FINAL STATEMENT OF REASONS
TITLE 27, CALIFORNIA CODE OF REGULATIONS

SECTION 25705(b) SPECIFIC REGULATORY LEVELS POSING NO SIGNIFICANT RISK

NO SIGNIFICANT RISK LEVEL: TRICHLOROACETIC ACID

This is the Final Statement of Reasons (FSOR) for the adoption of a No Significant Risk Level (NSRL) for trichloroacetic acid. On September 13, 2013, trichloroacetic acid was listed for purposes of Proposition 65[1] as a chemical known to the state to cause cancer. On May 22, 2020, the Office of Environmental Health Hazard Assessment (OEHHA) issued a Notice of Proposed Rulemaking to adopt a proposed amendment to Title 27, California Code of Regulations, section 25705(b)[2]. Specific Regulatory Levels Posing No Significant Risk, identifying an NSRL of 9.9 micrograms per day (µg/day) for trichloroacetic acid. The Initial Statement of Reasons (ISOR) sets forth the grounds for the amendment to the regulation.

SUMMARY

In developing the NSRL for trichloroacetic acid, OEHHA relied on two studies by DeAngelo et al. (2008)[3,4], a study by Bull et al. (2002)[5], Volume 106 in the series of International Agency for Research on Cancer (IARC) Monographs on the Evaluation of Carcinogenic Risks to Humans, entitled “Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents”[6], the National Toxicology Program (NTP) report entitled “Toxicology Studies of Bromodichloroacetic Acid (CAS No. 71133-14-7) in F344/N Rats and B6C3F1/N Mice and Toxicology and Carcinogenesis Studies of Bromodichloroacetic Acid in F344/NTac Rats and B6C3F1/N Mice (Drinking Water

[1] The Safe Drinking Water and Toxic Enforcement Act of 1986, codified at Health and Safety Code section 25249.5 *et. seq*., 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] DeAngelo AB, Daniel FB, Wong DM, George MH (2008). The induction of hepatocellular neoplasia by trichloroacetic acid administered in the drinking water of the male B6C3F1 mouse. J Toxicol Environ Health A 71(16):1056-68.
[4] Individual animal survival and tumor data provided by the study authors were obtained from the US EPA in August 2016 (104-week study) and January 2017 (60-week study).
[5] Bull RJ, Orner GA, Cheng RS, Stillwell L, Stauber AJ, Sasser LB, Lingohr MK, Thrall BD (2002). Contribution of dichloroacetate and trichloroacetate to liver tumor induction in mice by trichloroethylene. Toxicol Appl Pharmacol 182(1):55-65.
[6] International Agency for Research on Cancer (IARC 2014). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 106, Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents. IARC, World Health Organization, Lyon, France. Available from: https://publications.iarc.fr/130

Studies)"[7], 11 additional genotoxicity studies[8,9,10,11,12,13,14,15,16,17,18] and two reviews[19,20]. The 2015 NTP report primarily discusses toxicological effects of bromodichloroacetic acid, but also summarizes genotoxicity information on dichloroacetic acid, a metabolite of trichloroacetic acid. The NSRL for trichloroacetic acid is based upon the results of the most sensitive and robust scientific study deemed to be of sufficient quality[21].

PEER REVIEW

OEHHA provided the Notice of Proposed Rulemaking and the ISOR for the proposed NSRL for trichloroacetic acid to the members of the Carcinogen Identification Committee for their review and comment, as required by Section 25701(e). OEHHA

