult (Api et al. 2007). The event frequency, EVF, is the number of times per day the individual's hands are assumed to contact the contaminated surface. A value of eight times per day (once per hour) is commonly assumed for this parameter (Michaud et al. 1994). The contaminant transfer efficiency, TE, is the fraction of BE on the building surface assumed to transfer to the hand with each contact event. Brouwer et al. (1999) showed that the amount of contaminant transferred to the skin from a surface is less than 2%. Nonetheless, to be conservative, a value of 10% was assumed. ABS is the fraction of BE dermally absorbed once it has adhered to the hand. No study documenting the dermal absorption of BE could be identified in the literature. However, the dermal absorption of metals is uniformly very low. For example, the California Department of Toxic Substances Control (DTSC 1994) assumes a default dermal absorption value of only 1% for most inorganics for purposes of health risk assessment. A value of 1% dermal absorption was therefore assumed.

### Dermal contact followed by ingestion

Individuals may inadvertently ingest BE when the BE is incidentally transferred from the skin of the hands to the mouth. The CDI for this pathway was calculated as follows:

$$CDI_{ing} = \frac{SAF \times SA_D \times SCS \times EVF \times TE \times BA \times EF \times ED}{BW \times AT} \quad (5)$$

where:  $CDI_{ing}$  = Chronic Daily Intake from incidental ingestion (mg/kg-day); SAF = Fraction of hand surface assumed available for transferring BE to the mouth (unitless);  $SA_D$  = Skin surface area assumed to contact building surface per event ( $\text{cm}^2/\text{event}$ ); SCS = Surface contamination cleanup standard ( $\text{mg}/\text{cm}^2$ ); EVF = Event frequency (dermal contact events/day); TE = Contaminant transfer efficiency (unitless); BA = Gastrointestinal bioavailability of BE (unitless); EF = Exposure frequency (days/year); ED = Exposure duration (years); BW = Body weight (kg); and AT = Averaging time (days).

SAF, the fraction of the palmar surface of the hands available for transferring BE to the mouth, was assumed to be 10% of  $320\text{ cm}^2$ , or  $32\text{ cm}^2$ , which is close to the value assumed by Michaud et al. (1994) for the surface area of the fingertips. BA, the gastrointestinal bioavailability of BE, was conservatively assumed to be 100% or 1.0 in the absence of published data regarding this parameter.

### Inhalation

As in the case of the cancer-risk based SCS, the inhalation exposure pathway is also relevant for non-cancer health risks. However, for non-cancer risks the equation used to calculate the inhalation CDI differs from that used to calculate the CDI for cancer risks. Specifically, the averaging time (AT) parameter changes from 70 years (25,550 days) to 25 years

(9,125 days) in the non-cancer CDI equation (USEPA 2004). The CDI associated with this pathway was calculated as described above with the alternate value of AT used.

All exposure parameters discussed above are summarized in Table 1. Non-cancer risk is considered to be negligible if the Hazard Quotient (HQ) for a chemical, defined as the ratio of the CDI to the USEPA RfD:

$$HQ = \frac{CDI}{RfD} \quad (6)$$

is less than or equal to 1.

For the three exposure pathways included in the non-cancer risk SCS, this equation expands to:

$$HQ = \frac{CDI_{DA} + CDI_{ing}}{RfD_{ing}} + \frac{CDI_{inh}}{RfD_{inh}} \quad (7)$$

Setting HQ equal to 1, using the USEPA ingestion RfD of  $2\text{E-}03\text{ mg}/\text{kg}\text{-day}$  and the inhalation RfD of  $5.72\text{E-}06\text{ mg}/\text{kg}\text{-day}$ , and solving for SCS using iterative methods results in an SCS of  $0.07\text{ }\mu\text{g}/100\text{ cm}^2$ . Note that USEPA has not developed RfDs for the dermal exposure route and it is standard risk assessment practice to use the ingestion route RfD for the dermal route.

### Discussion

Health risk-based SCSs for BE were developed to facilitate the safe transfer of property (equipment and buildings) previously used in BE-related processes. For the cancer endpoint, the analysis resulted in a SCS of  $17\text{ }\mu\text{g}/100\text{ cm}^2$  based on resuspension of settled dust and subsequent inhalation exposure only. This value is well above the typical limits of detection for BE in  $100\text{ cm}^2$  wipe samples ( $0.05\text{--}0.005\text{ }\mu\text{g}$ ) (Day et al. 2007; Emond et al. 2007), as well as levels of BE typically found in the non-production areas of BE-related facilities ( $\sim 0.05\text{ }\mu\text{g}/100\text{ cm}^2$ ). It is also well above the current DOE release criteria of  $0.2\text{ }\mu\text{g}/100\text{ cm}^2$ .

