SAFE DRINKING WATER AND TOXIC ENFORCEMENT ACT OF 1986
PROPOSITION 65

INITIAL STATEMENT OF REASONS
TITLE 27, CALIFORNIA CODE OF REGULATIONS

PROPOSED AMENDMENT TO
SECTION 25805(b), SPECIFIC REGULATORY LEVELS: CHEMICALS CAUSING REPRODUCTIVE TOXICITY

BUTYL BENZYL PHTHALATE (ORAL EXPOSURE)

PURPOSE AND BACKGROUND OF PROPOSED AMENDMENT

PURPOSE

This proposed regulatory amendment would adopt an oral Maximum Allowable Dose Level (MADL) for butyl benzyl phthalate (BBP) under Proposition 65[1] in Title 27, California Code of Regulations, section 25805(b) [2]. The proposed oral MADL for BBP is 1,200 micrograms per day, and was derived using scientific methods outlined in Section 25803.

PROPOSITION 65 AND THE LISTING OF BBP

Proposition 65 was enacted as a voters' 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 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 to sources of drinking water. Warning is not required if exposure is at or below a safe harbor level – the MADL for a chemical listed as known to cause reproductive toxicity.

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[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 subsequent citations are to Title 27, California Code of Regulations, unless otherwise noted.
[3] Cal. Code of Regs., Title 27, Division 4. Chapter 1. Article 1. Preamble(a).
[4] Health and Safety Code, section 25249.12(a).

On December 2, 2005, BBP was added to the Proposition 65 list, based on formal identification as causing reproductive toxicity (developmental endpoint) by the National Toxicology Program (NTP) in a report by its Center for the Evaluation of Risks to Human Reproduction (CERHR).[5] The NTP (solely as to final reports of the CERHR) is identified as an authoritative body for reproductive toxicity under Proposition 65 (Section 25306(l)).

STUDY SELECTION FOR MADL DERIVATION

OEHHA reviewed the studies identified in the NTP final report, and conducted a search for any relevant studies published after the report was completed.

Human Studies

Many human studies have investigated potential associations of exposure to phthalates with developmental or reproductive toxicity in humans. Since the listing of BBP under Proposition 65 is based solely on developmental toxicity, studies of prenatal exposure and developmental outcomes were selected as the possible basis for MADL development. Studies of male or female reproductive effects or other effects that focused on postnatal exposure scenarios were not included in the selection of possible studies for MADL development.

Epidemiological studies in humans usually use urinary levels of phthalate metabolites rather than the parent compounds as measures of exposure to phthalates. This is because after ingestion, phthalates are quickly and extensively metabolized. BBP is metabolized by hydrolysis to monoesters including mono-butyl phthalate (MBP) and mono-benzyl phthalate (MBzP). In rats, about 36% of BBP was observed to be excreted in urine as MBzP[6]. MBP is generally present in the highest amount among all urinary metabolites in rats following oral administration of BBP[7]. In addition, MBP is also the active

[5] National Toxicology Program (NTP, 2003a). NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Butyl Benzyl Phthalate (BBP). Center for the Evaluation of Risks to Human Reproduction, NTP, U.S. Department of Health and Human Services, Research Triangle Park, NC.
[6] Koo, J. W., F. Parham, M. C. Kohn, S. A. Masten, J. W. Brock, L. L. Needham and C. J. Portier (2002). The association between biomarker-based exposure estimates for phthalates and demographic factors in a human reference population. Environ Health Perspect 110(4): 405-410.
[7] National Toxicology Program (NTP, 2003b). NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Di-n-Butyl Phthalate (DBP). Center for the Evaluation of Risks to Human Reproduction, NTP, U.S. Department of Health and Human Services, Research Triangle Park, NC.

metabolite of di-n-butyl phthalate (DBP)[8]. Therefore, MBP in biological samples can indicate exposure to BBP or DBP, or both. However, the presence of MBzP reflects exposure to BBP only; consequently, concentrations of MBzP reflect levels of BBP exposure. Epidemiological studies that demonstrate a statistically significant association of urinary levels of MBzP to developmental effects in humans can indicate developmental toxicity of BBP in humans.

Several epidemiological studies published after the release of the NTP-CERHR report investigated potential association between prenatal exposure to phthalates and health outcomes in newborn infants.[9,10,11,12,13,14] These studies were reviewed to determine whether or not they could provide a basis for the calculation of a MADL.