[7] National Toxicology Program (NTP 2015). Toxicology Studies of Bromodichloroacetic Acid (CAS No. 71133-14-7) in F344/N Rats and B6C3F1/N Mice and Toxicology and Carcinogenesis Studies of Bromodichloroacetic Acid in F344/NTac Rats and B6C3F1/N Mice (Drinking Water Studies). NTP Technical Report Series No. 583. US Department of Health and Human Services, NTP, Research Triangle Park, NC.
[8] Anderson KJ, Leighty EG, Takahashi MT (1972). Evaluation of Herbicides for Possible Mutagenic Properties. J. Agric. Food Chem. 20(3), pp 649–656.
[9] Zhang SH, Miao DY, Tan L, Liu AL, Lu WQ (2016). Comparative cytotoxic and genotoxic potential of 13 drinking water disinfection by-products using a microplate-based cytotoxicity assay and a developed SOS/umu assay. Mutagenesis. 31(1):35-41.
[10] Hu Y, Tan L, Zhang SH, Zuo YT, Han X, Liu N, et al. (2017). Detection of genotoxic effects of drinking water disinfection by-products using Vicia faba bioassay. Environ Sci Pollut Res Int. 24(2):1509-1517.
[11] Varshney M, Chandra A, Chauhan LK, Goel SK (2013). Micronucleus induction by oxidative metabolites of trichloroethylene in cultured human peripheral blood lymphocytes: a comparative genotoxicity study. Environ Sci Pollut Res Int. 20:8709-8716.
[12] Varshney M, Chandra A, Chauhan LK, Goel SK (2014). In vitro cytogenetic assessment of trichloroacetic acid in human peripheral blood lymphocytes. Environ Sci Pollut Res Int. 21(2):843-50.
[13] Hassoun E, Cearfoss J, Mamada S, Al-Hassan N, Brown M, Heimberger K, Liu MC (2014). The effects of mixtures of dichloroacetate and trichloroacetate on induction of oxidative stress in livers of mice after subchronic exposure. J Toxicol Environ Health A. 77(6):313-23.
[14] Stalter D, O'Malley E, von Gunten U, Escher BI. (2016). Fingerprinting the reactive toxicity pathways of 50 drinking water disinfection by-products. Water Res 91: 19-30.
[15] Kurinnyĭ A. (1984). Cytogenetic activity of the herbicide sodium trichloroacetate. TSitologiia i genetika 18(4): 318-319.
[16] Zuo YT, Hu Y, Lu WW, et al. (2017). Toxicity of 2,6-dichloro-1,4-benzoquinone and five regulated drinking water disinfection by-products for the Caenorhabditis elegans nematode. J Hazard Mater 321: 456-463.
[17] Ono Y, Somiya I, Kawamura M (1991). The evaluation of genotoxicity using DNA repairing test for chemicals produced in chlorination and ozonation processes. Water Science and technology 23(1-3): 329-338.
[18] Hassoun EA, Dey S (2008). Dichloroacetate- and trichloroacetate-induced phagocytic activation and production of oxidative stress in the hepatic tissues of mice after acute exposure. J Biochem Mol Toxicol 22(1): 27-34.
[19] National Research Council (NRC 1987). Chemistry and toxicity of selected disinfectants and by-products. Drinking water and health: disinfectants and disinfectant by-products 7: 133-143,182-133.
[20] Daniel F, Meier J, Deangelo A. (1993). Advances in research on carcinogenic and genotoxic by-products of chlorine disinfection: chlorinated hydroxyfuranones and chlorinated acetic acids. Annali dell'Istituto superiore di sanita 29(2): 279-291.
[21] Section 25703(a)(4).

received peer-review comments from committee members Jason Bush, PhD, Dana Loomis, PhD, MPH, Thomas Mack, MD, MPH, and Luoping Zhang, PhD.

RESPONSE TO PEER REVIEW COMMENTS

Comment 1: Drs. Loomis, Mack, and Zhang reviewed the materials, and indicated that they did not have any comments.

Response 1: OEHHA acknowledges the responses.

Comment 2: Dr. Bush reviewed the materials, and noted that trichloroacetic acid promotes cell proliferation, which may have an effect on sensitive populations such as pregnant women. He also indicated that he supports the rationale for the proposed NSRL for trichloroacetic acid, and concurs with the calculations and the proposed NSRL.

Response 2: OEHHA acknowledges the comments and that Dr. Bush had no objections to the proposed NSRL. No changes to the proposed regulation were made based on these comments.

PUBLIC COMMENTS

A public comment period was provided from May 22, 2020, to July 7, 2020. OEHHA received written public comments on the proposed rulemaking from the following organizations:

1. Southern California Water Coalition (SCWC)
2. American Chemistry Council's (ACC) Chlorine Chemistry Division (CCD)

RESPONSE TO PUBLIC COMMENTS

A summary of the relevant comments received and OEHHA's responses are provided in this FSOR. Some of the comments submitted included observations or opinions regarding the benefits of chlorine-based disinfection processes and other assessments OEHHA might perform on trichloroacetic acid and other disinfection by-products. Such remarks do not constitute an objection to, or recommendation specifically directed at the proposed action or the procedures followed in this rulemaking action. Accordingly, OEHHA is not required under the Administrative Procedure Act to respond to such comments in this FSOR. Because OEHHA is constrained by limitations upon its time and resources and is not obligated by law to respond to irrelevant comments[22], OEHHA does not provide responses to all of these remarks in this FSOR. However, the absence of responses to such remarks should not be construed to mean that OEHHA in any way agrees with them.

