

Effects of Brominated Flame Retardants: Health and Regulation

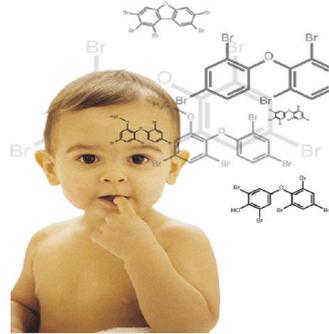
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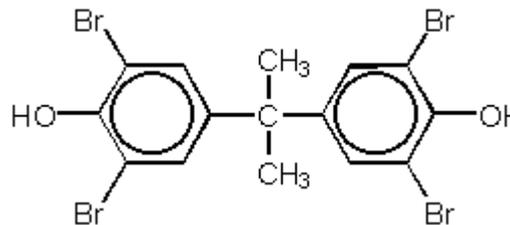
BFRs have had a lot of publicity:

found in breast milk, potential endocrine disruptors and developmental neurotoxicants.



(Tetrabromobisphenol A)

- Reactive (90%) & Additive (10%)
 - Primary use – Electronics/circuit boards
- Acute tox data – oral LD₅₀: 5-10 g/kg
- Low chronic toxicity
- Not teratogenic or mutagenic
- Limited data in biota
- Pharmacokinetics
 - Well-absorbed
 - Metabolites (glucuronides.sulfates) eliminated in bile - Parent excreted due to gut deconjugation
 - Short half-life (<2 days)



Health Effects of TBBPA

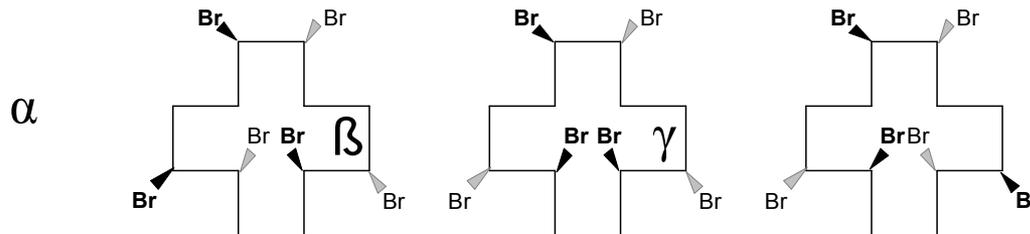
- Immunotoxic
 - Inhibits T cell activation : blocks CD25 (<3 μ M)
- Hepatotoxic
 - Toxic to primary hepatocytes: destroys mitochondria; membrane dysfunction (inhibits CYP2C9)
 - No evidence of being an enzyme inducer
- Neurotoxic
 - Oxidative Stress (Reistad et al, 2007)
 - Inhibits dopamine uptake
 - Generates free radicals
 - Increase Calcium
 - Hearing Deficits in rats – MOE~5!
 - Lilienthal et al., 2008

Health Effects of TBBPA (cont.)

Endocrine Disruption

- AhR Effects
 - Not relevant for commercial product (Contaminants? Combustion products?)
- Thyroid
 - TBBPA>T4 in relation to binding to transthyretin; some competition for TBG (Marchesini et al, 2008)
 - Thyroid Hormone Agonist, Antagonistic, Potentiating, or No Effect (Kitamura et al, 2002, 2005; Hamers et al, 2006; Schriks et al, 2006)
 - Decreased T4
- Estrogenic/Androgenic
 - Inhibits sulfotransferase (decreases estrogen clearance)
 - Developmental effects
 - increased testis and pituitary weight (Van den Ven, 2008)

Hexabromocyclododecane (HBCD)



- Additive
 - Used in Electronics; Textile Backings
 - Thermal Insulation in Buildings
- Ecotox
 - Algae, daphnia, NOEC = 3 ug/L
 - Fish, LC₅₀ > water solubility; PNEC = .03ug/L

HBCD Effects

- Mild acute toxicity, irritation, sensitization, mutagenicity (EU Commission, 2008)
- Liver Hypertrophy; Enzyme inducer (CAR/PXR)
- Repeated dose (rats) – increased liver weight, liver enzyme activity, TH effects – decreased T4, increased TSH (Chengelis, 2001; Germer, 2006; Germer, 2008; VanderVen, 2006)
- 2 gen repro study – decreased T4, increased TSH, repro effects on altered histology of ovary, decreased viability of F2 pups (Ema, 2008)
- DNT effects (mice) – spontaneous behavior, learning and memory deficits (Eriksson, 2006)
- In vitro –
 - anti-androgen; aromatase inhibitor, interactions with steroid hormone receptors (Canton, 2008; Hamers, 2006)
 - Potentiates T3 effects in rat pituitary cell line/T-screen (Schriks et al, 2006)
 - neurotoxic to rat cerebellar granule cells (Reistad et al, 2006)
 - Inhibits depolarization-evoked intracellular Ca⁺⁺increase and neurotransmitter release (Dingemans et al, 2009)

HBBD effects – *in vitro*

- Anti-androgen; aromatase inhibitor, interactions with steroid hormone receptors (Canton, 2008; Hamers, 2006)
- Potentiates T3 effects in rat pituitary cell line/T-screen (Schriks et al, 2006)
- Neurotoxic to rat cerebellar granule cells (Reistad et al, 2006)
- Inhibits depolarization-evoked intracellular Ca⁺⁺increase and neurotransmitter release (Dingemans et al, 2009)

HBCD effects –Low Dose

- One Generation Rat Reproduction Study (Van der Ven et al., 2009)
 - Decreased Bone Density
 - BMDL=0.056 mg/kg/d (females)
 - Decreased Retinoids
 - BMDL = 1.3 mg/kg/d (females)
 - Increase in Immune Response (increase in response to sheep red blood cells)
 - BMDL=0.46 mg/kg/d (males)
- Rat Developmental Neurotoxicity (Lilienthal et al., 2009)
 - Different from Effects with TBBPA or PBDE99
 - Hearing Deficit
 - Brainstem Auditory Evoked Potential (Cochlear)
 - BMDL=0.2 mg/kg/d (males)
 - Catalepsy (dopaminergic effect)
 - BMDL=0.6mg/kg/d (females)

Window of Susceptibility for HBCD

- Effects appear to be developmentally induced
- Lack of effects on TH may reflect window of susceptibility
- DNT effects may be TH related
- Development of cochlea depends on TH
- Immune effects could be related to retinoid effects

