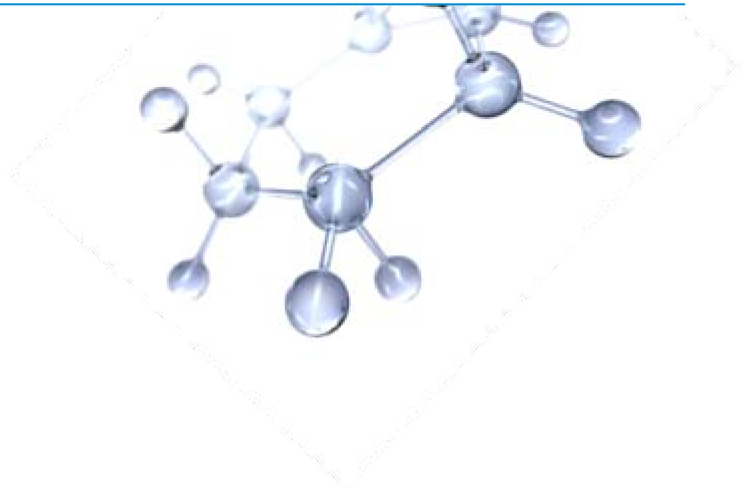
ExxonMobil

Taking on the world's toughest energy challenges.™



Approach To Cumulative Risk

U.S. Consumer Product Safety Commission
Bethesda, MD
March 23, 2010

Agenda

- Introductions
- Recap
- CPSIA CHAP Charge
- Cumulative Risk Approach
- Backup
  - Previous CPSC CHAP DINP Conclusions
  - New Reviews/Assessments
  - Differences in Phthalates

Recap

- ExxonMobil met with CPSC July 2009
  - Commitment to safety of our products
  - CPSIA support
  - Differences in phthalates and commercial uses
  - CHAP review
  - Scientific DINP/DIDP review
  - Cumulative risk assessment
- Fully support the CHAP
  - Approach to cumulative risk assessment
  - Willing to submit all available toxicological and environmental data on DINP and DIDP

CPSIA CHAP Charge

- The Panel shall complete an examination of the full range of phthalates and phthalate alternatives that are used in products for children
- And shall...
  - Examine health effects from full range of phthalates
  - Consider health effects from each phthalate and in combination
  - Examine exposure levels of all phthalates in humans and subpopulations (i.e. children and pregnant women) from children’s products
  - **Consider cumulative effect of total exposure to all phthalates in children’s products and in other products**
  - Review all relevant and objective studies of phthalates and phthalate alternatives
  - Consider health effects from exposure to phthalates from all exposure sources in addition to ingestion
  - Consider safe level of phthalates for humans and subpopulations
  - Consider health effects from phthalate alternatives

Overview

- Cumulative risk assessment: the accumulation of risk from multiple chemical and/or non-chemical stressors that may interact to produce an additive, synergistic, or antagonistic effect
- Aggregate risk assessment: the sum of the risks resulting from exposures to the same chemical via multiple sources and multiple routes
- Chemical mixtures risk assessment is encompassed within cumulative risk: two or more chemicals are involved which may cause the same or different effects to a target population (e.g., different organophosphates with the same mode of action)

Cumulative Risk

- Scientific community has several definitions of cumulative risk
  - EPA and World Health Organization's International Program on Chemical Safety - cumulative risk for categories of structurally-related chemicals which share a common mode of action
  - US National Research Council (NRC, 2008, 2009) –
    - 2008 – recommends assessing all chemicals showing common adverse health outcomes; these chemicals would not need to act through a common mechanism of toxicity
    - 2009 – recommends the incorporation of interaction between chemical and non-chemical stressors in cumulative risk assessments
- Data Requirements vs. Available Data
  - Given the state of the science in cumulative risk assessment; there exists no methodology at present to incorporate comprehensive cumulative risk, including chemical and non-chemical stressors, as a routine component of chemical analysis

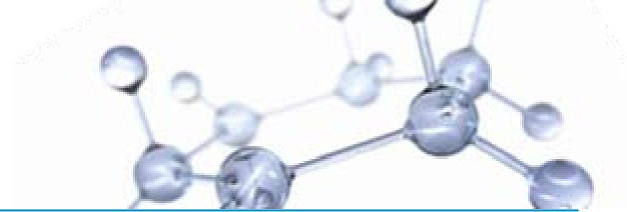
Cumulative Risk, cont.