[22] California Government Code section 11346.9(a)(3)

As explained in detail in the responses to comments, OEHHA declines to change the proposed NSRL based on the comments.

Comment 1 (SCWC, ACC): NSRLs should not be based on draft risk assessments still under development in other programs. CCD [ACC's Chlorine Chemistry Division] said they were "troubled by OEHHA's decision to move ahead with NSRLs before the Office has considered the information submitted in response to the PHG [Public Health Goal] proposal and before the science that is the basis for both the PHGs and NSRLs has been subject to peer review". They said the NSRL should not be released until the process for the PHG for haloacetic acids has been completed, and that it is premature and inappropriate for OEHHA to use draft PHG risk assessments to support Proposition 65 NSRLs or any other regulatory decisions until those draft risk assessments are completed. SCWC was concerned that using the draft PHG risk assessments as the basis for enforceable NSRLs would undermine the PHG development process because the proposed NSRLs would create an institutional bias against meaningful changes to the draft PHG risk assessments.

Response 1: The NSRL does not rely on the draft Public Health Goal (PHG). The NSRL has been developed in parallel with the PHG. This process allows for adequate time for the NSRL and the PHG to undergo external peer review and encourages consistency between the two programs within OEHHA. The entire process for the dose-response assessment and development of the NSRL for trichloroacetic acid was conducted in collaboration with the OEHHA program that produces PHGs. Both programs critically evaluated the same key mouse carcinogenicity studies of trichloroacetic acid (DeAngelo et al. 2008[23]; Bull et al. 2002[24]) and used the same data analysis principles, methods, and software to calculate the cancer potencies. After careful consideration by both programs, the 104-week DeAngelo et al. (2008) study was chosen for assessing the carcinogenic effects of trichloroacetic acid, and thus, the human cancer slope factor derived from that study was used as the basis for both the NSRL and the PHG. An assessment by one OEHHA program does not preclude another OEHHA program from making changes to a draft document. The proposed levels for both programs are based on the best available science and have undergone rigorous scientific review.

No changes to the proposed regulation were made based on this comment.

Comment 2 (SCWC, ACC): There is no justification for proposing the NSRLs at this time. Trichloroacetic acid was listed in 2013 and appears to have narrow consumer product applications. There is nothing in the ISOR indicating an increase in consumer

[23] DeAngelo et al. (2008). Full citation provided in footnote 3.
[24] Bull et al. (2002). Full citation provided in footnote 5.

product uses or other applications that would justify the development of an NSRL at this time.

Response 2: OEHHA develops NSRLs for chemicals listed as carcinogens under Proposition 65 as time and resources allow. There are no limits on the time between the date of listing and the development of an NSRL. In recent years, multiple haloacetic acids (HAAs) have been added to the Proposition 65 list and OEHHA has developed NSRLs for each of the five HAAs listed (trichloroacetic acid, dibromoacetic acid, dichloroacetic acid, bromochloroacetic acid, and bromodichloroacetic acid) in order to provide compliance assistance for businesses and guidance for Proposition 65 enforcers.

No changes to the proposed regulation were made based on this comment.

Comment 3 (SCWC): These NSRLs present a potential public health threat because they prioritize reduction of exposure to disinfection by-products (DBPs) over drinking water disinfection. OEHHA should establish alternative Safe Harbor Levels pursuant to Section 25703(b) that allows for such exceptions to the default NSRL.

Response 3: OEHHA followed the guidance in Section 25703(b), which states that "the risk level which represents no significant risk shall be one which is calculated to result in one excess case of cancer in an exposed population of 100,000, assuming lifetime exposure at the level in question, except where sound considerations of public health support an alternate risk level", and gives as one such example "where chlorine disinfection in compliance with all applicable state and federal safety standards is necessary to comply with sanitation requirements".

In developing the NSRL for this carcinogen, OEHHA conducted the evaluation necessary to identify a level that would meet the 1 in 100,000 standard. OEHHA recognizes the public health benefits of the use of chlorine disinfection, and at the same time notes that nothing in Proposition 65 prohibits or places limits on drinking water disinfection. In fact, the statute[25] expressly exempts all agencies of the federal, state, or local government, as well as entities operating public water systems, from the requirements of Proposition 65, including the warning requirement.

Nothing in the analysis for the NSRL prohibits a business from calculating an alternative risk level for this chemical, should the business determine that one is needed.

