INITIAL STATEMENT OF REASONS
TITLE 27, CALIFORNIA CODE OF REGULATIONS

PROPOSED AMENDMENT TO:
SECTION 25705(b) SPECIFIC REGULATORY LEVELS
POSING NO SIGNIFICANT RISK

ANTIMONY TRIOXIDE

SAFE DRINKING WATER AND TOXIC ENFORCEMENT ACT OF 1986
PROPOSITION 65

PURPOSE AND BACKGROUND OF PROPOSED AMENDMENT

This proposed regulatory amendment would adopt a No Significant Risk Level (NSRL) for antimony trioxide under Proposition 65[1] in Title 27, California Code of Regulations, section 25705(b)[2]. The proposed NSRL of 0.13 micrograms per day (µg/day) for antimony trioxide is based on a carcinogenicity study in rodents and was derived using the methods described in Section 25703.

Proposition 65 was enacted as a ballot initiative on November 4, 1986. The Office of Environmental Health Hazard Assessment (OEHHA) within the California Environmental Protection Agency is the lead state entity responsible for the implementation of Proposition 65[3]. OEHHA has the authority to adopt and amend regulations to implement and further the purposes of the Act[4].

The Act requires businesses to provide a warning when they cause an exposure to a chemical listed as known to the state to cause cancer or reproductive toxicity. The Act also prohibits the discharge of listed chemicals into sources of drinking water. Warnings

[1] The Safe Drinking Water and Toxic Enforcement Act of 1986, codified at Health and Safety Code section 25249.5 et. seq., commonly known as Proposition 65, hereafter referred to as “Proposition 65” or “the Act”.

[2] All further regulatory references are to sections of Title 27 of the Cal. Code of Regs., unless otherwise indicated.

[3] Section 25102(o).

[4] Health and Safety Code, section 25249.12(a).

are not required, and the discharge prohibition does not apply when exposures are insignificant. NSRLs provide guidance for determining when a warning is required for exposures to chemicals listed as causing cancer.

Antimony oxide (antimony trioxide) was listed as known to the state to cause cancer under Proposition 65 on October 1, 1990[5].

DEVELOPMENT OF PROPOSED NSRL

To develop the proposed NSRL for antimony trioxide, OEHHA relied on the 2017 National Toxicology Program (NTP) technical report entitled "Toxicology and Carcinogenesis Studies of Antimony Trioxide (CAS No. 1309-64-4) in Wistar Han [Crl:WI (Han)] Rats and B6C3F$_1$/N Mice (Inhalation Studies)"[6] and the NTP Report on Carcinogens "Monograph on Antimony Trioxide"[7]. The NTP technical report and the NTP Report on Carcinogens (RoC) monograph summarize the available data from rodent carcinogenicity studies, as well as other information relevant to the carcinogenic activity of antimony trioxide.

The NSRL for antimony trioxide is based upon the results of the most sensitive scientific study deemed to be of sufficient quality[8].

Selection of Studies Used to Determine Cancer Potency

OEHHA reviewed the available data from the rodent carcinogenicity studies of antimony trioxide and determined that the two-year inhalation studies conducted by NTP[9] in male and female B6C3F$_1$/N mice met the criterion in Section 25703 as being sensitive studies of sufficient quality.

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[5] https://oehha.ca.gov/proposition-65/proposition-65-list

[6] National Toxicology Program (NTP 2017). Toxicology and Carcinogenesis Studies of Antimony Trioxide (CAS No. 1309-64-4) in Wistar Han [Crl:WI (Han)] Rats and B6C3F1/N Mice (Inhalation Studies). NTP Technical Report Series No. 590. US Department of Health and Human Services, NTP, Research Triangle Park, NC. Available from https://ntp.niehs.nih.gov/go/tr590.

[7] National Toxicology Program (NTP 2018). Report on Carcinogens Monograph on Antimony Trioxide. RoC Monograph 13. US Department of Health and Human Services, NTP, Research Triangle Park, NC. Available from https://ntp.niehs.nih.gov/ntp/roc/monographs/antimony_final20181019_508.pdf.

[8] Section 25703(a)(4).

[9] NTP (2017), full citation provided in footnote 6.