An SCS was also developed based on non-cancer health risks. This SCS calculation included the dermal absorption, incidental ingestion (following dermal contact), and inhalation exposure pathways. This calculation resulted in a SCS of  $0.07\text{ }\mu\text{g}/100\text{ cm}^2$ . This value is just slightly above levels of BE typically found in the non-production areas of BE-related facilities, but is still well within the range of achievable detection limits. The non-cancer-based SCS is below the cancer-based SCS due to the significant contribution of the dermal absorption pathway to exposure. This pathway is not relevant to the cancer-based SCS because BE is not regulated as carcinogenic via the dermal pathway at this time. Consistent with standard risk assessment practice the lower of the cancer or non-cancer-based standards should be used in practice. Therefore, it is recommended that BE monitoring and control be conducted using the non-cancer based SCS of  $0.07\text{ }\mu\text{g}/100\text{ cm}^2$ . Application of this standard will adequately protect workers from both cancer and non-cancer health risks.

**Table 1.** Exposure Parameters Used to Develop the Beryllium Surface Contamination Cleanup Standard

Exposure Parameters	Symbol	Value	Units	Reference
<b>General Exposure Parameters</b>				
Adult body weight	BW	70	kg	USEPA (2004)
Averaging time for cancer risk	AT	25,550	days	USEPA (2004)
Averaging time for non-cancer risk	AT	9,125	days	USEPA (2004)
Exposure frequency for adult worker	EF	250	days/year	USEPA (2004)
Exposure duration for adult worker	ED	25	years	USEPA (2004)
<b>Dermal Absorption</b>				
Hand surface area contacting building surfaces per contact event	SA <sub>D</sub>	320	cm <sup>2</sup> /event	Api et al. (2007)
Number of times hands contact building surfaces per day	EVF	8	events/day	See text
Contaminant transfer efficiency (building surfaces to hands)	TE	0.1	unitless	See text
Dermal absorption efficiency of beryllium	ABS	0.01	unitless	DTSC (1994)
<b>Incidental Ingestion Following Dermal Contact</b>				
Fraction of hand skin surface area contributing to incidental ingestion	SAF	0.1	unitless	See text
Gastrointestinal bioavailability	BA	1.0	unitless	See text
<b>Inhalation</b>				
Resuspension factor	RF	1.00E-08	cm <sup>-1</sup>	Abu-Eid et al. (2002)
Inhalation rate for adult worker	InhR	20	m <sup>3</sup> /day	USEPA (2004)
Conversion factor	CF	1.00E+06	cm <sup>3</sup> /m <sup>3</sup>	

It is important to note a key uncertainty associated with the SCS calculations described here. Of all the exposure parameters used to calculate the SCS values, by far the greatest uncertainty is associated with the resuspension factor (RF). RFs are factors for converting a contaminant level on a surface (typically expressed in units of  $\mu\text{g}/100\text{cm}^2$ ) to a corresponding air concentration. They are empirically derived parameters and can vary by orders of magnitude. Historically, RFs have been used most commonly for radiologic dose assessment and occupational health protection in the nuclear industry. Use of the RF approach is in fact the standard method used by the Nuclear Regulatory Commission (NRC) for converting a surface contaminant concentration to an air concentration (Abu-Eid et al. 2002). Resuspension of a chemical contaminant depends on numerous empirical factors including contamination levels, room activity levels, humidity, and ventilation. The numerous interactive effects of these variables on resuspension are difficult to capture in the available limited studies. Additional studies are needed to develop a more robust RF value for BE and add additional certainty to these SCS estimates.

With regard to the non-cancer SCS, by far the most important exposure pathway is the dermal exposure pathway, with both the incidental ingestion and inhalation exposure pathways being negligible. BE control and monitoring efforts should therefore be focused on methods for preventing dermal contact with BE contaminated surfaces.

## Conclusions

Quantitative health risk assessment methods were used to develop an occupational SCS for BE that explicitly considers the relevant exposure pathways and toxicity endpoints, including both cancer and non-cancer endpoints. For the cancer endpoint at the 1E-06 risk level, the analysis resulted in an SCS of 17  $\mu\text{g}/100\text{cm}^2$  based on resuspension of settled dust and subsequent inhalation exposure only. For the non-cancer

endpoint, the analysis resulted in a SCS of 0.07  $\mu\text{g}/100\text{cm}^2$  based on dermal absorption, incidental ingestion following dermal contact, and inhalation. The non-cancer SCS was determined virtually entirely by the dermal absorption exposure pathway, with negligible contributions from the incidental ingestion and inhalation pathways. This analysis shows that application of the non-cancer SCS will adequately protect workers from both cancer and non-cancer health effects of BE. A key uncertainty in the development of any SCS, regardless of chemical, is the RF. This parameter is critical for estimating an airborne concentration from surface contamination and, hence, correctly estimating inhalation exposure. Additional studies are needed to develop more robust, chemical-specific estimates of RF to reduce uncertainty when developing health risk-based surface cleanup standards.

## Declaration of interest

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