Swan et al. (2005) analyzed levels of MBzP, MBP, and seven other phthalate monoesters in urine samples from 85 pregnant women at a mean gestational time of 28.3 weeks. The authors also performed genital examination and measured anogenital distance (AGD) in a total of 134 boys at 2-30 months of age. Data from 85 boys whose mothers' urine samples had been analyzed for phthalates were included in the statistical regression analysis of AGD and phthalate levels. The authors found that increased levels of MEP, MBP, MBzP, and mono-isobutyl phthalate in prenatal urine samples in mothers were associated with decreased AGD in boys after birth. The subjects were divided into three groups based on the phthalate levels in their mothers' urine ($<25^{th}$, $\geq 25^{th}$ to $< 75^{th}$, and $\geq 75^{th}$ percentile for the low, medium, and high exposure groups, respectively, with the lowest exposure group serving as a reference group). Based on MBP levels in the maternal urine samples, the odds ratios for shorter than expected age- and body weight-adjusted AGD in three groups were

[8] NTP (2003b). NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Di-n-Butyl Phthalate (DBP).
[9] Swan, S. H., K. M. Main, F. Liu, S. L. Stewart, R. L. Kruse, A. M. Calafat, C. S. Mao, J. B. Redmon, C. L. Ternand, S. Sullivan and J. L. Teague (2005). Decrease in anogenital distance among male infants with prenatal phthalate exposure. Environ Health Perspect 113(8): 1056-1061.
[10] Swan, S. H. (2008). Environmental phthalate exposure in relation to reproductive outcomes and other health endpoints in humans. Environ Res 108(2): 177-184.
[11] Huang, P. C., P. L. Kuo, Y. Y. Chou, S. J. Lin and C. C. Lee (2009). Association between prenatal exposure to phthalates and the health of newborns. Environ Int 35(1): 14-20.
[12] Engel, S. M., A. Miodovnik, R. L. Canfield, C. Zhu, M. J. Silva, A. M. Calafat and M. S. Wolff (2010). Prenatal phthalate exposure is associated with childhood behavior and executive functioning. Environ Health Perspect 118(4): 565-571.
[13] Suzuki, Y., M. Niwa, J. Yoshinaga, Y. Mizumoto, S. Serizawa and H. Shiraishi (2010). Prenatal exposure to phthalate esters and PAHs and birth outcomes. Environ Int 36(7): 699-704.
[14] Suzuki, Y., J. Yoshinaga, Y. Mizumoto, S. Serizawa and H. Shiraishi (2011). Foetal exposure to phthalate esters and anogenital distance in male newborns. Int J Androl.

1.0, 3.8 (95% CI = 1.2 – 12.3), and 10.2 (95% CI = 2.5-42.2). Based on MBzP levels, the odds ratios for shorter than expected age- and body weight-adjusted AGD in the three groups were 1.0, 3.1 (95% CI = 1.002 – 9.8), and 3.8 (95% CI = 1.03 – 13.9). The results from this study indicate high maternal urinary levels of MBP or MBzP, which are associated with increased risk of reduced AGD in boys.

In the second study by Swan (2008)[15], a total of 106 boys were included in the statistical analysis and 68 of them were among the subjects in the 2005 report[16]. The statistical method to control for factors (e.g., age) used in the 2008 report was also different from that used in the 2005 report. Urinary concentration of MBP but not MBzP in the mothers during gestation was significantly and inversely related to AGD.

Huang et al. (2009) found an association between AGD and MBP in female newborns; in a Japanese study, Suzuki et al. (2010, 2011) found spot urine samples from pregnant women correlated with DEHP exposure (but not other phthalates) and AGD in newborn males in one study and no significant associations with birth outcomes and phthalates in a second study[17]. Maternal MBzP was not significantly associated with developmental effects in the newborns.

In summary, multiple studies found an association between maternal urinary levels of MBP, a common metabolite of DBP and BBP, and adverse developmental outcome. However, an association between urinary levels of MBzP and developmental effects in humans was found in one study but not in others. Because of co-exposure to multiple phthalates, definitive findings for individual phthalates pose a challenge. Because of multiple concurrent phthalate exposures, and the lack of a strong study establishing a quantitative relationship between BBP exposure and effect, the human data do not provide a sufficient basis for developing the MADL for BBP.

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[15] Swan, S. H. (2008). Environmental phthalate exposure in relation to reproductive outcomes and other health endpoints in humans. Environ Res 108(2): 177-184.