In the NTP studies[10], groups of 60 mice of each sex were exposed to antimony trioxide aerosol by inhalation at concentrations of 0, 3, 10, or 30 milligrams per cubic meter (mg/m^3), 6 hours plus 12 minutes per day, 5 days per week for up to 105 weeks. Ten male and ten female mice from each group were selected for interim evaluation at 12 months. The lifetime average daily doses of antimony trioxide administered in the studies were calculated to be 0, 0.62, 2.07 and 6.21 milligrams per kilogram of body weight per day (mg/kg-day) in male mice, and to be 0, 0.63, 2.12 and 6.35 mg/kg-day in female mice. Survival of the mid- and high-dose male and female mice was significantly less than that of their respective chamber control groups[11].

In male mice, a statistically significant increase in the incidence of lung alveolar/bronchiolar carcinomas was observed, with a statistically significant positive trend. In addition, a small, treatment-related, and statistically significant increase in the incidence of fibrous histiocytoma or fibrosarcoma (combined) in the skin was observed (0/50, 1/50, 3/50, 4/50). The tumor incidence data used to estimate cancer potency from this study in male mice are presented in Table 1.

Table 1. Lung tumor incidences[a] of treatment-related lesions in male $B6C3F_1$/N mice administered antimony trioxide by inhalation for up to 2 years (NTP 2017)

Organ	Tumor Type	Administered Concentrations (mg/m^3)				Trend test *p*-value[b]
		0	3	10	30	
Lung	Alveolar/bronchiolar carcinoma[c] (first occurrence of tumor: day 367[d])	4/60	18/58***	21/58***	29/60***	$p < 0.001$

[a] The numerator represents the number of tumor-bearing animals and the denominator represents the number of animals alive at the time of first occurrence of tumor.
[b] *p*-values for exact trend test conducted by OEHHA.
[c] Treatment group tumor incidences with asterisks indicate significant results from Fisher pairwise comparison with controls (performed by OEHHA): *** $p < 0.001$
[d] Since tumors were observed on day 367 in the interim sacrifice group, day 367 is assumed to be the first day of tumor (effective number)

[10] NTP (2017), full citation provided in footnote 6.

[11] *Ibid*.

In female mice, statistically significant increases in incidences of lung alveolar/bronchiolar adenoma, carcinoma, and alveolar/bronchiolar adenoma and carcinoma (combined) were observed, with statistically significant positive trends. A statistically significant increase in the incidence of malignant lymphoma was observed, with a statistically significant positive trend. The tumor incidence data used to estimate cancer potency from this study in female mice are presented in Table 2.

Table 2. Tumor incidences[a] of treatment-related lesions in female B6C3F$_1$/N mice administered antimony trioxide by inhalation for up to 2 years (NTP 2017)

Organ	Tumor Type	Administered Concentrations (mg/m^3)				Trend test *p*-value[b]
		0	3	10	30	
Lung	Alveolar/bronchiolar adenoma or carcinoma[c] (first occurrence of tumor: 367)	3/47	22/46***	27/44***	19/44***	$p = 0.018$
All organs	Malignant lymphoma[c] (first occurrence of tumor: day 367)	7/46	17/46*	20/43**	30/48***	$p < 0.001$

[a] The numerator represents the number of tumor-bearing animals. The denominator has been adjusted with the poly-3 method to account for intercurrent mortality during the 105-week study.
[b] *p*-values for exact trend test conducted by OEHHA.
[c] Treatment group tumor incidences with asterisks indicate significant results from Fisher pairwise comparison with controls (performed by OEHHA): * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Model Used to Estimate Cancer Potency

The NTP Report on Carcinogens Monograph on Antimony Trioxide[12] reviewed the mechanistic data for antimony trioxide and concluded:

> "In summary, based on studies using antimony(III) trioxide and other antimony(III) compounds, antimony(III) trioxide is electrophilic, can cause oxidative stress, likely inhibits DNA repair, can cause oxidative damage, and is likely to decrease cell differentiation. These effects can contribute to carcinogenesis, and all are biologically plausible in humans."

Regarding genotoxicity, NTP[13] summarized:

[12] NTP (2018), full citation provided in footnote 7.

[13] *Ibid*.

> "(1) antimony(III) trioxide and other antimony(III) compounds are not mutagenic in bacterial or mammalian cells, (2) antimony(III) trioxide can cause DNA damage in mouse lung *in vivo* after long-term inhalation exposure, and (3) antimony(III) trioxide can cause chromosomal aberrations *in vitro*, micronucleus formation *in vivo*, and SCE *in vitro*."