- Existing Methodology Overview
  - Hazard Index (HI) using reference and benchmark doses
  - Margin of Exposure (MoE) using Toxicity Equivalency factor (TEF)
  - Biologically based assessments

- ExxonMobil Suggested Methodology
  - Only for purposes of the CPSIA direction to consider cumulative effects, ExxonMobil suggests a modified HI approach as providing a conservative (overestimate of risk) approach

- Results
  - Hazard index approach clearly demonstrates that even for highly sensitive populations such as children and women of reproductive age, phthalates do not pose a cumulative risk for the demonstrated endpoint. As the HI methodology likely overestimates risk, further efforts to develop a more complex assessment are not justified

Accepted Approaches for Well Defined Mixtures



- Hazard Index Approach
  - Advantages
    - defined, transparent methodology; extensive mechanistic research data are not needed; uncertainty is well incorporated
  - Limitations
    - A common mode of action is not required, only a defined endpoint effect; dose addition is assumed at low levels; does not account for toxicokinetic and toxicodynamic differences; relative potency is not determined
- Toxic Equivalency Factor Approach
  - Advantages
    - A mode of action is defined for the mixture; potency is incorporated; can be used as a combined approach with the hazard index methodology
  - Limitations
    - Assumes no significant interactions among the chemicals; requires confidence in a single endpoint effect and associated parameter; determination of most potent/toxic compound can be subjective
- Biologically Based/ Physiologically Based Pharmacokinetics Approach
  - Advantages
    - Highly comprehensive; lower uncertainty
  - Limitations
    - Highly data intensive and reliant on extensive mechanistic research

Recently Proposed Approaches

- Benson, 2009 -- Employed relative potency factor/hazard index approach for six phthalates demonstrating that humans are likely not suffering adverse developmental effects from current environmental exposure to the mixture
  - Advantages
    - Transparent methodology; mixture composition was based on a common mode of action; potency was considered
  - Limitations
    - Common toxic effect was broadly defined; calculation of point of departure (POD) was inconsistent
- NRC 2008, 2009 -- Recommended that phthalates and all other chemicals that affect male reproductive development in animals be assessed. Includes consideration of chemical and non-chemical stressors (e.g. psychosocial risk, dietary, physical factors), all routes and pathways of exposure, and varying susceptibilities of the population (burden of disease)
  - Advantage
    - All encompassing assessment of probability for adverse outcome of interest
  - Limitations
    - Highly complex; no currently acceptable methodology; time intensive; data intensive

Objectives

- Meet the charge defined in the CPSIA to consider cumulative health effect of total exposure to all phthalates in children’s products and in other products.
- Employ a currently accepted method using available objective hazard and exposure data
- Understand which phthalates drive the toxicity of the mixture and the likelihood of an adverse effect from the mixture based on the predicted exposures to the chemicals
- Focus on sensitive subpopulations
  – children, especially those mouthing toys
  – women of reproductive age

