



Toxicological Summary for: Butyl Benzyl Phthalate

CAS: 85-68-7

Synonyms: BBP; Butylbenzyl phthalate; Butyl benzylphthalate; 1,2-Benzenedicarboxylic acid, butyl phenylmethyl ester

Acute Non-Cancer Health Risk Limit (nHRL_{Acute}) = 100 µg/L

$$= \frac{(\text{Reference Dose, mg/kg/d}) \times (\text{Relative Source Contribution}) \times (\text{Conversion Factor})}{(\text{Acute intake rate, L/kg/d})}$$

$$= \frac{(0.15 \text{ mg/kg/d}) \times (0.2)^* \times (1000 \text{ µg/mg})}{(0.289 \text{ L/kg-d})}$$

$$= 104 \text{ rounded to } \mathbf{100 \text{ µg/L}}$$

*MDH utilizes the EPA Exposure Decision Tree (EPA 2000) to select appropriate RSCs (MDH 2008, Appendix K). Typically an RSC of 0.5 is utilized for nonvolatile contaminants. However, there is evidence that there are significant known or potential sources other than ingestion of water. An RSC of 0.2 was selected rather than the default value of 0.5 for nonvolatile contaminants.

Reference Dose/Concentration:	0.15 mg/kg-d (Sprague Dawley rats)
Source of toxicity value:	MDH, 2012
Point of Departure (POD):	20 mg/kg-d (NOAEL from Nagao et al., 2000)
Human Equivalent Dose (HED):	20 x 0.23 = 4.6 mg/kg-d (Minnesota Department of Health (MDH) 2011)
Total uncertainty factor:	30
Uncertainty factor allocation:	3 for interspecies extrapolation to address potential differences in toxicodynamics and 10 for intraspecies variability
Critical effect(s):	Decreased pup body weight and decreased serum thyroid hormone levels
Co-critical effect(s):	None
Additivity endpoint(s):	Developmental (E) (body weight, thyroid hormone levels)

Short-term Non-Cancer Health Risk Limit (nHRL_{Short-term}) = 100 µg/L

$$\begin{aligned} &= \frac{(\text{Reference Dose, mg/kg/d}) \times (\text{Relative Source Contribution}) \times (\text{Conversion Factor})}{(\text{Short-term intake rate, L/kg/d})} \\ &= \frac{(0.15 \text{ mg/kg/d}) \times (0.2)^* \times (1000 \text{ µg/mg})}{(0.289 \text{ L/kg-d})} \\ &= 104 \text{ rounded to } \mathbf{100 \text{ µg/L}} \end{aligned}$$

*MDH utilizes the EPA Exposure Decision Tree (EPA 2000) to select appropriate RSCs (MDH 2008, Appendix K). Typically an RSC of 0.5 is utilized for nonvolatile contaminants. However, there is evidence that there are significant known or potential sources other than ingestion of water. An RSC of 0.2 was selected rather than the default value of 0.5 for nonvolatile contaminants.

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Uncertainty factor allocation:	3 for interspecies extrapolation to address potential differences in toxicodynamics and 10 for intraspecies variability
Critical effect(s):	Decreased pup body weight and decreased serum thyroid hormone levels
Co-critical effect(s):	None
Additivity endpoint(s):	Developmental (E) (body weight, thyroid hormone levels)

Subchronic Non-Cancer Health Risk Limit (nHRL_{Subchronic}) = Short-term nHRL = 100 µg/L

$$\begin{aligned} &= \frac{(\text{Reference Dose, mg/kg/d}) \times (\text{Relative Source Contribution}) \times (\text{Conversion Factor})}{(\text{Subchronic intake rate, L/kg/d})} \\ &= \frac{(0.15 \text{ mg/kg/d}) \times (0.2)^* \times (1000 \text{ µg/mg})}{(0.077 \text{ L/kg-d})} \\ &= 390 \text{ rounded to } \mathbf{400 \text{ µg/L}} \end{aligned}$$

Reference Dose/Concentration Use the Short-term RfD**

**The calculated Subchronic RfD (0.83 mg/kg-d) is higher than the Short-term RfD (0.15 mg/kg-d), which is based on developmental effects. The Subchronic RfD must be protective of all types of adverse effects that could occur as a result of subchronic exposure, including short-term effects (MDH 2008, page 34). Therefore, the Subchronic RfD is set to the Short-term RfD.