As noted above, it appears that the carcinogenicity of antimony trioxide may be the result of multiple mechanisms of action, including several types of both genotoxic and non-genotoxic processes. A multistage model was used to derive cancer potency estimates from the male and female mouse NTP studies. The data from the female mouse study was modeled using poly-3 corrected incidences to account for differences in mortality that were significant but not large between treated groups and the control group. A time-to-tumor extension of the multistage model was used to derive a cancer potency estimate from the male mouse NTP study following the guidance in Section 25703. There are no specific mechanistic data to suggest any deviation from the standard assumptions, including low-dose linearity, usually applied in cancer dose-response analysis. There are no principles or assumptions scientifically more appropriate, based on the available data, than the approach to the dose-response assessment of these studies described above, i.e., application of the multistage model and the time-to-tumor extension of that model.

The lifetime probability of a tumor at a specific site given exposure to the chemical at dose d is modeled using the multistage polynomial:

$$p(d) = \beta_0 + (1 - \beta_0)\left(1 - \exp\left[-\left(\beta_1 d + \beta_2 d^2 + \cdots + \beta_j d^j\right)\right]\right)$$

where the background probability of tumor, β_0, is between 0 and 1 and the coefficients β_i, i = 1…j, are positive. The β_i are parameters of the model, which are taken to be constants and are estimated from the data. The parameter β_0 provides the basis for estimating the background lifetime probability of the tumor.

To derive a measure of the cancer response to antimony trioxide (per mg/kg-day) in the studies described above, the dose associated with a 5% increased risk of developing a tumor was calculated and the lower bound for this dose was estimated using the multistage polynomial model for cancer in US EPA's Benchmark Dose Software

(BMDS)[14]. The multistage model is the default approach to modeling lifetime cancer bioassay data, as stated in US EPA's 2005 cancer risk assessment guidelines[15].

For carcinogens that induce tumors at multiple sites and/or in different cell types at the same site in a particular species and sex, US EPA's BMDS[16] can be used to derive maximum likelihood estimates (MLEs) for the parameters of the multisite carcinogenicity model by summing the MLEs for the individual multistage models for the different sites and/or cell types. This multisite model provides a basis for estimating the cumulative risk of carcinogen treatment-related tumors. In order to derive a measure of the total cancer response in a given study, the dose associated with a 5% increased risk of developing a tumor at one or more of the sites of interest was calculated and the lower bound for this dose was estimated using the multisite model in BMDS. The ratio of the 5% risk level to that lower bound on dose is known as the multisite "animal cancer slope factor (CSF_{animal})," or "animal cancer potency."

To account for the treatment-related intercurrent mortality observed in the female mouse study, the poly-3 method was used to adjust the denominator (N) of tumor (i.e., lung alveolar/bronchiolar adenoma or carcinoma or malignant lymphoma) incidence as shown in Table 2. The differential mortality was accounted for by assigning a reduced contribution towards N, proportional to the third power of the fraction of time on study, only to animals without the respective treatment-related tumors that died before terminal sacrifice (day 732 on study)[17]. The equation is shown below:

$$\text{Contribution to N} = \left(\frac{\text{Days on study}}{\text{732 days}}\right)^3$$

Animal cancer potencies were estimated using this approach for the female mouse inhalation study described in Table 2.

When a large fraction of the animals dies before the end of the study, as occurred in the male mouse study, the multistage-in-dose Weibull-in-time (multistage Weibull) model can be used to estimate the cancer potency. The multistage Weibull model is an

[14] US EPA Benchmark Dose Software (BMDS) Version 3.2. National Center for Environmental Assessment, US EPA. Available from: https://www.epa.gov/bmds.

[15] US EPA (2005). US Environmental Protection Agency. Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum. Washington, DC. EPA/630/P-03/001B. March 2005.

[16] US EPA BMDS, full citation provided in footnote 14.

[17] Bailer AJ and Portier CJ (1988). Effects of treatment-induced mortality and tumor-induced mortality on test for carcinogenicity in small samples. Biometrics 44(2):417-431.

extension of the multistage polynomial model given above, with the probability of a tumor at a specific site by time t and given exposure to the chemical at dose d given as:

$$p(t,d) = 1 - \exp[-(\beta_0 + \beta_1 d + \beta_2 d^2 + ... + \beta_j d^j)(t - t_0)^c]$$

where the coefficients β_i, i = 0…j, are positive, $0 \leq t_0 < t$, where t_0 is commonly interpreted as the latency period[18], and the age exponent, c, is restricted to be between 0 and 10. Carcinogenic potency for a given site is derived by applying a maximum likelihood modeling approach to estimate the model parameters (β_i, t_0, and c). Using the multistage Weibull model, the CSF_{animal} is defined as the upper 95% confidence bound on β_1 estimated at the assumed standard lifetime of 104 weeks for mice.