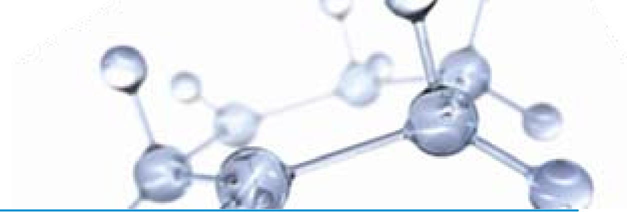
Proposal

- Use the Hazard Index Approach, a currently accepted methodology, to conduct a practical screening assessment for mixture toxicity
  - Identify phthalates that likely drive the toxicity of the mixture
- For simplicity of the example, first conduct the assessment on those phthalates named in the CPSIA: DBP, DEHP, BBP, DIDP, DINP, DnOP.
  - A focused cumulative risk assessment, limited to those phthalates named in the CPSIA will help inform whether further assessment is needed
- Use common toxicological endpoints such as repeated dose effects (i.e. increased liver weight and Palmitoyl CoA induction)
- Use either biomonitoring data to calculate estimated exposures or indirect exposure estimates for the populations of interest
- If indications of risk are identified, develop data necessary to conduct a more extensive assessment

Hazard Index Approach

- Overestimates risk; first-pass screen for mixture toxicity to determine whether more extensive assessment is necessary
- Levels of conservatism toward overestimating risk
  - Dose-addition – Since a complete dose-response assessment for the phthalates of interest is lacking, it is assumed that dose addition occurs across the entire dose-response continuum
  - NOAEL/LOAEL – Point estimates do not represent equi-effective doses
  - Modified Points of Departure – adjustment factors used in the calculation of MPOD are quantitative judgments of qualitative deficiencies in the database

Hazard Index Approach



- Identify common toxicological endpoint
- Define the phthalate mixture
- Evaluate evidence and quality of data
- Identify point of departure (NOAEL or LOAEL)
- Develop a Modified Point of Departure (MPOD)
- Establish exposure estimates
- Calculate the hazard quotient for each phthalate
- Sum the hazard quotients to calculate the hazard index

Identify Endpoint

- Endpoints should be chosen based on the commonality of the endpoint, availability of adequate published data, and toxicological concern

Endpoint	Key Data	Observed	Not Observed	No Data
Repeated Dose Effects/ Peroxisome Proliferation[1]	Increased Liver Weight, Increase in Palmitoyl CoA activity	DBP, BBP, DEHP DINP, DIDP, DnOP		
Male Reproductive/ Developmental Indicators[3]	Decrease in Anogenital Distance	DBP, BBP, DEHP	DINP[2], DIDP	DnOP
	Nipple Retention	DBP, BBP, DEHP	DINP[2], DIDP	DnOP
	Alterations in the weight of sexual organs and accessory glands	DBP, BBP, DEHP	DINP, DIDP	DnOP
	Decreased Testosterone	DBP, BBP, DEHP	DINP[2]	DIDP, DnOP

[1] Peroxisome Proliferation is not considered a relevant endpoint for assessment of human risk. For the purposes of demonstrating cumulative risk methodology it is included
[2] Limited study data available
[3] Significant reproductive and developmental adverse effects are only observed for low molecular weight phthalates (DEHP, BBP, DBP) and NOT for high molecular weight phthalates (DINP, DIDP)

Define the Phthalate Mixture

Evidence for Endpoint of Interest?
e.g. *Repeated dose toxicity*

No → **Exclude from CRA**

Yes ↓

Available Key Data using relevant, objective studies?
e.g. *Increased liver weight*

No → **Exclude from CRA**

Yes ↓

Adequate Exposure Data?

No → **Exclude from CRA**

Yes ↓

Include in CRA

Determine POD and Develop MPOD

- Application of uncertainty Factors

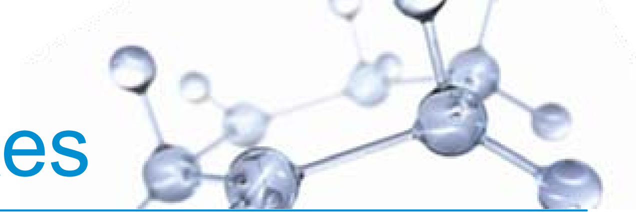
Source of Uncertainty	Uncertainty Factor
Interspecies Differences	10
Intraspecies Differences	10
Use of a LOAEL	3
Subacute to Chronic Adjustment [1]	6
Subchronic to Chronic Adjustment [2]	2

