Table 2. Summary of New Animal / *In Vivo* Data for HBCD										
Target Organ/ System	**Study Type**	**Species/ Strain/Sex (Number/ group)**	**Exposure Route**	**Doses/ Concentrations**	**Duration**	**Author Reported Effect Dose/ Concentration/ Result**	**Reviewer Reported Effect Dose/ Concentration/ Result**	**Effect Measured**	**Reference**	**Data Quality Evaluation**
Skin and Connective Tissue	Subchronic (30-90 days)	Rat Other Male (15/group)	Oral	0 , 100 , 300 , 1000 mg/kg-bw/day	7 days/week for 90 weeks	NOAEL = 1000 mg/kg - bw/day	Reviewer Agreed with Author	No effects were reported on neurological/behavior, body weight, renal, ocular, gastrointestinal, cardiovascular, respiratory, or skin endpoints.	ACC (2002)	High
Skin and Connective Tissue	Subchronic (30-90 days)	Rat Other Female (15/group)	Oral	0 , 100 , 300 , 1000 mg/kg-bw/day	7 days/week for 90 weeks	NOAEL = 0.048	NOAEL = 1000 mg/kg - bw/day	No effects were reported on mortality, neurological/behavior, body weight, hematology, renal, ocular, reproductive, gastrointestinal, cardiovascular, respiratory, or skin endpoints.	ACC (2002)	High
Thyroid	Short-term (1-30 days)	Rat Other Female (6/group)	Oral	0, 3, 30 mg/kg-bw/day	7 days	Not Reported	NOAEL = 30 mg/kg - bw/day	TSH, T3, LH, FSH, Leptin, and Cortocosterone levels	Miller et al (2016)	Medium
Thyroid	Subchronic (30-90 days)	Rat Other Male (15/group)	Oral	0 , 100 , 300 , 1000 mg/kg-bw/day	7 days/week for 90 weeks	NOAEL = 0.048	LOAEL = 1000 mg/kg - bw/day	Follicular cell hypertrophy in thyroid, decreased T4 levels, increased prostate gland weight, decreased survival	ACC (2002)	High
Thyroid	Subchronic (30-90 days)	Rat Other Female (15/group)	Oral	0 , 100 , 300 , 1000 mg/kg-bw/day	7 days/week for 90 weeks	NOAEL = 0.048	LOAEL = 300 mg/kg - bw/day	Follicular cell hypertrophy in thyroid, decreased T4 levels	ACC (2002)	High

1.3 Mechanistic / *In Vitro* Data Identified from OPPT Literature Search

Table 3. Summary of New Mechanistic / *In Vitro* Data for HBCD

Target Organ/ System	Study Type	Species/ Strain/Cell type (Number/ Group)	Exposure Route	Doses/ Concentrations	Duration	Effect Dose/ Concentration/ Result	Effect Measured	Reference	Data Quality Evaluation
Immune	Short-term	Human peripheral blood mononuclear cells (PBMCs), monocyte-depleted peripheral blood mononuclear cells (MD-PBMCs), and natural killer (NK) cells (4-6 donors, n ≥ 4 replicates)	*In vitro*	0, 0.05, 0.1, 0.25, 0.5, 1, 2.5, 5 µM	24 h, 48 h and 6 days	No effect up to 5 µM	Cell viability with or without inhibitors of NFκB (BAY11-7085), MEK ½ (PD98059), p38 (SB202190), and JNK (JNK B178D3)	(Almughamsi and Whalen, 2016) (Data not shown in report, but provided in a supplementary document available at https://link.springer.com/article/10.1007%2Fs00204-015-1586-6)	High
Immune	Short-term	Human PBMCs, MD-PBMCs and NK cells (4-6 donors, 3 replicates)	*In vitro*	0, 0.05, 0.1, 0.25, 0.5, 1, 2.5, 5 µM	24 h, 48 h and 6 days	LOEC PBMCs and MD-PBMCs: 0.05 µM LOEC NK cells: 0.1 µM (results varied among donors; LOEC based on a significant effect occurring in at least one donor at all durations)	Increased IFN-γ secretion	(Almughamsi and Whalen, 2016)	High
Immune	Short-term	Human MD-PBMCs (4 donors, 3 replicates)	*In vitro*	0, 0.5, 1, 2.5 µM	24 h	Inhibitors of NF-κB and MEK ½ diminished the ability of HBCD to increase IFN-γ secretion; inhibitors of p38	Increased IFN-γ secretion: effect of inhibitors of NFκB (BAY11-7085), MEK ½ (PD98059), p38 (SB202190), and JNK (JNK B178D3)	(Almughamsi and Whalen, 2016)	High