[16] Swan, S. H., K. M. Main, F. Liu, S. L. Stewart, R. L. Kruse, A. M. Calafat, C. S. Mao, J. B. Redmon, C. L. Ternand, S. Sullivan and J. L. Teague (2005). Decrease in anogenital distance among male infants with prenatal phthalate exposure. Environ Health Perspect 113(8): 1056-1061

[17] Huang, P. C., P. L. Kuo, Y. Y. Chou, S. J. Lin and C. C. Lee (2009). Association between prenatal exposure to phthalates and the health of newborns. Environ Int 35(1): 14-20.
Suzuki, Y., M. Niwa, J. Yoshinaga, Y. Mizumoto, S. Serizawa and H. Shiraishi (2010). Prenatal exposure to phthalate esters and PAHs and birth outcomes. Environ Int 36(7): 699-704.
Suzuki, Y., J. Yoshinaga, Y. Mizumoto, S. Serizawa and H. Shiraishi (2011). Foetal exposure to phthalate esters and anogenital distance in male newborns. Int J Androl. Published online at doi: 10.1111/j.1365-2605..

Studies in Laboratory Animals

The NTP-CERHR Monograph includes reviews of a number of developmental toxicity studies. The Expert Panel report, which comprises part of the Monograph, identified 182 milligrams per kilogram of bodyweight per day (mg/kg-day) in mice to be the lowest No Observable Adverse Effect Level (NOAEL) for the developmental effects of BBP[18]. The NOAEL was observed in a standard teratology study conducted by the National Toxicology Program (NTP) in Swiss CD-1 mice[19]. The animals were treated with BBP in diet from gestational days (GD) 6 to 15. BBP at 910 mg/kg-day caused significant increases in prenatal mortality and visceral, skeletal and external malformations. Subsequent to the completion of the Expert Panel report, Nagao et al. (2000)[20] published the results of a two-generation reproductive toxicity study of BBP in Sprague-Dawley rats. The NOAEL in this study was 20 mg/kg-day. This NOAEL was used by NTP in the NTP-CERHR report in judging the level of concern of human exposures (NTP-CERHR Monograph, p. 4). The NTP noted that the LOAEL in this study was at 100 mg/kg-day, and the effect at this dose was reduced pup weights.

Two additional pertinent studies were published after the publication of the NTP-CERHR Monograph, a two-generation reproduction study by Tyl et al. (2004)[21] and a mechanistic study by Sumner et al. (2009)[22]. The latter found prenatal exposure to 25 mg/kg-day of BBP caused malformations in the reproductive system and abnormal metabolic changes in the offspring at 25 mg/kg-day, a dose level just above the NOAEL used by the NTP.

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[18] National Toxicology Program (NTP, 2003b). NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Di-n-Butyl Phthalate (DBP). Center for the Evaluation of Risks to Human Reproduction, NTP, U.S. Department of Health and Human Services, Research Triangle Park, NC.

[19] Price CJ, Field EA, Marr MC, Myers CB. Final report on the developmental toxicity of butyl benzyl phthalate (CAS No. 85-68-7) in CD-1-Swiss mice. NTP-90-114. Research Triangle Park: National Toxicology Program, National Institute of Environmental Health Sciences, 1990. Original data accessible at: http://ntp-apps.niehs.nih.gov/ntp_tox/index.cfm.

[20] Nagao, T., R. Ohta, H. Marumo, T. Shindo, S. Yoshimura and H. Ono (2000). Effect of butyl benzyl phthalate in Sprague-Dawley rats after gavage administration: a two-generation reproductive study. Reprod Toxicol 14(6): 513-532.

[21] Tyl RW, Myers CB, Marr MC, Fail PA, Seely JC, Brine DR, Barter RA, Butala JH. (2004). Reproductive toxicity evaluation of dietary butyl benzyl phthalate (BBP) in rats. Reprod Toxicol. 18(2):241-64.

[22] Sumner S., R. Snyder, J. Burgess, C. Meyers, R. Tyl, C. Sloan and T. Fennell (2009). Metabolomics in the assessment of chemical-induced reproductive and developmental outcomes using non-invasive biological fluids: application to the study of butylbenzyl phthalate. J. Appl. Toxicol. 29: 703-714.

Under the Proposition 65 program, developmental effects resulting entirely or predominantly from prenatal exposure are considered in establishing the MADLs for the developmental toxicity endpoint. Therefore, in the multi-generation Nagao et al. and Tyl et al. studies, only effects on the fetus such as reduced birth weight or alterations in the anogenital distance at birth are considered here for the development of the MADL. Major findings from the three sensitive studies on the developmental effects resulting from prenatal exposure to BBP are summarized in Table 1.