HBCD – Human Effects

- Positive Association between prenatal exposure and testis weight (Meijer et al., 2008)
- MOE for HBCD for high end humans = 180-1000
- MOE for occupational exposure = 1.5-8.2

HBCD Pharmacokinetics

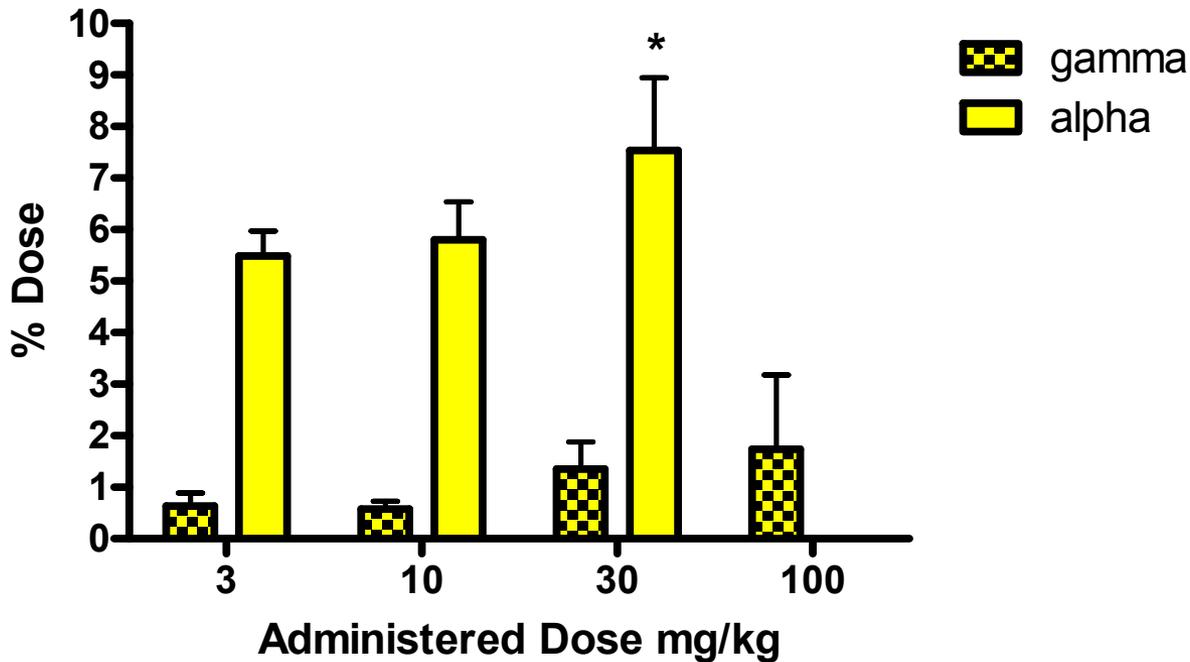
- Metabolites in Chicken Eggs and Fish (Hiebl and Vetter, 2007)
 - Pentabromocyclododecene
- Tissue Concentrations in rats 3 days after treatment (~19mg/kg, ip) (Reistad et al, 2006)
 - Brain=49 ng/g; liver=1250ng/g
 - Pattern looks like technical mixture – mostly gamma
- HBCD Commercial Mixture studies in rats (cited in NAS, 2000)
 - *Study 1* : In 72 hours, 16% of the dose in urine, 72% feces
 - High levels in the liver, kidney and lung
 - Half-life ~ 2h
 - *Study 2*: [C¹⁴]HBCD was administered daily for 5 days at 500 mg/kg
 - Fecal excretion only 33%/day
 - Levels in adipose tissue only
 - *Study 3*: Induction of hepatic CYP2B and CYP3A enzymes (CAR and PXR)
 - Inhibition of CYP1A enzymes (AhR) (Ronisz 2004).

Isomer- Specific Pharmacokinetics in Mice: HBCD γ

- Tissue disposition is not a function of dose.
- Tissue disposition was not changed after a single or repeated exposure.
- HBCD- γ is very well absorbed orally.
- HBCD- γ is rapidly metabolized and eliminated.
- *In vivo* biotransformation of HBCD- γ to HBCD- α was not detected.
- HBCD- γ has a higher body burden in infantile as compared to adult animals.