No changes to the proposed regulation were made based on this comment.

Comment 4 (ACC): The NSRL does not consider the long history of low-level exposure to these substances (i.e., HAAs) and several other DBPs considered to be liver carcinogens by OEHHA (chloroform, bromodichloromethane, and dibromochloromethane). This history reveals a lack of consistent evidence of an

[25] Health and Safety Code section 25249.11(b)

increased incidence of liver cancer resulting from exposure to DBPs in the multiple epidemiology studies that have been conducted.

Response 4: The NSRL for trichloroacetic acid was based on a study conducted in mice because it was deemed to be a sensitive study of sufficient quality, consistent with the requirements described in Section 25703. To our knowledge, no human epidemiological studies of sufficient quality and sensitivity have been published in the scientific literature that would be adequate for conducting a cancer dose-response assessment for trichloroacetic acid. Thus, the 104-week DeAngelo et al. (2008) study in mice in which liver tumors were observed was chosen as the most sensitive and robust study of sufficient quality. Regarding the lack of consistent evidence of an increased incidence of liver cancer in humans, tumor site concordance across species is neither required, nor predicted, for chemical carcinogens. It is a generally accepted principle that although there may be site concordance between humans and animal test species in specific cases, it is not necessarily going to occur. For risk assessment purposes, site concordance is not assumed unless there is evidence to support this assumption[26]. In the absence of data to the contrary, the ability of an agent to induce tumors in animals is considered predictive of the potential for the agent to induce tumors in humans.

No changes to the proposed regulation were made based on this comment.

Comment 5 (SCWC, ACC): For trichloroacetic acid, there is consistent evidence of liver tumors in male mice but evidence for tumors is less consistent in female mice, and tumors have not been reported in rat studies.

Response 5: A more accurate characterization of the available evidence on the carcinogenicity of trichloroacetic acid from studies conducted in animals is the following summary by IARC[27]:

> "Several long-term bioassays (some including more than one experiment) have primarily focused on induction of liver tumours by trichloroacetic acid, with only limited pathology analyses of other tissues. Four drinking-water studies in male mice and two studies in female mice showed an increased incidence of hepatocellular adenoma and/or hepatocellular carcinoma. The only available study in rats given trichloroacetic acid in drinking-water had limited capacity to detect a carcinogenic response."

In short, liver tumors have been consistently observed in multiple long-term cancer bioassays in male and female mice, pathology analyses of tissues other than the liver

[26] OEHHA (2009). Technical Support Document for Cancer Potency Factors. Available from http://oehha.ca.gov/air/crnr/technical-support-document-cancer-potency-factors-2009
[27] IARC (2014). Full citation provided in footnote 6.

have been limited in these studies, and the only long-term cancer bioassay conducted in rats had a limited capacity to detect a carcinogenic response.

No changes to the proposed regulation were made based on this comment.

Comment 6 (ACC): The key study selected by OEHHA (DeAngelo et al., 2008) reported a high incidence of tumors in the control group, which diminishes the significance of the findings in the dose groups.

Response 6: The incidence of liver tumors in the control group of the 104-week $B6C3F_1$ mouse study by DeAngelo et al. (2008) study does not diminish the significance of the findings in the dose groups. Hepatocellular adenomas and carcinomas are commonly observed in male $B6C3F_1$ mice. NTP historical control data[28] reports incidences of 208/339 (61.4%) for adenomas and 95/339 (28%) for carcinomas in NTP drinking water studies conducted in male $B6C3F_1$ mice from 1984 to 1994[29]. Thus, an incidence of 31/56 (55.4%) hepatocellular adenoma or carcinoma combined in the control animals in the DeAngelo et al. (2008) male $B6C3F_1$ mouse study is not unusual.

Regardless of the incidence found in the control group, there was a significant increase in hepatocellular adenoma or carcinoma incidence in male mice in the 104-week study of DeAngelo et al. (2008) by exact trend test, as shown in Table 1 of the ISOR. Other studies also demonstrated significant increases in the incidences of hepatocellular adenoma or carcinoma by pairwise comparison with controls or by exact trend test (the 60-week study by DeAngelo et al. 2008 and the 52-week study by Bull et al. 2002). Thus, it has been shown through multiple studies that trichloroacetic acid induces liver tumors in mice. The 104-week DeAngelo et al. (2008) study was chosen because it had more animals per treatment group than the other two studies, and did not require a correction factor to extrapolate to two years to estimate lifetime animal cancer incidence in contrast to the other studies, which lasted only 60 or 52 weeks.