Table 1. Three sensitive studies in animals on the developmental toxicity of BBP

Reference	Study Design	Critical Dev. Effect	Dose levels (mg/kg-day)
Nagao et al., 2000[20]	Two-generation reproduction study. SD rats; 25 rats per sex per group. 0, 20, 100, and 500 mg/kg-day by oral gavage.	High dose: reduction in viability in PND0-4,birth weight and anogenital distance (AGD) on PND 0 in F1 males, but not in F2 males. Mid dose: reduced birth weight in F1 male and females, but not in F2 males. Low dose: no effect.	LOEL: 100 NOEL: 20
Tyl et al., 2004[21]	Two-generation reproduction study. SD rats; 30 rats per sex per group. 0, 50, 250, and 750 mg/kg-day (estimated) via feed.	High dose: reduction implantations and live birth in F2; reduced AGD in F1 and F2 males; reduced birth weight in F1 males and females. Mid dose: reduced AGD in F1 and F2 males. Low dose: no effect.	LOEL: 250 NOEL: 50
Sumner et al., 2009[22]	Mechanistic study; Pregnant SD rats, 3 dams per group. 17, 16, and 6 male offspring in the control, low and high dose group, respectively. 0, 25, and 750 mg/kg-day by oral gavage from GD 14 to 19.	High dose: All six male pups had retained areolae and reduced AGD on PND (p=0.0019, Fisher's exact test as calculated by OEHHA) Three male pups had retained nipples at PND 11 or 13. On PND 26, all six male pups had reduced AGD and were missing part or all of the epididymis and seminal vesicles. Low dose: Seven of the 16 male pups had retained areolae on PND 11, but not on PND 26 (p=0.0027, Fisher's exact test as calculated by OEHHA). Two male pups had reduced AGD on PND 21, but not on PND 0 or 26. Significant alterations in metabolomics in male pups from both treated groups on PND 26.	LOEL: 25

The study by Sumner et al. (2009)[22] reported obvious adverse effects of BBP on the developing male reproductive system in rats following gestational exposure at

25 mg/kg-day, the lowest effective dose level for developmental endpoints in studies in laboratory animals. In this study, three pregnant Sprague-Dawley rats received 0, 25 and 750 mg/kg-day of BBP in corn oil, respectively, by oral gavage from GD 14 to 19. The authors reported 17 male /13 female, 16 male /15 female, and 6 male/9 female in the control, low-dose, and high-dose groups, respectively. None of the pups in the control group or female pups in the BBP-treated groups showed alterations in the reproductive system. In the low dose (25 mg/kg-day) group, a total of 9 males in the three litters had reproductive findings, including seven males with retained areolae on PND 11 and two males with reduced AGD on PND 21 (but not on PND 0). None of the males in the low-dose group had reduced AGD or retained areolae on PND 26. Gestational treatment with 750 mg/kg-day of BBP caused severe damage to the reproductive system of the male pups. All six male pups in this dose group had retained areolae, reduced AGD and partially or completely missing epididymis and seminal vesicles. Four of the six male pups had missing or abnormal testes.

The study did not report the statistical significance of the incidence of alterations in the reproductive system of the offspring. OEHHA calculated statistical significance values. The high incidences of retained areolae in male pups in both the 750 mg/kg-day dose group (p=0.0019, Fisher's exact test) and the 25 mg/kg-day dose group (p=0.0027, Fisher's exact test) were statistically significant on an individual pup basis. Therefore, as noted above, 25 mg/kg-day in this study is a LOEL[23]. The NOEL in the study by Tyl et al (2004) was 50 mg/kg-day, which is higher than the LOEL in the study by Sumner et al. (2009). The highest developmental NOEL among the studies that does not exceed the lowest LOEL is 20 mg/kg-day, reported in the study by Nagao et al. (2000)[20]. Also, as noted above, the NTP identified 20 mg/kg-day as the NOEL for developmental effects for this compound in the NTP-CERHR report.

The Nagao study is a two-generation reproductive toxicity study. Groups of SD rats, 25 animals per group per sex, were treated by oral gavage with BBP in corn oil at doses of 0, 20, 100, and 500 mg/kg-day. Endpoints for developmental effects resulting from prenatal exposure include parameters assessed on postnatal day 0 such as the number of live and dead pups, viability of pups on PND 0-4, birth weight, AGD on PND 0, and external malformations. There were no general, developmental, or reproductive effects at the low dose (20 mg/kg-day). At the mid-dose level (100 mg/kg-day), BBP treatment reduced birth

[23] Studies cited identified no observed adverse effects levels (NOAELs) and lowest observed adverse effects levels (LOAELs). For purposes of Proposition 65, these are equivalent to NOELs and LOELs and are reported as such.

weights in F1 pups (both sexes). In the high dose group (500 mg/kg-day), viability and birth weights were reduced in F1 pups, and the AGD of F1 male pups on PND 0 was shortened. There were no developmental effects in F2 pups at any dose, and none of F1 or F2 pups in the BBP-treated groups showed dose-related external malformations at birth. The dose of 100 mg/kg-day is identified as a LOEL, and 20 mg/kg-day as the NOEL. This study was well designed and reported, and is of sufficient quality to serve as the basis for the MADL.