[1, 2] Only applicable to the Repeated Dose endpoint

- Calculation of MPOD

REPEATED DOSE EFFECTS – Increased Liver Weight and PCoA Activity					
Key Effect	POD (mg/kg/d)	POD Type	Uncertainty Factors	MPOD	Reference for POD
DBP	152	NOAEL	200	0.76	EU Risk Assessment, 2004
BBP	639	LOAEL	1800	0.36	EU Risk Assessment, 2007
DEHP	37.6	NOAEL	200	0.19	EU Risk Assessment, 2006
DINP	88	NOAEL	100	0.88	EU Risk Assessment, 2003
DnOP	36.8	NOAEL	200	0.18	Poon et al., 1996
DIDP	150	NOAEL	200	0.75	EU Risk Assessment, 2003 Hazelton Laboratory, 1968

Establish Exposure Estimates



- Biomarker Exposure Estimates (CDC NHANES Data)
  - Calculate daily intake (ug/kg/d) of diester from creatinine corrected urinary metabolite levels for each of the phthalates except DIDP and DnOP for the 50th and 95th percentiles
  - Populations of interest
    - + Children ages 6 – 11 yrs
    - + Females ages 15 – 44 yrs
    - + Total Population 6+ yrs
- Indirect Exposure Estimates (Clark et al., 2009, unpublished)
  - Use of the concentration of the phthalate ester in each medium of exposure and the rate of intake of that medium to quantify exposure
  - Populations of Interest
    - + Children ages 5 – 11 yrs
    - + Toddlers ages 6 months – 4 yrs
    - + Adults ages 20+ yrs
- Exposure from Toys
  - Used estimated oral exposure (99th percentile) to DINP from soft plastic toys based on mouthing and migration studies (Babich et al., 2004)
  - This estimated exposure was added to daily intake estimates of toddlers ages 6 mos – 4 yrs

Biomarker Exposure Estimates

- 3rd and 4th Centers for Disease Control and Prevention (CDC) reports based on National Health and Nutrition Examination Survey (NHANES) data– measurement of phthalate ester metabolites in urine to back calculate exposure to the parent diester
- David, 2000; Kohn et al., 2000 Methodology

DI = (UC[1] x CE[2] / (F_{ue}[3] x 1000)) x (MWd/MWm)

where:

DI	**=**	**Daily intake (µg/kg/day)**
UC	**=**	**Urinary concentration – creatinine corrected (µg/kg)**
CE	**=**	**Creatinine excretion (mg/kg/day)**
F_{ue}	**=**	**Fractional urinary excretion of the metabolite (unitless)**
MWd	**=**	**Molecular weight of the diester**
MWm	**=**	**Molecular weight of the metabolite**

[1] Urinary concentrations were for the phthalate's respective monoester
[2] Constants were used for total population (20 mg/kg/day), children (11 mg/kg/day), and females (18 mg/kg/day)
[3] F_{ue} values were derived from several published studies concerning the metabolism of phthalates

Biomarker Exposure Estimates

- Exposure calculations based on monoester data mainly from the CDC 4th report (2003-2004 data)
- Calculated Daily Intake (ug/kg/d) – 95th Percentile

Phthalate	Children (6 – 11 yrs)	Females (15 – 44 yrs)	Total Population (6+ yrs)
DBP	2.7	3.4	3.3
BBP	4.2	1.9	2.3
DEHP	7.5	14.6	12.1
DnOP[1]	1.5	1.4	1.4
DINP	2.4	2.9	3.9
DIDP[2]	2.4	2.9	3.9

[1]DnOP data only available in CDC NHANES 1999-2000 data set
[2]DINP data used for DIDP (Silva et al., 2007)

ExxonMobil
Taking on the world's toughest energy challenges.