No changes to the proposed regulation were made based on these comments.

Comment 7 (ACC): Although OEHHA considered and rejected two other studies with male mice, it is unclear why the study by Pereira (1996) was excluded. That study reported liver tumors in female mice exposed to TCA for up to 576 days (82 weeks). Benchmark dose (BMD) modeling of the results of the Pereira study produces a 95% lower confidence limit on the BMD for a 10% response (BMDL10) of 4.67 mg/kg per day compared to a BMDL10 of 1.50 mg/kg per day for the study by DeAngelo et al. (2008).

[28] NTP (1999). National Toxicology Program Historical Controls. US Department of Health and Human Services. Available from https://ntp.niehs.nih.gov/ntp/research/database_searches/historical_controls/path/m_orlwr.txt

[29] DeAngelo et al. (2008) was conducted from 1991 to 1993. Control data should be compared to historical control data that are gathered within 2 or 3 years one way or the other of the study under review.

Response 7: OEHHA carefully evaluated and modeled the treatment-related tumor findings from the studies by DeAngelo et al. (2008)[30], Bull et al. (2002)[31], and Pereira (1996)[32]. The dose-response data were modeled by setting the benchmark response (BMR) to 5% in US EPA's BMDS[33]. OEHHA determined that it was appropriate to set the BMR to correspond to an extra risk of 5% when fitting the multistage cancer model to the data for trichloroacetic acid. In doing so, OEHHA followed a common scientific practice that is consistent with use of a BMR of 5% in other cancer dose-response assessments developed for Proposition 65[34] and other OEHHA programs[35], as well as the guidance in the resources provided by US EPA regarding use of BMDS[36].

The resulting cancer potencies obtained from modeling the data demonstrated that Pereira (1996) was less sensitive than DeAngelo et al. (2008) and Bull et al. (2002). Therefore, OEHHA decided not to report the results of Pereira et al. (1996) in the ISOR. As explained in Section 25703(a)(3), the "Risk analysis shall be based on the most sensitive study deemed to be of sufficient quality." Of the remaining studies (104-week DeAngelo et al. 2008, 60-week DeAngelo et al. 2008, and 52-week Bull et al. 2002), the 104-week study by DeAngelo et al. (2008) was the most appropriate study because it was the longest duration and had more animals per treatment group, and was thus chosen to derive the NSRL.

No changes to the proposed regulation were made based on these comments.

Comment 8 (SCWC, ACC): The mouse tumors appear to result from a non-genotoxic mechanism that can be defined as a threshold mechanism (i.e., no cancer risk below a threshold exposure level) and is of questionable relevance to humans. Peroxisome

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[30] DeAngelo et al. (2008). Full citation provided in footnote 3.
[31] Bull et al. (2002). Full citation provided in footnote 5.
[32] Pereira MA (1996). Carcinogenic activity of dichloroacetic acid and trichloroacetic acid in the liver of female B6C3F1 mice. Tox Sci 31(2):192-199.
[33] US EPA Benchmark Dose Software (BMDS) Version 2.7. National Center for Environmental Assessment, US EPA. Available from: https://www.epa.gov/bmds.
[34] *E.g.*, OEHHA (2017a). Initial Statement of Reasons Title 27, California Code of Regulations, Proposed Amendment to Section 25705(b) Specific Regulatory Levels Posing No Significant Risk: Vinylidene Chloride. Available at https://oehha.ca.gov/media/downloads/proposition-65/chemicals/isorvinylidenechloride092217.pdf; and OEHHA (2017b). Initial Statement of Reasons Title 27, California Code of Regulations, Proposed Amendment to Section 25705(b) Specific Regulatory Levels Posing No Significant Risk: Malathion. Available at https://oehha.ca.gov/media/downloads/crnr/malathionnsrlisor012017.pdf.
[35] *E.g.,* OEHHA (2018). Air Toxics Hot Spots Program Tertiary-Butyl Acetate Cancer Inhalation Unit Risk Factor, Technical Support Document for Cancer Potency Factors. Appendix B. Air and Site Assessment and Climate Indicator Branch, OEHHA, California Environmental Protection Agency, August. Available at https://oehha.ca.gov/media/downloads/crnr/tbaccanceriur081018.pdf; and OEHHA (2016). Air Toxics Hot Spots Program Perchloroethylene Inhalation Cancer Unit Risk Factor Technical Support Document for Cancer Potency Factors, Appendix B. Air, Community, and Environmental Research Branch, OEHHA, California Environmental Protection Agency, September. Available at https://oehha.ca.gov/media/downloads/crnr/pceurf090816.pdf.
[36] US EPA (2012). Benchmark Dose Technical Guidance. Risk Assessment Forum, US EPA. Available at: https://www.epa.gov/sites/production/files/2015-01/documents/benchmark_dose_guidance.pdf.

proliferation has been demonstrated in a number of short- and long-term trichloroacetic acid exposure studies in both rats and mice.