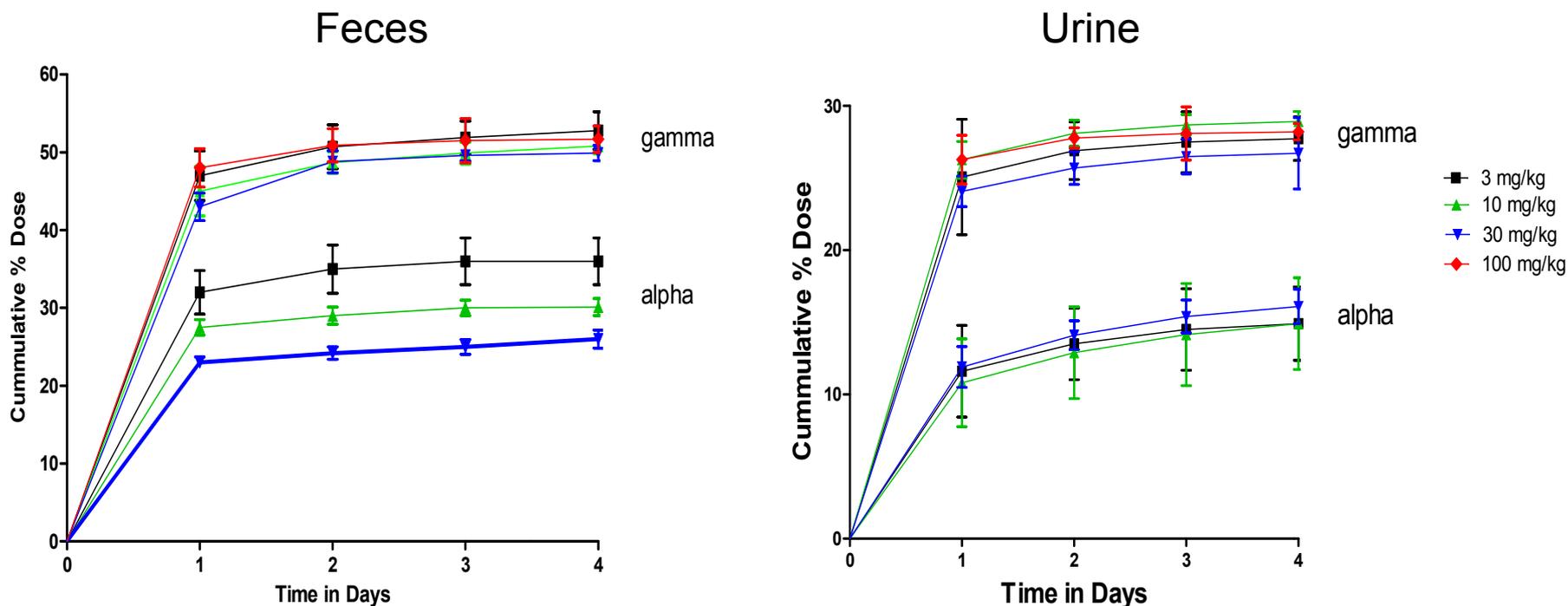
HBCD deposition in adipose tissue

single oral dose in adult C57BL/6 female mice after 4 days



[C¹⁴]HBCD- α at any dose produces a greater accumulation in fat than γ
[C¹⁴]HBCD- α shows dose-dependent disposition

Elimination of [C¹⁴]HBCD- γ and - α single oral dose in C57BL/6 mice



Less fecal and urinary elimination with alpha as compared to gamma suggests biological persistence of alpha with potential bioaccumulation.

IMPACT

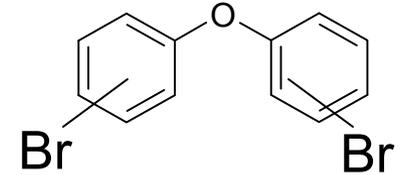
Does this suggest that all the toxicity studies of commercial HBCD under-predict the risk?



Polybrominated Diphenyl Ethers (PBDEs)

Additive BFRs

- *Deca* (DBDE) – largest volume (75% in EU)
 - 97% DBDE; 3% NBDE
 - Polymers, electronic equipment, textile backing
- *Octa* (OBDE) – *no longer made*
 - 6%HxBDE; 42%HpBDE; 36% OBDE; 13%NBDE; 2%DBDE multiple congeners (unclear if any PeBDE)
 - Polymers, esp. office equipment
- *Penta* (PeBDE) – *no longer made*
 - Flexible polyurethane foam (up to 30%)
 - **Cushions; mattresses; carpet padding**
 - Mainly PeBDE+TeBDE, some HxBDE



Ecotoxicity

- PeBDE>>OBDE>DBDE
- PeBDE - Highly toxic to invertebrates
- DE71 – endocrine disruption in *Xenopus* (0.7 μ g/l)
 - Decrease Testosterone, Increase Estradiol, Increase in phenotypic female frogs
- DE71 – developmentally toxic to fish (1ng/l)
 - Tail asymmetry; delayed hatching; behavioral changes; learning deficits
- Σ PBDEs ~ Baltic porpoise die-off (lymphoid depletion)
- BDE99 \rightarrow depletion of Vitamin E in duck eggs
- DE71 – altered reproductive behaviors in Kestrels at environmental levels (Fernie et al, 2008); decreased reproductive success (Fernie et al, 2009)
- Measured in fish, sea turtles, birds, mammalian wildlife and domestic animals
- BDE 47, 99, 100 \rightarrow decreases in T4/retinoids; increased oxidative stress in Kestrels

PBDE Toxicity in Laboratory Animals

- Hepatotoxic
 - hepatocyte hypertrophy
 - Decrease in retinyl esters (BMDL~0.5 mg/kg/d)
- Enzyme Induction
 - Cytochrome P450
 - *Purified* BDEs are *Not* CYP1A inducers
 - Induction of CYP2B,3A - Via CAR/PXR
 - UDP-glucuronyl transferase; Sulfotransferase
 - Weak Inducer
 - Transporters – Mdr1, Mrp2/3, OATP1a4

DE71 Effects on Hepatic Metabolism (Szabo et al, 2009)

TABLE 9
Summary on the Effect of DE-71 on THs, Hepatic Protein
Activity, and Gene Expression^a

Target	PND 4	PND 21	PND 60
T ₄	↓↓	↓↓	—
T ₃	—	—	—
EROD	↑↑↑	↑↑↑	—
PROD	↑↑↑	↑↑	—
BROD	↑↑	↑↑↑	—
UGT-T ₄	↑	↑	—
SULT-T ₄	—	—	—
D1-T ₄	↓↓↓	↓	—
Cyp1a1	↑↑↑	↑↑↑	↑
Cyp2b1	↑↑↑	↑↑↑	—
Cyp2b2	↑↑↑	↑↑↑	—
Cyp3a1	↑	↑↑	—
Ugt1a1	—	—	—
Ugt1a6	↑↑	—	—
Ugt1a7	↑	↑	—
Ugt2b	↑	↑↑	—
Sult1a1	—	—	—
Sult1b1	—	↑↑	—
Sult1c1	—	—	—
Mdr1	↑↑↑	↑↑↑	—
Mrp2	↑	↑↑↑	↓
Mrp3	↑↑↑	↑↑↑	—
Oatp1a4	—	↑	—
Oat2	—	—	—
Ntcp	—	—	—
Tr	—	↓	—
d1	↓↓	↓	—

Note. Overall hepatic effects after perinatal exposure to the polybrominated diphenyl ether mixture, DE-71 on THs, hepatic protein activity and gene expression. Data was measured in male rat at PND 4, 21, and 60 after perinatal exposure to 0, 1.7, 10.2, and 30.6 mg/kg/day between GD 6 to PND 21.

^aThree, two, and one arrow refers to significant effects at doses 1.7, 10.2, and 30.6 mg/kg/day, respectively.