Table 3. Summary of New Mechanistic / *In Vitro* Data for HBCD									
Target Organ/ System	**Study Type**	**Species/ Strain/Cell type (Number/ Group)**	**Exposure Route**	**Doses/ Concentrations**	**Duration**	**Effect Dose/ Concentration/ Result**	**Effect Measured**	**Reference**	**Data Quality Evaluation**
						and JNK had no effect			
Immune	Short-term	Human PBMCs, MD-PBMCs and NK cells (3-9 donors, number of replicates was not specified)	*In vitro*	0, 0.05, 0.1, 0.25, 0.5, 1, 2.5, 5 µM	24 h, 48 h and 6 days	LOEC MD-PBMCs: 5 µM (~13% of control cell viability at 6 days); No effect up to 5 µM in PBMCs and NK cells; pathway inhibitors did not affect cell viability	Cell viability with or without inhibitors of Caspase 1 (Caspace 1-inhibitor II), NFκB (BAY11-7085), MEK ½ (PD98059), p38 (SB202190), and JNK (JNK B178D3)	(Almughamsi and Whalen, 2016)	High
Immune	Short-term	Human PBMCs, MD-PBMCs and NK cells (3-9 donors, 3 replicates)	*In vitro*	0, 0.05, 0.1, 0.25, 0.5, 1, 2.5, 5 µM	24 h, 48 h and 6 days	LOEC 0.05 µM for all cell types (results varied among donors; LOEC based on a significant effect occurring in at least one donor at all durations)	Increased IL-1β secretion	(Anisuzzaman and Whalen, 2016)	High
Immune	Short-term	Human MD-PBMCs (4-9 donors, 3 replicates)	*In vitro*	0, 0.5, 1, 2.5 µM	24 h	Inhibitors of MEK ½ and p38 reproducibly diminished the ability of HBCD to increase IL-1β secretion (i.e., across donors); inhibitors of Caspace 1, NFκB and JNK had no	Increased IL-1β secretion: effect of inhibitors of Caspace 1 (Caspace 1-inhibitor II), NFκB (BAY11-7085), MEK ½ (PD98059), p38 (SB202190), and JNK (JNK B178D3)	(Anisuzzaman and Whalen, 2016)	High

Table 3. Summary of New Mechanistic / *In Vitro* Data for HBCD									
Target Organ/ System	**Study Type**	**Species/ Strain/Cell type (Number/ Group)**	**Exposure Route**	**Doses/ Concentrations**	**Duration**	**Effect Dose/ Concentration/ Result**	**Effect Measured**	**Reference**	**Data Quality Evaluation**
						reproducible effect on IL-1β secretion			
Immune	Short-term	Human monocyte-derived dendritic cells (7 volunteers, 3 replicates)	*In vitro*	0, 0.1, 1, 10, 20 µM	24 h	No effect up to 20 µM	Cell viability	(Canbaz et al., 2016)	High
Immune	Short-term	Human monocyte-derived dendritic cells (5 volunteers, 3 replicates)	*In vitro*	0, 0.1, 1, 10, 20 µM	24 h	Increased IL-8 LOEC:10 µM IL-6 and TNFα: no effect up to 20 µM	Cytokine production (IL-6, IL-8, TNFα)	(Canbaz et al., 2016)	High
Immune	Short-term	Human monocyte-derived dendritic cells (7 volunteers, 3 replicates)	*In vitro*	0, 0.1, 1, 10, 20 µM	24 h	Increased CD86 LOEC: 10 µM All other phenotypes: no effect up to 20 µM	Expression of phenotypic cell markers (CD86, CD80, CD83, CD40, HLA-DR, $CD80^+$, $CD83^+$, $CD40^+$)	(Canbaz et al., 2016)	High
Hepatic	Short-term	Human heptatoma HepG2 cells (10 replicates)	*In vitro*	0, 0.05, 0.5, 1, 5, 10 mg/L	24, 48, and 72 h	LOEC: 0.05 mg/L at 24 and 72 h (decreased)	Cell viability	(Wang et al., 2016)	High
Hepatic	Short-term	Human heptatoma HepG2 cells (6 replicates)	*In vitro*	0, 0.05, 1, 10 mg/L	24 h	LOEC: 0.05 mg/L for increased reactive oxygen species (ROS); increased catalase; decreased long-chain acyl-CoA dehydrogenase, lactate dehydrogenase, adenosine-triphosphate (ATP), $Ca2^+$-	ROS; oxidative stress markers (glutathione, malondialdehyde, total protein, superoxide dismutase, catalase); activity of metabolic enzymes (metabolomics analysis)	(Wang et al., 2016)	High