Response 8: As stated in the ISOR, IARC reviewed the mechanistic data for trichloroacetic acid in the 2014 monograph[37], and concluded there is "moderate evidence suggesting that trichloroacetic acid may act through multiple nongenotoxic mechanisms, leading to liver carcinogenesis". The monograph also stated that the "available evidence for nongenotoxic mechanisms for the rodent (mouse) liver tumours induced by trichloroacetic acid comprises the following: (i) epigenetic effects (especially DNA hypomethylation); (ii) cytotoxicity and oxidative stress; (iii) alteration of proliferation and apoptosis, and clonal expansion; (iv) PPARα activation; and (v) disruption of gap-junctional communication." These nongenotoxic mechanisms of tumor induction are supported by evidence from humans and experimental animals (see pages 417-425 of the 2014 monograph). Thus, there are multiple mechanisms through which trichloroacetic acid induces tumors, several of which are relevant to humans. The commenters did not provide evidence to support the assertion that there is a threshold or that the mechanism is not relevant to humans and did not recommend an alternative model that would be more suitable than the linear multistage model OEHHA used to derive the NSRL.

No changes to the proposed regulation were made based on these comments.

Comment 9 (SCWC): Separate evaluations by the National Toxicology Program and US EPA indicate that the PHG for trichloroacetic acid should not be based on carcinogenic effects.

Response 9: This comment is not relevant to the proposed rulemaking for the NSRL. Comments regarding the PHG will be addressed by the PHG program. Nevertheless, it bears reiterating that IARC has classified trichloroacetic acid as possibly carcinogenic to humans (Group 2B), based on sufficient evidence in experimental animals[38].

No changes to the proposed regulation were made based on these comments.

Alternatives Determination

In accordance with Government Code section 11346.9(a)(4), OEHHA has, throughout the adoption process of this regulation, considered available alternatives to determine whether any alternative would be more cost effective in carrying out the purpose for which the regulation was proposed, or would be as cost effective and less burdensome to affected private persons than the proposed action. No alternatives have been suggested. OEHHA has determined that no reasonable alternative would either be more effective in carrying out the purpose for which the action is proposed or would be

[37] IARC (2014). Full citation provided in footnote 6.
[38] *Ibid*.

as effective and less burdensome to affected private persons, or would be more cost-effective to affected private persons and equally effective in implementing the statutory policy or other provision of law than the proposed regulation.

For chemicals listed under the Act as known to cause cancer, the Act exempts discharges to sources of drinking water and exposures of people without provision of a warning if the exposure poses “no significant risk” of cancer (Health and Safety Code, section 25249.10(c)). The Act does not specify numerical levels of exposure that represent no significant risk of cancer.

The purpose of this regulation is to establish an NSRL for trichloroacetic acid. At or below this level, the Act does not require a warning or prohibit discharges of the chemical to sources of drinking water. Thus, adopting this level will allow businesses subject to the Act to determine whether a given discharge to sources of drinking water or a given exposure to this chemical is subject to the warning requirement or discharge prohibition provisions of the Act (Health and Safety Code, section 25249.5 and 25249.6).

Although Section 25703 describes principles and assumptions for conducting risk assessments to derive No Significant Risk Levels, some businesses subject to the Act do not have the resources to perform these assessments. Yet each business with ten or more employees must determine whether its activities or products are subject to the discharge prohibition or warning requirements of the Act. Adopting an NSRL for this chemical provides an efficient way of determining if a business is in compliance with the Act.

Local Mandate Determination

OEHHA has determined this regulatory action will not pose a mandate on local agencies or school districts, nor does it require reimbursement by the State pursuant to Part 7 (commencing with Section 17500) of Division 4 of the Government Code. OEHHA has also determined that no nondiscretionary costs or savings to local agencies or school districts will result from this regulatory action. Proposition 65 provides an express exemption from the warning requirement and discharge prohibition for all state and local agencies. Thus, these regulations do not impose any mandate on local agencies or school districts.