MADL CALCULATION

The following calculations were performed in accordance with Section 25803 to derive the oral MADL for BBP:

- Calculation of NOEL dose for a 58 kg woman:
  20 mg/kg-day × 58 kg = 1160 mg/day,
  or 1,200 mg/day after rounding

- Derivation of the MADL by dividing the NOEL expressed in mg/day by one thousand (Section 25801(b)(1)):
  $MADL_{oral}$ = 1,200 mg/day / 1000 = **1,200 micrograms/day**

This MADL applies to exposure to BBP by the oral route.

PROPOSED REGULATORY AMENDMENT

The proposed change to Section 25805(b) is provided below in underline:

Chemical name	*Level (micrograms per day)*
…	
Butyl Benzyl Phthalate (BBP)	1,200 (oral)
…	

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 a MADL, that does not trigger the warning requirement or discharge prohibition.

NECESSITY

This proposed regulatory amendment would adopt a MADL that conforms with the Proposition 65 implementing regulations and reflects the currently available scientific knowledge about BBP. The MADL provides assurance to the regulated community that exposures or discharges at or below it are considered not to pose a significant risk of developmental or reproductive harm. Exposures at or below the MADL are exempt from the warning and discharge provisions of Proposition 65[24].

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

OEHHA reviewed the 2003 National Toxicology Program (NTP) Monograph on the Potential Human Reproductive and Developmental Effects of Butyl Benzyl Phthalate from the NTP Center for the Evaluation of Risks to Human Reproduction (CERHR)[5]. OEHHA determined that the most sensitive study deemed to be of sufficient quality is the oral two-generation reproductive toxicity study reported by Nagao et al. (2000), and that there were no subsequently published studies that were more sensitive. OEHHA used the values from this study as the basis for calculating the oral MADL for BBP proposed for adoption into Section 25805(b). A copy of the 2003 NTP-CERHR BBP monograph and the study by Nagao et al (2000)[20] will be included in the regulatory file for this action, and are available from OEHHA upon request. OEHHA also relied on the attached Economic Impact Assessment in developing this proposed regulation

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

The proposed MADL provides a "safe harbor" value that aids businesses in determining if they are complying with the law. The alternative to the amendment to Section 25805(b) would be to not promulgate a MADL for the chemical. Failure to promulgate a MADL would leave the business community without a safe harbor level to assist them in determining compliance with Proposition 65.

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

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. 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 MADL 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.

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 any monetary values for this proposed regulation given that its use is entirely voluntary and it only provides compliance assistance for businesses subject to the Act.

Impact on the Creation, Elimination, or Expansion 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. BBP is listed under Proposition 65; therefore, businesses and individuals who manufacture, distribute or sell products with BBP in the state must provide a warning if their product or activity exposes the public or employees to this chemical.

Benefits of the Proposed Regulation: The MADL provides a “safe harbor” value that aids businesses in determining if they are complying with the law. Some businesses may not be able to afford the expense of establishing a MADL and therefore may be exposed to litigation for a failure to warn or for a prohibited discharge of the listed chemical. Adopting this regulation will save these businesses those expenses and may reduce litigation costs. By providing a MADL, this regulatory proposal does not require, but may encourage, businesses to lower the amount of the listed chemical in their product to a level that does not cause a significant exposure, thereby providing a public health benefit to Californians.

Problem being addressed by this proposed rulemaking: Proposition 65 does not provide specific 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 that does not require a warning or trigger the discharge prohibition.

How the proposed regulation addresses the problem: The proposed regulation would adopt a specific regulatory level for a listed chemical to provide compliance assistance for businesses that are subject to the requirements of the Act. While OEHHA is not required to adopt such levels, adopting them provides

a “safe harbor” for businesses and provides certainty that they are complying with the law if the exposures or discharges they cause are below the established level.

Reasonable alternatives to the proposed regulation: OEHHA determined that the only alternative to the proposed regulation would be to not adopt a MADL for this chemical. This alternative was rejected because it would fail to provide businesses with the certainty that the MADL can provide.