Endocrine Disrupting Effects

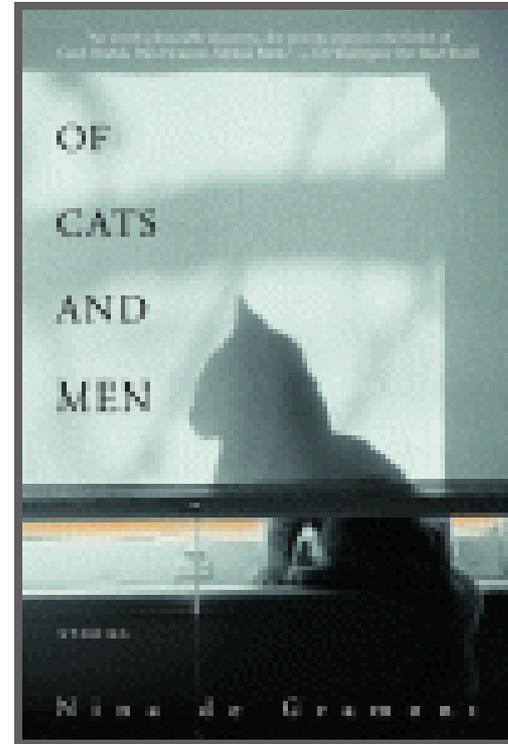
- Estrogens
 - *In vivo*
 - BDE99 – decreased E2
 - DE71 – induction of adrenal CYP17 (BMDL~0.3mg/kg/d)
 - *In vitro*
 - OH-PBDEs may be anti-estrogenic
 - **Inhibit aromatase (Canton et al, 2008)**
 - Sulfotransferase inhibition could be estrogenic
- Androgens
 - *In vivo*
 - DE71- decreased weight of epididymis, seminal vesicles and ventral prostate, decreased LH, sperm head deformities
 - BDE99 – decreased testosterone
 - *In vitro*
 - DE71, BDE100, BDE47 – antiandrogenic (non-competitive inhibition)

Endocrine Disrupting Effects (cont.)

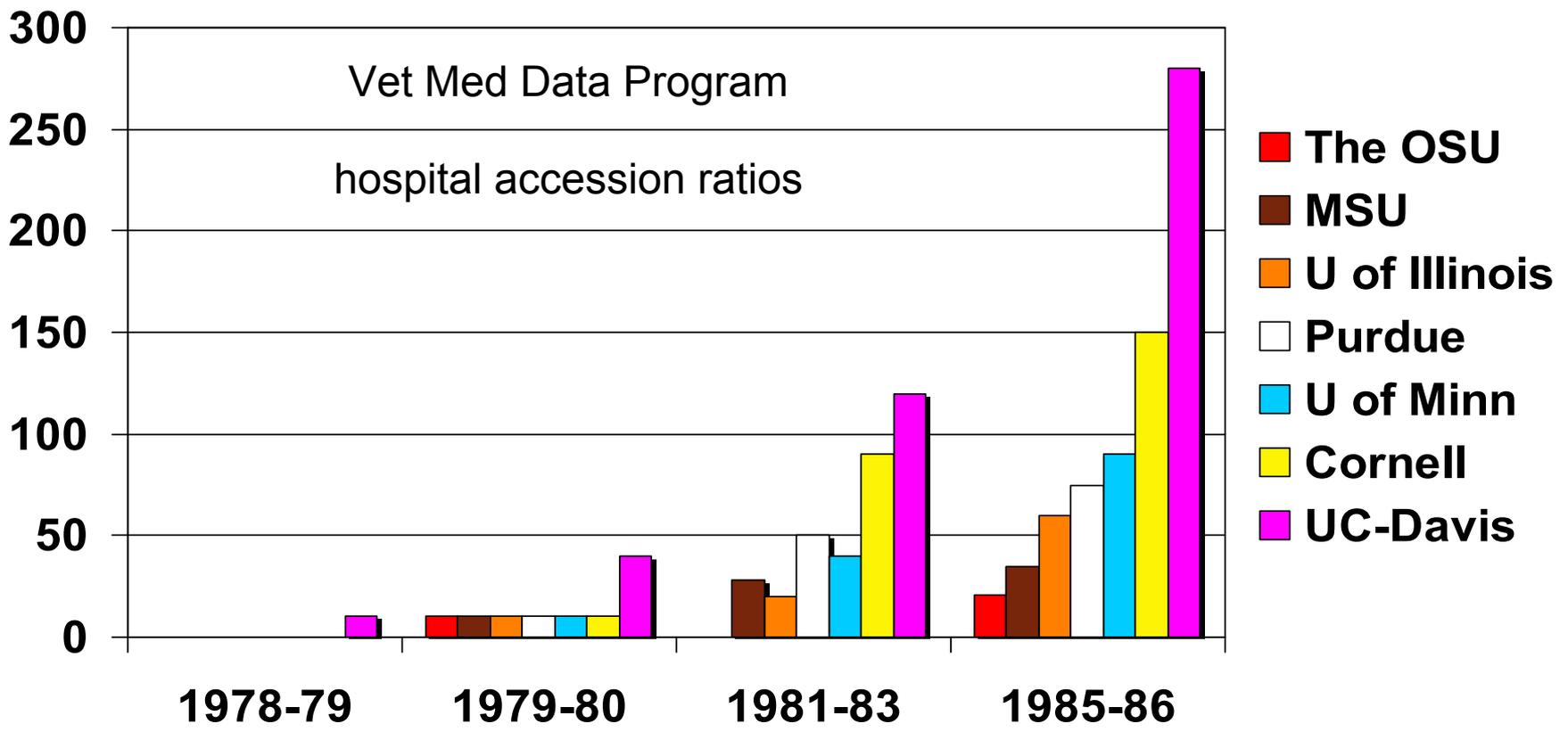
- AhR (dioxin) Effects
 - Contamination of all commercial PBDEs
 - Combustion produces PBDDs/PBDFs
- Thyroid Homeostasis
 - Decrease in T4 (DE71: BMDL~1 mg/kg/d)
 - Decrease in Hepatic Deiodinase I
 - OH-PBDE metabolites bind to serum transport proteins *in vitro*
 - Induction of TTR mRNA
 - Bind to TTR and TBG with high affinity (Marchesini et al, 2008)
 - Parent PBDEs - Effects on T4 seen *in vivo*
 - Induction of UDP-glucuronyl transferase/ sulfotransferase
 - Not a low dose effect