Calculation of Average Daily Doses

The lifetime average dose in units of mg/kg-day of antimony trioxide was calculated for each dose group, based on the dose level, duration, exposure regimen, and animal body weights reported by NTP[19]. The average body weights for male and female mice were calculated to be 0.0466 kg and 0.0436 kg, respectively, from the data reported by NTP[20] for control animals.

The inhalation rates (IR), in m^3/day, for male and female mice were calculated based on the equation of Anderson et al. (1983)[21], which was derived using experimental data on animal breathing rates (m^3/day) and corresponding body weights (kg):

$$IR_{mice} = 0.0345 \text{ m}^3\text{/day} \times (bw_{mice}/0.025 \text{ kg})^{2/3}$$

The calculated inhalation rates were 0.052 m^3/day for male mice and 0.050 m^3/day for female mice. Average doses (D_{avg}) were determined by multiplying the chamber air concentration (C_{air}) of antimony trioxide in units of mg/m^3 by the following factors: the inhalation rate divided by the body weight; 6.2/24 to account for the six hours and 12

[18] When all tumors at a given site are considered incidental, as was the case with the lung tumors and malignant lymphoma observed in the male mouse study, t_0 is not estimated and the probability of tumor (p(t,d)) by time t and lifetime dose rate d is given as: $p(t,d) = 1 - \exp[-(\beta_0 + \beta_1 d + \beta_2 d^2 + ... + \beta_j d^j)t^c]$

[19] NTP (2017), full citation provided in footnote 6.

[20] *Ibid.*

[21] Anderson EL and the Carcinogen Assessment Group of the US EPA (1983). Quantitative approaches in use to assess cancer risk. *Risk Analysis* 3:277-295.

minutes per day exposure; 5/7 to account for a five day per week dosing regimen. The equation for lifetime average dose (mg/kg-day) calculation is:

$$D_{avg} = C_{air}\left(^{mg}/_{m^3}\right) \times \frac{IR_{mice}}{bw_{mice}} \frac{\left(^{m^3}/_{day}\right)}{kg} \times \frac{6.2}{24} \times \frac{5}{7}$$

Thus, the lifetime average doses were calculated to be 0.62, 2.07, and 6.21 mg/kg-day for the low-, mid-, and high-dose groups in male mice and 0.63, 2.12, and 6.35 mg/kg-day for the low-, mid-, and high-dose groups in female mice.

Estimation of Human Cancer Potency

Human cancer potency is estimated by an interspecies scaling procedure. According to Section 25703(a)(6), dose in units of mg per kg body weight scaled to the three-quarters power is assumed to produce the same degree of effect in different species in the absence of information indicating otherwise. Thus, for each of the studies described above, scaling to the estimated human potency (CSF_{human}) is achieved by multiplying the animal potency (CSF_{animal}) by the ratio of human to animal body weights (bw_{human}/bw_{animal}) raised to the one-fourth power when CSF_{animal} is expressed in units $(mg/kg\text{-}day)^{-1}$:

$$CSF_{human} = CSF_{animal} \times (bw_{human} / bw_{animal})^{1/4}$$

The default human body weight is 70 kg. The average body weights for male and female mice were calculated to be 0.0466 kg and 0.0436 kg, respectively, based on the data reported for control animals by NTP (2017)[22]. The derivations of the human cancer slope factors using these body weights are summarized below in Table 3.

[22] NTP (2017), full citation provided in footnote 6.

Table 3. Derivation of CSF_{human} using mean animal body weights for the studies and data presented in Tables 1 and 2

Sex/ Strain/ Species	Type of neoplasm	Body Weight (kg)	CSF_{animal} $(mg/kg\text{-}day)^{-1}$	CSF_{human} $(mg/kg\text{-}day)^{-1}$
Male B6C3F1/N mice[a]	Lung alveolar/bronchiolar carcinoma	0.0466	0.0641	0.4
Female B6C3F1/N mice[b]	Lung alveolar/bronchiolar adenoma or carcinoma[c]	0.0436	0.680	Not calculated
	Malignant lymphoma		0.188	
	Multisite: Lung alveolar/bronchiolar adenoma or carcinoma and malignant lymphoma		0.818	**5.2**

[a] The multistage Weibull model was used for analyses of male mice.
[b] The linearized multistage model was used for analyses of female mice, with a poly-3 correction.
[c] The top dose group had to be removed during the modeling process to achieve sufficient goodness of fit.