Table 3. Summary of New Mechanistic / *In Vitro* Data for HBCD									
Target Organ/ System	**Study Type**	**Species/ Strain/Cell type (Number/ Group)**	**Exposure Route**	**Doses/ Concentrations**	**Duration**	**Effect Dose/ Concentration/ Result**	**Effect Measured**	**Reference**	**Data Quality Evaluation**
						ATPase, Na^+/K^+-ATPase			
Hepatic	Short-term	Human hepatocytes LO2 (6 replicates, experiments repeated 3 times)	*In vitro*	0, 10^{-13}, 10^{-11} M	48 h	LOEC: 10^{-13} for ROS; no effect up to 10^{-11} M for cell viability and DNA single strand breaks; CYP2B6 induction (tested at 10^{-13} M only)	Cell survival, ROS and DNA single strand breaks (comet assay); expression of metabolic enzymes (CYP1A1, CYP1B1, CYP2B6)	(An et al., 2016)	High
Hepatic	Short-term	Human hepatocytes LO2 (6 replicates, experiments repeated 3 times)	*In vitro*	0, 50 µM	48 h	Decreased cell survival and increased ROS and DNA single strand breaks	Cell survival, ROS and DNA single strand breaks (comet assay)	(An et al., 2016)	High
Hepatic	Short-term	Human hepatocytes LO2 (6 replicates, experiments repeated 3 times)	*In vitro*	0, 50 µM with 48h of pretreatment with 10^{-13}, 10^{-11} M	48 h	Pretreatment with low concentrations of HBCD produced an adaptive response for cell survival, ROS and DNA single-strand breaks	Cell survival, ROS, DNA single-strand breaks (comet assay)	(An et al., 2016)	High
Hepatic	Short-term	Human hepatocytes LO2 (6 replicates, experiments repeated 3 times)	*In vitro*	0, 50 µM with 48h of pretreatment with 10^{-13}, 10^{-11} M followed by a 1-hour treatment with PI3K inhibitors LY294002 (10 µM), wortmannin	48 h	The adaptive response for cell survival, ROS and DNA single-strand breaks was eliminated by pretreatment with inhibitors of PI3K and p38	Cell survival, ROS, DNA single-strand breaks (comet assay)	(An et al., 2016)	High

Table 3. Summary of New Mechanistic / *In Vitro* Data for HBCD									
Target Organ/ System	**Study Type**	**Species/ Strain/Cell type (Number/ Group)**	**Exposure Route**	**Doses/ Concentrations**	**Duration**	**Effect Dose/ Concentration/ Result**	**Effect Measured**	**Reference**	**Data Quality Evaluation**
				(100 µM), MK-2206 (10 µM) or p38 inhibitor SB203580 (10 µM)					
Hepatic	Short-term	Human liver cells LO2 cells (3 replicates, experiments repeated 3 times)	*In vitro*	0, 10^{-7}, 10^{-6}, 10^{-5} M	24 and 48 h	LOEC: 10^{-5} M for β- and γ-HBCD at 48 h No effect up to 10^{-5} M for β- and γ-HBCD at 24 h and α-HBCD at 24 or 48 h	Cell viability	(Huang et al., 2016)	Low
Hepatic	Short-term	Human hepatoma cells HepG2 (3 replicates, experiments repeated 3 times)	*In vitro*	0, 10^{-7}, 10^{-6}, 10^{-5} M	24 and 48 h	LOEC: 10^{-5} M for β- and γ-HBCD at 24 and 48 h and α-HBCD at 48 h No effect up to 10^{-5} M for α-HBCD at 24 h	Cell viability	(Huang et al., 2016)	Low
Hepatic	Short-term	Human liver cells LO2 cells (3 replicates, experiments repeated 3 times)	*In vitro*	0, 10^{-7}, 10^{-6}, 10^{-5} M	24 hours	LOEC β- and γ-HBCD: 10^{-5} M No effect up to 10^{-5} M for α-HBCD	ROS	(Huang et al., 2016)	Low
Hepatic	Short-term	Human hepatoma cells HepG2 (3 replicates, experiments repeated 3 times)	*In vitro*	0, 10^{-7}, 10^{-6}, 10^{-5} M	24 hours	LOEC β- and γ-HBCD: 10^{-6} M LOEC for α-HBCD: 10^{-5} M	ROS	(Huang et al., 2016)	Low
Hepatic	Short-term	Human liver cells LO2 cells (3 replicates, experiments repeated 3 times)	*In vitro*	0, 10^{-7}, 10^{-6}, 10^{-5} M	24 hours	LOEC α- and β-HBCD: 10^{-6} M LOEC γ-HBCD: 10^{-5} M	DNA single-strand breaks (comet assay)	(Huang et al., 2016)	Low