The Subchronic nHRL must be protective of the acute and short-term exposures that occur within the subchronic period and therefore, the Subchronic nHRL is set equal to the Short-term nHRL of 100 µg/L. Additivity endpoints: Developmental (E) (body weight, thyroid hormone levels).

Chronic Non-Cancer Health Risk Limit (nHRL_{Chronic}) = nHRL_{Short-term} = 100 µg/L

$$= \frac{(\text{Reference Dose, mg/kg/d}) \times (\text{Relative Source Contribution}) \times (\text{Conversion Factor})}{(\text{Chronic intake rate, L/kg/d})}$$

$$= \frac{(0.15 \text{ mg/kg/d}) \times (0.2)^* \times (1000 \text{ µg/mg})}{(0.043 \text{ L/kg-d})}$$

$$= 698 \text{ rounded to } 700 \text{ µg/L}$$

Reference Dose/Concentration Use the Short-term RfD**

**The calculated Chronic RfD (1.1 mg/kg-d) is higher than the Short-term RfD (0.15 mg/kg-d), which is based on developmental effects. The Chronic RfD must be protective of all types of adverse effects that could occur as a result of chronic exposure, including short-term effects (MDH 2008, page 34). Therefore, the Chronic RfD is set to the Short-term RfD."

The Chronic nHRL must be protective of the acute and short-term exposures that occur within the chronic period and therefore, the Chronic nHRL is set equal to the Short-term nHRL of 100 µg/L. Additivity endpoints: Developmental (E) (body weight, thyroid hormone levels).

Cancer Health Risk Limit (cHRL) = "Not Applicable"##

Cancer classification:	Group C (US EPA IRIS 1993) "Likely to be carcinogenic" (US EPA PPRTV 2002)
Slope factor:	0.0019 per mg/kg-d#
Source of slope factor:	US EPA PPRTV 2002
Tumor site(s):	Pancreas

MDH has chosen to not use the EPA PPRTV cancer slope factor to generate a cancer HRL. MDH considers BBP to be a nonlinear carcinogen based on lack of positive genotoxicity data and evidence of clear morphological continuum from focal pancreatic acinar cell hyperplasia (preneoplastic lesion) to adenoma to carcinoma in male rats (NTP 1997). Carcinogenicity was equivocal in female rats despite 2-fold higher dose levels and negative in mice (NTP 1997). The 2 year NTP 1997 cancer bioassay NOAEL_{HED} was 32.4 mg/kg-d. The RfD (0.15 mg/kg-d) is 162-fold lower than the NTP study NOAEL_{HED} and is therefore considered to be protective against cancer.

Volatile: Yes (Low)

Summary of Guidance Value History:

The 2012 HBVs (100 µg/L) are the same as the 1993 HRL chronic value (100 µg/L), however, the basis of the value has changed as the result of: 1) utilization of more recent toxicity information; 2) removal of the 10-fold Group C uncertainty factor; 3) utilization of more recent intake rates which incorporate higher intake rates during early life; and 4) rounding to one significant digit. In 2015, the 2012 HBVs were adopted into rule as HRLs.