Hyperthyroidism

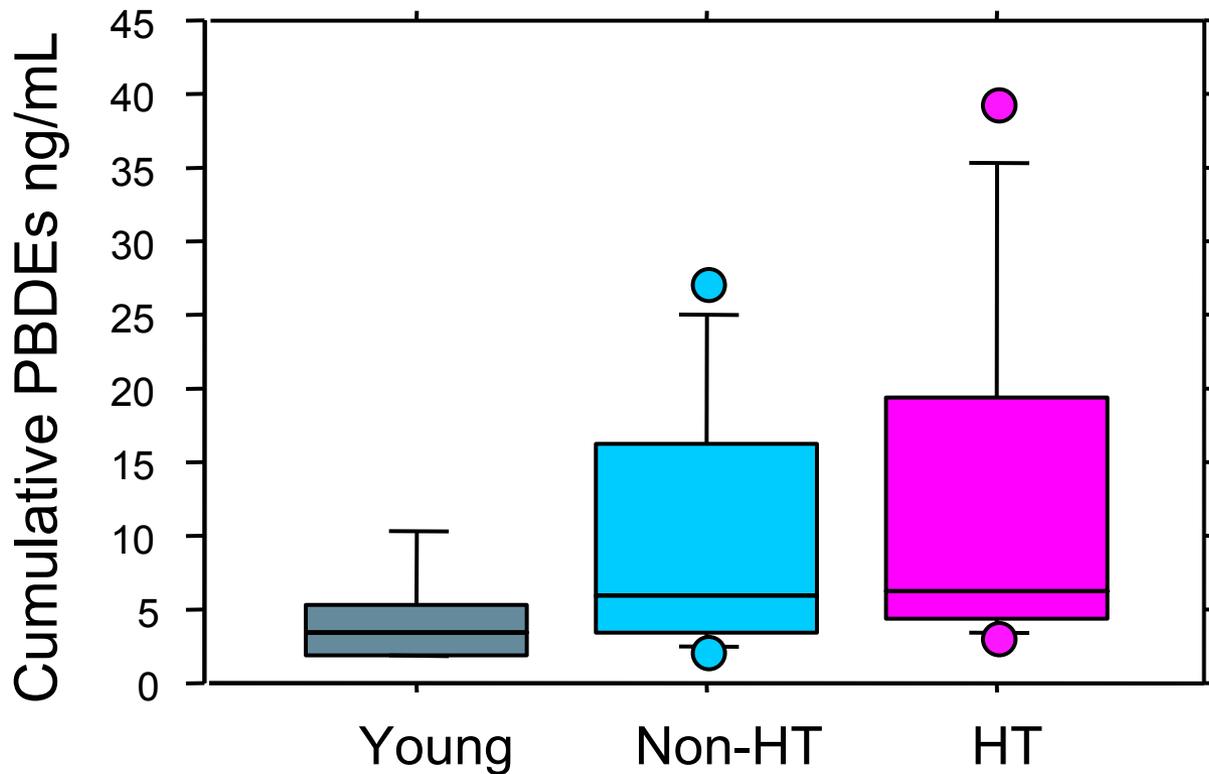
- Cats and humans
- Histologic changes
 - benign hyperplasia,
 - benign nodular hyperplasia
 - TNG, TMNG
- No evidence of auto-antibodies
 - (NOT Graves' disease)
- Age
 - Older (> 8 yr, mean = 14 yr)
- Insidious onset
- Cause – unknown.



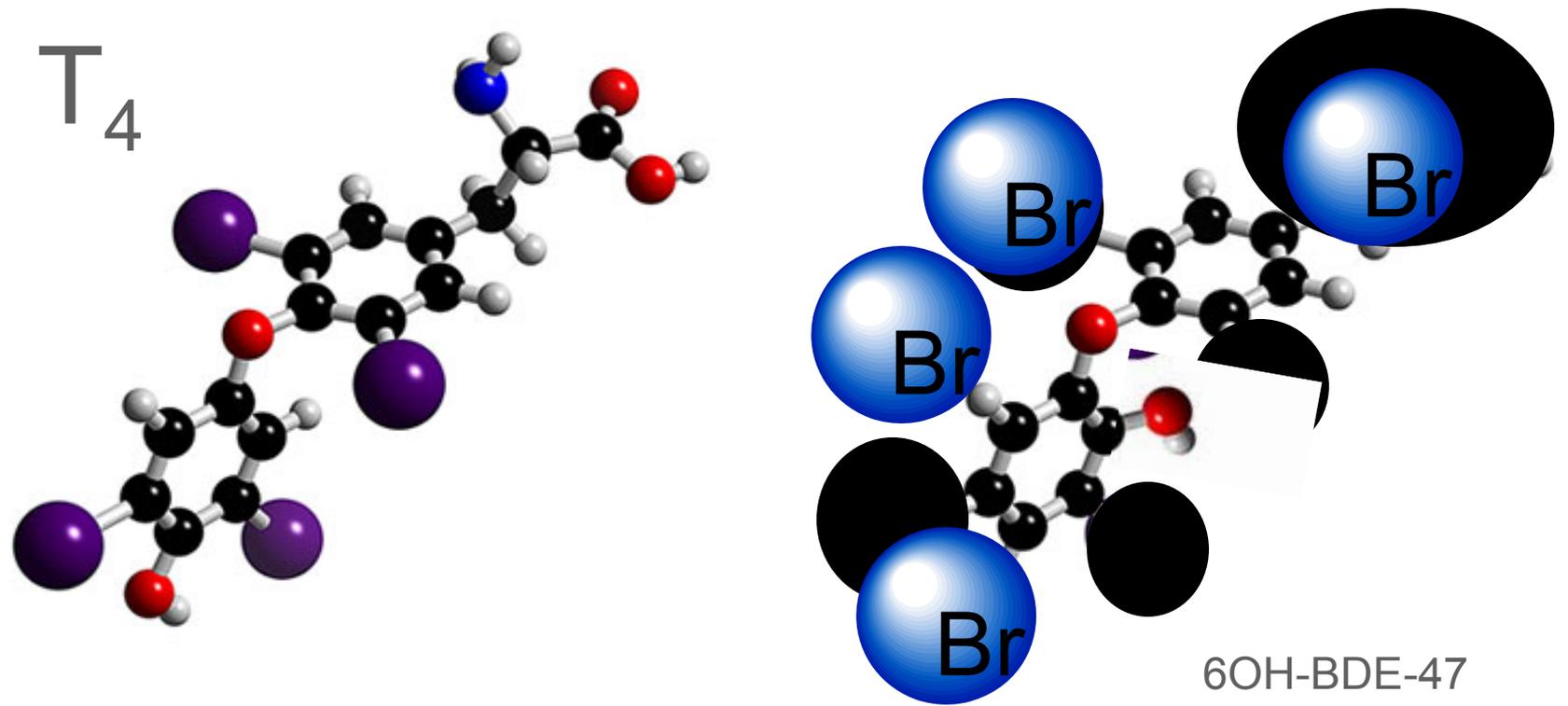
Increases in Feline HT in U.S. PHAR's (per 1000 cats)



Serum Σ PBDE levels (ng/mL) in cats based on health status.