As shown in Table 3, female mice were the most sensitive to the carcinogenic effects of antimony trioxide and thus the NSRL for antimony trioxide will be based on the human cancer slope factor of 5.2 $(mg/kg\text{-}day)^{-1}$, derived from the study in female mice.

Calculation of No Significant Risk Levels

The NSRL can be calculated from the cancer slope factor as follows. The Proposition 65 no-significant-risk value is one excess case of cancer per 100,000 people exposed, expressed as 10^{-5}. This value is divided by the slope factor, expressed in units of one divided by milligram per kilogram body weight per day. The result of the calculation is a dose level associated with a 10^{-5} risk in units of mg/kg-day. This dose then can be converted to an intake amount in units of mg per day by multiplying by the body weight for humans. When the calculation is for the general population, the body weight is

assumed to be 70 kg[23]. The intake can be converted to a µg per day amount by multiplying by 1000. This sequence of calculations can be expressed mathematically as:

$$\text{NSRL} = \frac{10^{-5} \times 70\ \text{kg}}{\text{CSFhuman}} \times 1000\ \mu\text{g/mg}$$

As indicated previously, the human cancer slope factor for antimony trioxide derived from the female mouse study data and exposure parameters presented in Table 2 is 5.2 per mg/kg-day. Inserting this number into the equation above results in an NSRL of 0.13 µg/day.

PROPOSED REGULATORY AMENDMENT

Section 25705(b)

The proposed change to Section 25705(b) is provided below, in underline.

> (1) The following levels based on risk assessments conducted or reviewed by the lead agency shall be deemed to pose no significant risk:

Chemical name	Level (micrograms per day)
Acrylonitrile	0.7
…	
<u>Antimony oxide (Antimony trioxide)</u>	<u>0.13</u>
…	

PROBLEM BEING ADDRESSED BY THIS PROPOSED RULEMAKING

Proposition 65 does not provide guidance regarding how to determine whether a warning is required or a discharge is prohibited. OEHHA is the implementing agency for Proposition 65 and has the resources and expertise to examine the scientific literature and calculate a level of exposure, in this case an NSRL, under which a warning is not required, and under which a discharge is not prohibited.

[23] Section 25703(a)(8).

ECONOMIC IMPACT ASSESSMENT (see below)

NECESSITY

This proposed regulatory amendment would adopt an NSRL that conforms with the Proposition 65 implementing regulations and reflects the currently available scientific knowledge about antimony trioxide. The NSRL provides assurance to the regulated community that exposures or discharges at or below this level are considered not to pose a significant risk of cancer. This regulation is needed to convey that information to the public and the regulated population. Exposures at or below the NSRL are exempt from the warning and discharge requirements of Proposition 65[24].

BENEFITS OF THE PROPOSED REGULATION

See "Benefits of the Proposed Regulation" under ECONOMIC IMPACT ANALYSIS below.

TECHNICAL, THEORETICAL, AND/OR EMPIRICAL STUDIES, REPORTS, OR DOCUMENTS

The following documents were relied on by OEHHA for calculating the NSRL for antimony trioxide.

- The 2017 NTP report entitled "Toxicology and Carcinogenesis Studies of Antimony Trioxide (CAS No. 1309-64-4) in Wistar Han [Crl:WI (Han)] Rats and B6C3F1/N Mice (Inhalation Studies)."[25].
- The NTP Report on Carcinogens "Monograph on Antimony Trioxide"[26]
- The publication by Anderson *et al.* (1983)[27].

[24] Health and Safety Code sections 25249.9(b) and 25249.10(c).

[25] National Toxicology Program (NTP 2017). Toxicology and Carcinogenesis Studies of Antimony Trioxide (CAS No. 1309-64-4) in Wistar Han [Crl:WI (Han)] Rats and B6C3F1/N Mice (Inhalation Studies). NTP Technical Report Series No. 590. US Department of Health and Human Services, NTP, Research Triangle Park, NC. Available from https://ntp.niehs.nih.gov/go/tr590.

[26] National Toxicology Program (NTP 2018). Report on Carcinogens Monograph on Antimony Trioxide. RoC Monograph 13. US Department of Health and Human Services, NTP, Research Triangle Park, NC. Available from https://ntp.niehs.nih.gov/ntp/roc/monographs/antimony_final20181019_508.pdf.