Table 3. Summary of New Mechanistic / *In Vitro* Data for HBCD									
Target Organ/ System	**Study Type**	**Species/ Strain/Cell type (Number/ Group)**	**Exposure Route**	**Doses/ Concentrations**	**Duration**	**Effect Dose/ Concentration/ Result**	**Effect Measured**	**Reference**	**Data Quality Evaluation**
Hepatic	Short-term	Human hepatoma cells HepG2 (3 replicates, experiments repeated 3 times)	*In vitro*	0, 10^{-7}, 10^{-6}, 10^{-5} M	24 hours	LOEC β- and γ-HBCD: 10^{-5} M No effect up to 10^{-5} M for α-HBCD	DNA single-strand breaks (comet assay)	(Huang et al., 2016)	Low
Cancer and Endocrine	Short-term	Human LNCaP prostate cancer cells (3 replicates)	*In vitro*	0, 10^{-8}, 10^{-7}, 10^{-6}, 10^{-5} M	4 days	Increased cell growth at 10^{-8} M only	Cell viability/proliferation	(Kim et al., 2016)	High
Cancer and Endocrine	Short-term	Human LNCaP prostate cancer cells (3 replicates)	*In vitro*	10^{-8} M (co-treated with Casodex, a non-steroidal anit-androgen 10^{-9}M)	6 days	Increased cell growth blocked by anti-androgen	Cell viability/proliferation	(Kim et al., 2016)	High
Cancer and Endocrine	Short-term	Human LNCaP prostate cancer cells (3 replicates)	*In vitro*	10^{-8} M	3 and 5 days	Enhanced cell migration	Cell mobility/migration	(Kim et al., 2016)	High
Cancer and Endocrine	Short-term	Human LNCaP prostate cancer cells (3 replicates)	*In vitro*	10^{-8} M	24 and 48 h	Increased mRNA and protein expression of cyclin D1; increased protein expression of cyclin E; decreased mRNA and protein expression of p27; decreased protein levels of bax	mRNA and protein expression of cell cycle (cyclin D1, cyclin E, p21, p27), apoptosis (BCL-2, bax) and metastasis (cathepsin D) related genes	(Kim et al., 2016)	High
Respiratory	Short-term	Human bronchial epithelial cells (BEAS-2B) (3 replicates)	*In vitro*	0, 0.01, 0.1, 1, 10 µg/mL	24 h	Increased cell number at 0.1 and 1 µg/mL; decreased cell number at 10 µg/mL	Cell viability/ proliferation	(Kim et al., 2016)	High