Molecular mimicry



PBDEs are now considered “endocrine disruptors”

Developmental Reproductive Effects

- DE71– pubertal exposures
 - Delay in puberty
 - Effects on male organs
 - Anti-androgenic *in vitro*
 - esp. BDE 100,47
- BDE-99/47– *in utero* exposures
 - Delay in puberty
 - Ovarian toxicity
 - Male organ effects and decreased sperm

Developmental Neurotoxicity

- DE-71 – Rats
 - Deficits in sensory and cognitive function
 - Altered sex-dependent behaviors
 - Effects on thyroid, cholinergic, and dopaminergic systems
- BDE-99, 209 (47,153,203,206) - mice and rats
 - Infantile Exposure (“Rapid Brain Growth”) - Permanent effects on learning
 - Perinatal Exposure – Delay in sensory-motor development
- BDE-99+PCB-52 *or* PFOA *or* MeHg – Mice
 - Effects may be more than additive

Developmental Neurotoxicity of PBDEs

- Mechanisms?
 - Depression in serum T4
 - Anti-cholinergic/Anti-dopaminergic
 - Alterations in key proteins involved in normal brain maturation – GAP43, CaMKII, BDNF (Viberg et al, 2008)
 - Detrimental effects on cytoskeletal regulation and neuronal maturation (Almet et al, 2008)
 - Oxidative Stress (Giordano et al, 2008)
- PBDEs alter cell signaling *in vitro*
 - DE71, BDEs 47, 99, 153
 - Altered PKC and calcium homeostasis (associated with learning and memory)
 - Altered phorbol ester binding

Due to hydroxy metabolites? (Dingemans et al, 2008)



Outline of NIEHS/NTP DE71 Studies

- DE71 Subchronic studies - F344/N rats & B6C3F1 mice (completed; Dunnick and Nyska, 2009)
 - Primary toxicity to liver (hepatocytic hypertrophy, fatty change, single cell necrosis)
 - Thyroid effects in Rats
- DE71 in utero/postnatal/adult exposure cancer study in Wistar rats (ongoing)
- DE71 2-year traditional cancer study in B6C3F1 mice (ongoing)
- DE71 administered by oral gavage in corn oil

Trends in BDE Toxicokinetics

	209	99	47
MW	959	565	486
Log k_{ow}	10	6.7	6.5
Absorption	low (~1-50% oral, ~2-20% dermal)	moderate to high	high (>80% oral and i.t., ~60% dermal)
Distribution	blood-rich tissues	lipophilic tissues	lipophilic tissues
Metabolism	Moderate -> high(OH and deBr)	low -> moderate (OH and deBr)	low (OH-BDEs)
Excretion	high (>80% in feces (~50% metabolite)	moderate (>80% in feces)	moderate to high (species specific patterns)

Half-life estimates of PBDEs

- Deca BDE-209 in humans is on the order of 1 week
- Hepta BDE-183 is nearly 3 months
- Tetra BDE-47 is calculated to be as long as 1.8 years (range 1.4 – 2.4 yr)
- Penta BDE-99 is 3 years (range 1.8 – 4.0 yr)

Hagmar 2000



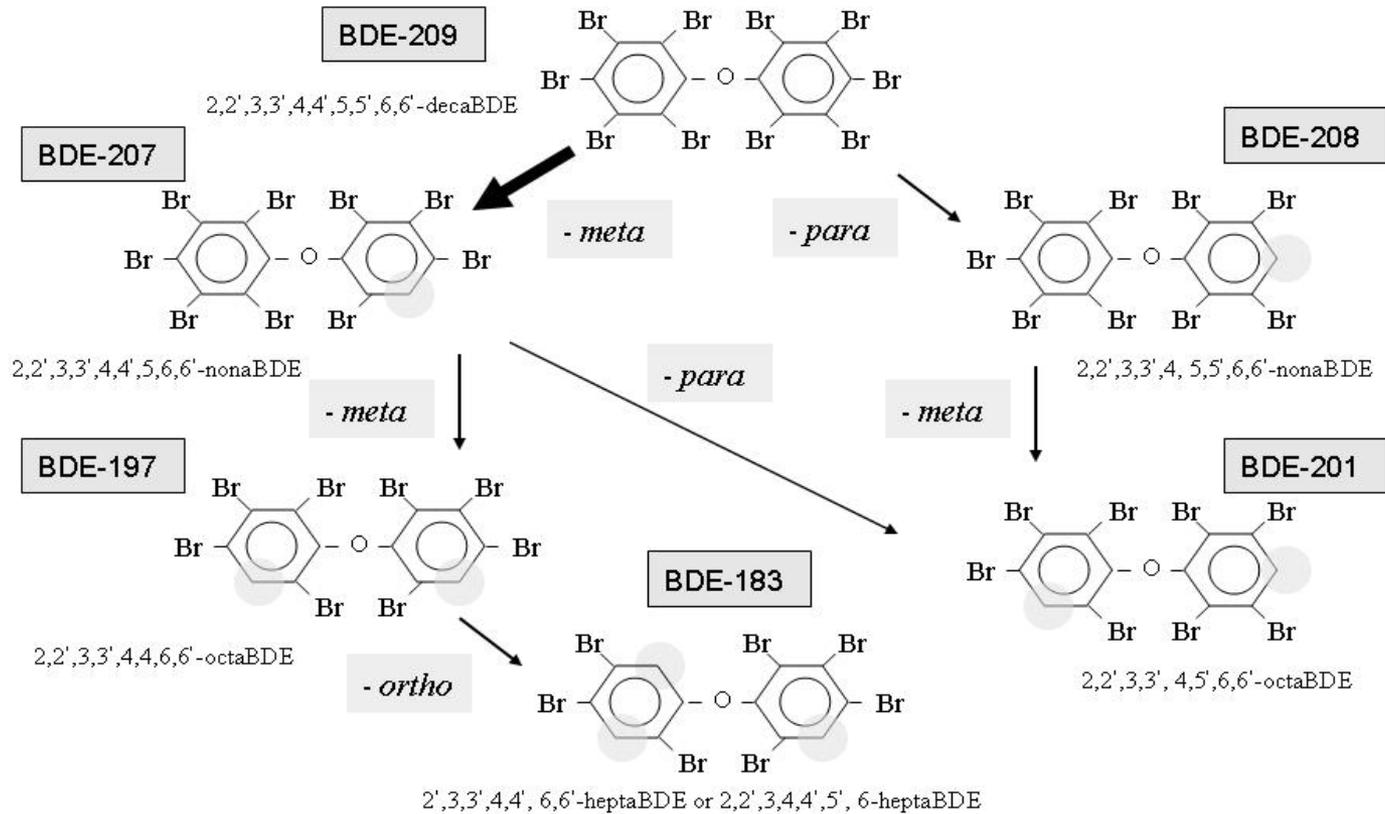
[PBDE] in adipose, liver, and plasma ~carcass lipid wt

BDE	Adipose	Liver	Plasma
47	1.00	0.22	2.11
99	1.01	0.19	2.31
100	0.94	0.26	3.66
153	0.62	0.32	3.26
154	0.72	0.15	4.23
183	0.43	0.32	4.42
197	0.34	1.15	6.62

Deca in Rats after 21 Days of Treatment (Huwe, 2005)

BDE	Total Dose Ingested (ng)	%Ingested Dose
209	78,190	4.3
208	390	22
Nona-2	130	516
203	9	45
Octa-2	4	2100

FIGURE 3. Possible debromination pathways of BDE-209 in cats.