Summary of toxicity testing for health effects identified in the Health Standards Statute:

	Endocrine	Immunotoxicity	Development	Reproductive	Neurotoxicity
Tested?	Yes	Yes	Yes	Yes	Yes
Effects?	Yes ¹	No ²	Yes ³	Yes ⁴	No ⁵

Note: Even if testing for a specific health effect was not conducted for this chemical, information about that effect might be available from studies conducted for other purposes. Most chemicals have been subject to multiple studies in which researchers identify a dose where no effects were observed, and the lowest dose that caused one or more effects. A toxicity value based on the effect observed at the lowest dose across all available studies is considered protective of all other effects that occur at higher doses.

Comments on extent of testing or effects:

- ¹ Potential estrogen activity of BBP and the major BBP metabolites MBuP and MBeP has been investigated in both *in vitro* and *in vivo* studies. Only weak estrogen activity at high concentrations/doses of BBP was reported.

Some epidemiology studies have identified associations between phthalate exposure and changes in reproductive development in newborn boys. However, these effects were not consistently observed and studies were generally accompanied by multiple confounding factors such that it is not possible to draw conclusions.

Multiple studies in laboratory animals have demonstrated antiandrogen-like activity of BBP and its major metabolites. Indicators of antiandrogenic activity include reduced anogenital distance, areolas in neonatal males, reduced testicular weight, and disrupted testicular migration following *in utero* and lactational exposure.

Decreased pup body weight and serum levels of thyroid hormones in developing laboratory animals exposed to BBP *in utero* and via lactation have been reported at dose levels below those causing male reproductive developmental effects. Decreased body weight and changes in thyroid hormone levels are identified as critical effects and form the basis of the RfD.

- ² Several mechanistic toxicological studies and epidemiological studies have been conducted to evaluate immunotoxicity, mainly on other phthalates (e.g., DEHP). The mechanistic studies typically utilized topical or subcutaneous injection as the route of exposure. Epidemiological studies have suggested an association with PVC-related exposure and asthma.

Limited studies specifically evaluating immunologic effects have been conducted in laboratory animals. No significant immune suppression or enhancement was observed in rats treated with 0.6, 1.2 or 2.4% BBP for up to 12 months. Limited studies on a related phthalate, DEHP, suggest that immunological effects could occur at a similar order of magnitude dose as those causing male developmental effects. It is anticipated that the RfD for BBP will be protective of male developmental effects as well as immunological effects.

- ³ Some epidemiology studies have identified an association between phthalate exposure and male reproductive and neurobehavioral development. However, effects were not consistently observed and further studies are needed before conclusions can be drawn.

Studies in laboratory animals have identified the male reproductive system, particularly in during the developmental stage, to be a target for the toxicity of BBP. Decreased pup body weight and serum levels of thyroid hormones in developing laboratory animals exposed to BBP *in utero* and via lactation have been reported at dose levels below those causing male reproductive developmental

effects. Decreased body weight and changes in thyroid hormone levels are identified as critical effects and form the basis of the RfD.

The developmental effects of the major BBP metabolites (mono butyl phthalate (MBP) and mono benzyl phthalate (MBzP)) was similar to the effects observed after exposure to BBP, suggesting that MBP and MBzP may be responsible for the developmental effects of BBP.

- ⁴ BBP and its major metabolites (MBP & MBzP) have been found to adversely affect the reproductive organs in experimental animal studies which may impact fertility. The developmental period is a sensitive life stage to the male reproductive effects of BBP. Main effects reported include a decrease reproductive organ weights, damage to the testis, epididymis, prostate, seminal vesicle and to reduced sperm concentrations, and at higher BBP doses reduced fertility, in addition to increases in relative liver and kidney weights. Decreases in pup body weight and changes in serum thyroid hormone levels, the basis of the RfD, occurred at dose level lower than those associated with male reproductive developmental effects.
- ⁵ Some epidemiological studies have reported associations between maternal phthalates and metabolites and neurobehavioral changes in offspring. Two 2-generational studies conducted in laboratory animals have assessed neurological endpoints. Neither study reported evidence of neurological impairment. A related phthalate, DBP, has also been evaluated for neurodevelopmental effects and no neurobehavioral impairment was observed.

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