Table 3. Summary of New Mechanistic / *In Vitro* Data for HBCD									
Target Organ/ System	**Study Type**	**Species/ Strain/Cell type (Number/ Group)**	**Exposure Route**	**Doses/ Concentrations**	**Duration**	**Effect Dose/ Concentration/ Result**	**Effect Measured**	**Reference**	**Data Quality Evaluation**
Respiratory and Immune	Short-term	Human bronchial epithelial cells (BEAS-2B) (3 replicates)	*In vitro*	0, 0.01, 1, 10 µg/mL	24 h	LOEC: 10 µg/mL for increased expression of ICAM-1, IL-6 and IL-8	Expression of proinflammatory proteins (ICAM-1, IL-6 and IL-8)	(Koike et al., 2016)	High
Respiratory and Immune	Short-term	Human bronchial epithelial cells (BEAS-2B) (3 replicates)	*In vitro*	0, 3 µg/mL in the presence of protein kinase inhibitors (10 µM) following 1h pretreatment	24 h	Protein kinase inhibitors eliminated the HBCD increases in IL-6 and IL-8	Change in IL-6 and IL-8 expression by inhibitors of p38 (SB203580), MEK (PD98059) and EGF receptor-selective tyrosine kinase (AG1478)	(Koike et al., 2016)	High
Respiratory and Immune	Acute	Human bronchial epithelial cells (BEAS-2B) (3 replicates)	*In vitro*	0, 1, 3, 10 µg/mL	24 h	LOAEL: 3 µg/mL (increased)	EGF production	(Koike et al., 2016)	High
Respiratory and Immune	Acute	Human bronchial epithelial cells (BEAS-2B) (3 replicates)	*In vitro*	0, 10 µg/mL	15 minutes	Increased	EGF receptor phosphorylation	(Koike et al., 2016)	High
Respiratory	Acute	Human bronchial epithelial cells (BEAS-2B) (3 replicates)	*In vitro*	0, 10 µg/mL	1-24 hours	Increased activation of NFκB and AP-1; no change in STAT	Activation of nuclear transcription factors NFκB, AP-1, and STAT	(Koike et al., 2016)	High

1.4 Epidemiological Study Data

Table 4. Summary of Epidemiological Study Data for HBCD

Target Organ/ System	Outcome/ Endpoint	Study Population	Exposure	Results	Reference	Data Quality Evaluation
Neurological/ Behavior	Reaction time, errors of omission, errors of commission, digit symbol total latency, forward digit span, backward digit span, and finger tapping	High school students (n=515), 13.6-17 years of age from two industrial areas or from the general Flemish population	Serum HBCD (median <level of quantification, range below quantification to 234 ng/L)	HBCD was not significantly associated with any of the neurobehavioral tests.	(Kicinski et al. 2012)	Medium
Neurological/ Behavior	Movement ABC, coordination, fine manipulative abilities, tremors, sensory integration, choreiform dyskinesia, and DCD-Q	5-6 year old boys and girls (n=62) from the Groningen-infant-compare (GIC) cohort (2001-2002)	Maternal serum HBCD at 35 weeks gestation	There was a significant correlation between HBCD and coordination, but there was no significant association with any other motor outcome.	(Roze et al. 2009)	Medium
Reproductive	Testosterone, sex hormone-binding globulin, LH, FSH, E2, and inhibin measured in serum at 3 months of age (only testosterone data was provided for HBCD)	Infant boys (n=55) from the Groningen-infant-compare (GIC) cohort in the Netherlands	Maternal serum HBCD at 35 weeks of gestation	Trend toward significant correlation between HBCD and free testosterone ($p<0.10$), but no significant correlation noted for other hormones.	(Meijer et al. 2012)	Medium
Thyroid	Thyroid-stimulating hormone (TSH) in neonates	Norwegian Human Milk Study, 2003-2006: Multi-center cohort of mothers (n=193 babies with maternal breast milk HBCD samples) recruited from six counties in Norway.	HBCD measured in breast milk; exposure stratified by quintile < 0.13, 0.13–0.52, 0.53–0.79, 0.8–1.24, and 1.29–31.2 ng/g lipid with 62 subject in the lowest quintile and 31-34 subjects in higher quintile	HBCD was not significantly associated with thyroid stimulating hormone (TSH) levels in neonates.	(Eggesbø et al., 2011)	High

Table 4. Summary of Epidemiological Study Data for HBCD						
Target Organ/ System	**Outcome/ Endpoint**	**Study Population**	**Exposure**	**Results**	**Reference**	**Data Quality Evaluation**
Thyroid	Free T3, free T4, TSH	High school students (n=515) age 13.6-17 years of age from two industrial areas (163 from Genk, 178 from Menen) or from the general Flemish population (n=174)	Serum HBCD (median <level of quantification, range below quantification to 234 ng/L)	HBCD was not significantly associated with free T3, free T4, or TSH.	(Kicinski et al. 2012)	Medium
Thyroid	Serum triiodothyronine (T3), free thyroxine (FT4), and thyroid stimulating hormone (TSH)	Mother-infant pairs of infants with hypothyroidism (n=12), Seoul, Korea, Nov 2009-May 2010, 23-37 years	Serum beta-HBCD (mean 0.461 ng/g lipid)	Mother's T3 was negatively associated with beta-HBCD concentration. Non-significant associations with all other thyroid hormone-HBCD diastereomer combinations.	(Kim and Oh 2014)	Medium

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