Pharmacokinetics of BDE209

- Absorption – DBDE can be absorbed
 - Depends on Matrix (>10%)
- Distribution – Different from other PBDEs
 - Liver and Blood/NOT Fat!
- Metabolism – Extensive
 - *In vitro* by rat CYP2B (Silvia et al, 2008)
 - Debromination, hydroxylation, O-methylation
 - Reactive Intermediates
- Excretion – feces is major route
- Half Life -<3 days in rats , ~15 days in humans
 - much faster than tetra-hexa PBDEs

Deca Toxicity in Adult Rodents

- 28d Oral toxicity in Rats (van der Ven et al., 2008)
 - BMDL ~ 0.2 mg/kg
- Hepatic Effects
 - Induction of CYP2B
- Thyroid Effects
- Adrenal Effects
 - Induction of CYP17
- Deca is the **ONLY** Commercial PBDE mixture ever tested for carcinogenicity
 - Positive in 2 yr feeding study in rats and mice (NTP)
 - Liver and Thyroid

Developmental Effects of DBDE

- Developmental Reproductive Toxicity
 - Decrease in Sperm Function (*Tseng et al, 2006*)
 - Increase in Oxidative Stress
- Developmental Immunotoxicity
 - “Continuous exposure to high-dose PBDE-209 in female rats during pregnancy and lactation results in possible adverse effect on the immune function of the offspring rats.” (*Zhou et al, 2006*)
 - Changes in lymphocyte subsets
- Developmental Neurotoxicity
 - Permanent effects on behavior, learning, and memory (*Viberg et al, 2003, 2007; Rice et al, 2007*)
 - Similar to what observed with BDE-47,99,153 + several PCBs
 - Also seen with 206 and 203 (*Viberg et al, 2006, 2008*)

What causes Deca Effects?

- BDE209?
- Breakdown to lower brominated congeners?
 - Metabolic/Photolytic/Anaerobic
- Metabolism via reactive intermediates?
- PBDD/PBDF Contaminants?

PBDE Effects in People

- Cryptorchidism
 - Main et al, 2007
- Reproductive Hormone Effects
 - Meeker et al., 2009 - Decrease in Androgens and LH; Increase in FSH and Inhibin
 - Meijer et al, 2008 -Decrease in Testosterone
- Decreased Sperm Quality
 - Akutse et al, 2008
- Diabetes
 - Lim et al, 2008
- Thyroid Homeostasis
 - Yuan et al, 2008 - elevated TSH
 - Herbstman et al, 2008 – decrease in TT4
 - Turyk et al, 2007 – elevated T₄
 - Meeker et al, 2009 – elevated T₄, TBG
 - Dallaire et al, 2009 -Elevated T₃ ~BDE47

RfD values for PBDEs (IRIS, US EPA, 2008)

- **BDE 47: RfD=1.2 x 10⁻⁴ mg/kg-day** based on decreased habituation in mice in a neurobehavioral study reported by Eriksson et al 2001. Benchmark dose modeling was applied to this dataset to develop a POD (0.35 mg/kg). An UF of 3000 was then applied to develop the RfD (intraspecies variability (10), interhuman variability (10), extrapolation from subchronic to chronic (3), and database deficiencies (10).
BDE 99: RfD=1 x10⁻⁴ mg/kg-day based on rearing habituation in a neurobehavioral study reported by Viberg et al 2004. Benchmark dose modeling was applied to this dataset to develop a POD (0.32 mg/kg). An UF of 3000 was then applied (based on the UFs described for BDE 47) to develop the RfD.
BDE 153: RfD=1.5x10⁻⁴ mg/kg-day based on spontaneous motor behavior and learning ability in mice as reported by Viberg et al 2003. USEPA concluded that this was the only available study appropriate for dose-response . As such, the USEPA relied on the NOAEL of 0.45 mg/kg as the POD. As for BDEs 47 and 99, an UF of 3000 was then applied to develop the RfD.
BDE 209 RfD=0.007 mg/kg-day based neurobehavioral changes in mice as reported by Viberg et al, 2003. USEPA relied on NOAEL of 2.22 mg/kg-day as the POD and applied UFs for interhuman variability (10), interspecies variability (10), and extrapolation from subchronic to chronic exposures (3). The oral **CSF of 7x10⁻⁴ /mg/kg-day** was based on neoplastic nodules or carcinomas (combined) in the liver of male rats in a two-year bioassay conducted by the National Toxicology Program (NTP).

Potential Health Risk of PBDEs

- Top 5% of current human exposure in US - >400 ng/g lipid
 - If humans are 25% lipid, then their “dose” is ~0.1 mg/kg body weight
- Significant dose causing DRT
 - Rats ~0.06 mg BDE99/kg
- Significant dose causing DNT
 - Mice ≤ 0.8 mg BDE99/kg
 - Rats ≤ 0.7 mg BDE47/kg
- Rodent body burdens associated with DNT: ≤ 10 X higher than total PBDE body burdens in high end of general population in North America
- **Margin of exposure for PBDEs appears low or non-existent for susceptible populations**

Additional concern: are PBDEs interacting with other PBTs?

(PCBs? MeHg? PFOA?)