[27] Anderson EL and the Carcinogen Assessment Group of the US EPA (1983). Quantitative approaches in use to assess cancer risk. Risk Analysis 3:277-295.

Copies of these documents will be included in the regulatory record for this proposed action. These documents are available from OEHHA upon request.

OEHHA also relied on the following Economic Impact Analysis, included in this document, in developing this proposed regulation.

REASONABLE ALTERNATIVES TO THE REGULATION AND THE AGENCY'S REASONS FOR REJECTING THOSE ALTERNATIVES

The NSRL provides a "safe harbor" value that aids businesses in determining if they are complying with the law. The alternative to the proposed amendment to Section 25705(b) would be to not adopt an NSRL for the chemical. Failure to adopt an NSRL would leave the business community without a "safe harbor" level to assist businesses in complying with Proposition 65. No alternative that is less burdensome yet equally as effective in achieving the purposes of the regulation in a manner that achieves the purposes of the statute has been proposed.

REASONABLE ALTERNATIVES TO THE PROPOSED REGULATORY ACTION THAT WOULD LESSEN ANY ADVERSE IMPACT ON SMALL BUSINESSES

OEHHA is not aware of significant cost impacts that small businesses would incur in reasonable compliance with the proposed action. Use of the proposed NSRL by businesses is voluntary and therefore does not impose any costs on small businesses. In addition, Proposition 65 is limited by its terms to businesses with 10 or more employees (Health and Safety Code, section 25249.11(b)) so it has no effect on very small businesses.

EVIDENCE SUPPORTING FINDING OF NO SIGNIFICANT ADVERSE ECONOMIC IMPACT ON BUSINESS

Because the proposed NSRL provides a "safe harbor" level for businesses to use when determining compliance with Proposition 65, OEHHA does not anticipate that the regulation will have a significant statewide adverse economic impact directly affecting businesses, including the ability of California businesses to compete with businesses in other states.

EFFORTS TO AVOID UNNECESSARY DUPLICATION OR CONFLICTS WITH FEDERAL REGULATIONS CONTAINED IN THE CODE OF FEDERAL REGULATIONS

Proposition 65 is a California law that has no federal counterpart. There are no federal regulations addressing the same issues and, thus, there is no duplication or conflict with federal regulations.

ECONOMIC IMPACT ANALYSIS
Gov. Code section 11346.3(b)

It is not possible to quantify specific monetary values for this proposed regulatory action given that use of the NSRL is entirely voluntary and the NSRL only provides compliance assistance for businesses subject to the Act.

Impact on the Creation or Elimination of Jobs/Businesses in California: This regulatory proposal will not affect the creation or elimination of jobs within the State of California. Proposition 65 requires businesses with ten or more employees to provide warnings when they expose people to chemicals that are known to cause cancer or developmental or reproductive harm. The law also prohibits the discharge of listed chemicals into sources of drinking water. Antimony trioxide is listed under Proposition 65; therefore, businesses that manufacture, distribute, sell or use products with antimony trioxide in the state must provide a warning if their product or activity exposes the public or employees to significant amounts of the chemical. The regulatory proposal does not create additional compliance requirements, but instead provides "safe harbor" values that aid businesses in determining whether a warning is required for a given exposure.

Impact on the Creation of New Businesses or Elimination of Existing Businesses within the State of California: This regulatory action will not impact the creation of new businesses or the elimination of existing businesses within the State of California. The regulatory proposal does not create additional compliance requirements, but instead provides a "safe harbor" value that aids businesses in determining if they are complying with the law.

Impact on Expansion of Businesses within the State of California: This regulatory action will not impact the expansion of businesses within the State of California. The regulatory proposal does not create additional compliance requirements, but instead provides a "safe harbor" value that aids businesses in determining if they are complying with the law.

Benefits of the Proposed Regulation: Currently, businesses may be subject to litigation for failure to warn of an exposure to antimony trioxide or for causing a prohibited discharge of the listed chemical. The NSRL provides a "safe harbor" value that aids businesses in determining if they are complying with the law. Adopting this regulation will save these businesses those expenses and may reduce litigation costs. By providing a safe harbor level, this regulatory proposal does not require, but may

encourage, businesses to lower the amount of the listed chemical in their products or releases from their facilities to a level that does not cause a significant exposure, thereby providing a public health benefit to Californians.