Regulation of BFRs

- TBBPA – not regulated
- HBCD
 - Banned in Norway
 - EU “SVHC”
- PBDEs
 - Penta/Octa Commercial Products
 - US –Voluntarily Withdrawal end of 2004
 - Bans in Several States
 - SNUR in place
 - Europe – Banned July 31, 2004
 - Use Stopped in Many EU countries ~10 years ago
 - Targeted for Elimination under the Stockholm Convention (5/9/09)
 - Deca Product
 - US – HPV
 - Banned in Washington and Maine
 - Proposed Bans in many other States
 - Canada
 - Ban upheld – 3/30/09
 - Europe
 - Banned in Sweden – Jan, 2007]
 - Banned in EU – July, 2008

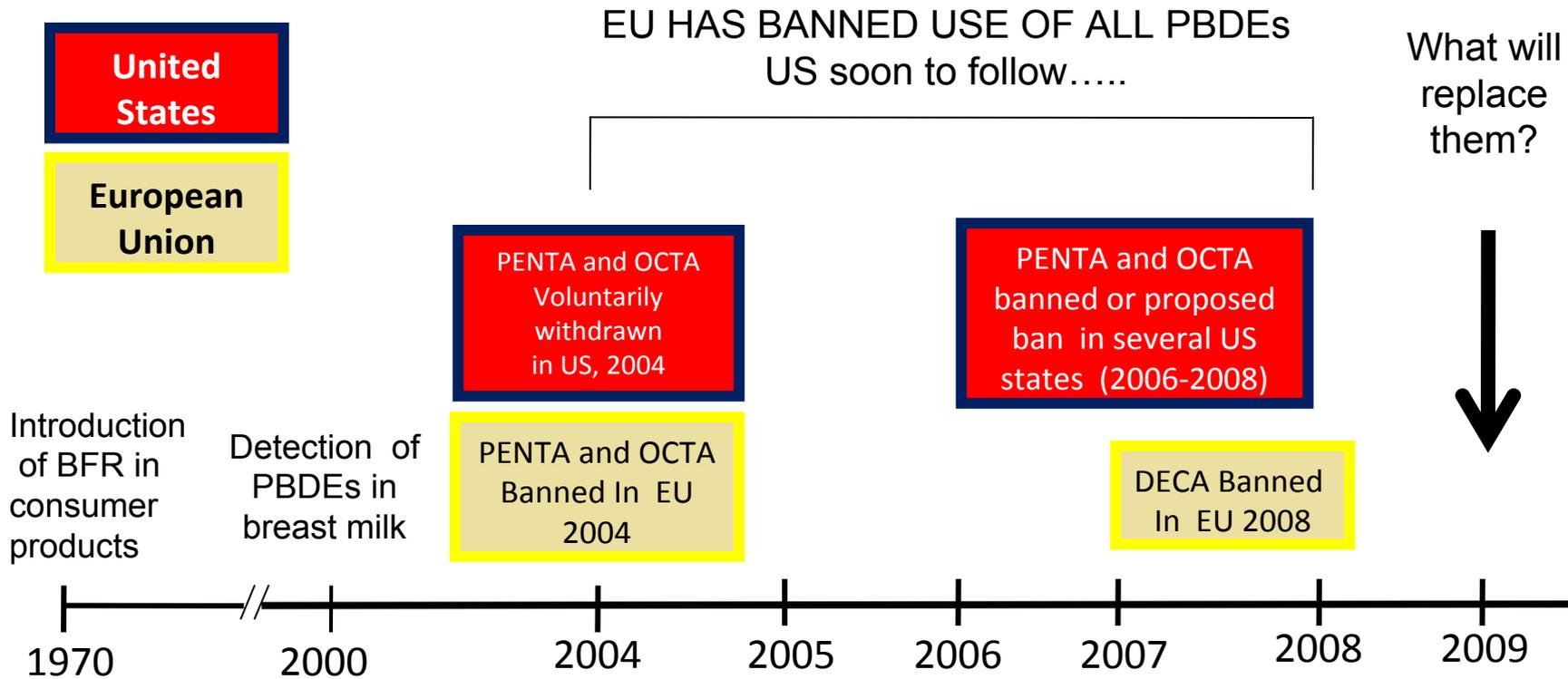
Considerations for Alternatives

- Alternative Chemicals - Other BFRs or Other classes of FRs
- Minimize potential for hazard and exposure
 - *Low persistence and bioaccumulation*
 - **for breakdown products as well as parent chemicals**
 - *Low toxicity – less potential for harm even if some exposure*
 - *Low exposure – less potential for release*
- Other Considerations
 - *Aesthetic and performance considerations: appearance, durability, fires safety*
 - *Process equipment, cost*
 - *Alternative technologies/design*
- Alternative Technologies
 - **Barriers**
 - *Surface treatments*
 - *Graphite-impregnated foams*
- **Minimize risk to human health and the environment**

THANK YOU!

- David Szabo
 - Janice Huwe
 - Janice Dye
 - Janet Diliberto
 - Dan Axelrad
-
- And...a host of other colleagues, students, and friends around the world

REGULATORY HISTORY of BFR



Ban on Deca in Canada was upheld – March 30th, 2009

PBDE Policy Developments in 2008

- Europe
 - Inclusion of Deca in RoHS reinstated – bans use in electronics
- Sweden
 - Reversed a ban on Deca in textile, furniture, and some cables
 - Challenged by EU
- Norway
 - Implemented a ban on Deca in textiles, furniture, insulation (non-transportation)
 - Also bans manufacture, import, sales of Deca
- Canada
 - Bans Deca manufacture
 - Import, sale, and use unrestricted
 - **3/29/09: Environment Canada said it would “prohibit the manufacture, use, sale, offer for sale, and import of specified new electronic and electrical products” containing Deca in amounts >0.1% by weight.**

PBDE Policy Developments in 2008 (cont.)

- POPs Treaty
 - Review committee: listing for commercial Octa should not include the octa- and nona-congeners
 - Recommend listing the hexa- and hepta-BDEs present in commercial Octa
 - BDEs 153,154,175 and 183 as markers for enforcement purposes
 - Recommendations for listing of congeners related to commercial Pena and Octa will be considered at 4th Conference of Parties, May 2009

**TARGETED FOR ELIMINATION UNDER STOCKHOLM CONVENTION:
MAY 9, 2009**

PBDE Policy Developments in 2008 (cont.)

- Washington State
 - Determined that safer, technically feasible alternatives to Deca are available for use in TVs, computers, and residential upholstered furniture
 - State Fire Marshall determined that these identified alternatives meet applicable fire safety standards
 - Public Comment on these findings through 12/17/08
 - **Restrictions on the use of Deca in these products took effect January 1, 2009**

PBDE Policy Developments in 2008 (cont.)

- Consumer Product Safety Flammability Standard
 - Proposed rule “does not rely on FR chemical filling material additives or fabric treatments, and allows the use of fire-blocking barriers, like those used in mattresses, to protect interior fillings from fire growth.”
 - CPSC preparing to conduct full scale testing on likely approaches to compliance with proposed standard