Indirect Exposure Estimates

- Clark, 2009 unpublished: Use of the concentration of the phthalate ester in each medium of exposure and the rate of intake of that medium to quantify intake of the phthalate ester

$$D = \sum (Ci \times IRi \times Ai / BW)$$

where:

D	=	Absorbed dose of PE (µg/kg/d)
Ci	=	Concentration of PE in medium (µg/g)
IRi	=	Intake rate of medium (g/d)
Ai	=	Absorption factor (unitless)
BW	=	Body weight (kg)

- Calculated Daily Intake (ug/kg/d) – 95th Percentile

Phthalate	Toddler (0.5 – 4 yrs)	Children (5 – 11 yrs)	Adult Population (20+ yrs)
DBP	12	8.1	3.0
BBP	6.1	4.0	1.4
DEHP	124	81	31
DnOP	ND	ND	ND
DINP	8.7	5.5	2.0
DIDP	ND	ND	ND

ND = No data

Indirect Exposure Estimates

- Additional DINP Exposure from Mouthing of Toys
- Estimated oral exposure (99th percentile) to DINP from soft plastic toys based on mouthing and migration studies (Babich et al., 2004)
  - This estimated exposure (1.5 ug/kg/d) was added to the DINP daily intake estimate of toddlers ages 6mos – 4 yrs
- Calculated Daily Intake (ug/kg/d) – 95th Percentile

Phthalate	Toddler (0.5 – 4 yrs)	Toddler (0.5 – 4 yrs) + DINP Exposure from Toys
DBP	12	12
BBP	6.1	6.1
DEHP	124	124
DnOP	ND	ND
DINP	8.7	10.2
DIDP	ND	ND

ND = No data

Phthalate Mixture

- Repeated dose data for the effect of increased liver weight and increased Palmitoyl CoA activity is available for all 6 phthalates
- Exposure data is not available for DIDP; however, DINP exposure was used as a conservative estimate of exposure to DIDP (Silva et al., 2007)
- DnOP total population exposure estimate was used for the female 15-44 yrs population

Population	Phthalates Included in Cumulative Risk Assessment
Biomarker-Based Exposure Estimate	
Children (6 – 11 yrs)	DBP, BBP, DEHP, DINP, DnOP, DIDP
Females (15 – 44 yrs)	DBP, BBP, DEHP, DINP, DnOP, DIDP
Total Population (6+ yrs)	DBP, BBP, DEHP, DINP, DnOP, DIDP
Indirect Exposure Estimate	
Toddlers (6 mos – 4 yrs)	DBP, BBP, DEHP, DINP
Children (5 – 11 yrs)	DBP, BBP, DEHP, DINP
Total Population (20+ yrs)	DBP, BBP, DEHP, DINP

Calculate Hazard Quotient

- The hazard quotient (HQ) is a ratio of the expected exposure to a chemical compared to the modified point of departure (MPOD) value for that chemical
  - HQ = Exposure metric / MPOD

Phthalate Ester	Using Bio Marker Based Exposure			Using Indirect Exposure		
	MPOD	Exposure 95th Percentile (mg/kg/day) Children 6 – 11 yrs[1]	HQ	MPOD	Exposure 95th Percentile (mg/kg/day) Children 5 – 11 yrs[2]	HQ
DBP	0.76	0.0027	0.0036	0.76	0.0081	0.0107
BBP	0.36	0.0042	0.0118	0.36	0.0040	0.0113
DEHP	0.19	0.0075	0.0399	0.19	0.0810	0.4309
DINP	0.88	0.0024	0.0026	0.88	0.0055	0.0063
DIDP	0.75	0.0024	0.0037	0.75	ND	ND
DnOP	0.18	0.0008	0.0045	0.18	ND	ND

[1] Children as defined by the CDC NHANES dataset are 6-11 yrs
[2] Children as defined by the Clark, 2009, unpublished data are 5-11 yrs

ND = No data

Calculate Hazard Index

- The hazard index (HI) for a mixture is calculated by taking the sum of the hazard quotients for the individual compounds present in the mixture.
  - If values are less than or equal to 1, then the risk is acceptable and no additional risk management measures are required
  - HI = Σ(HQ)i (i for n chemicals in set)

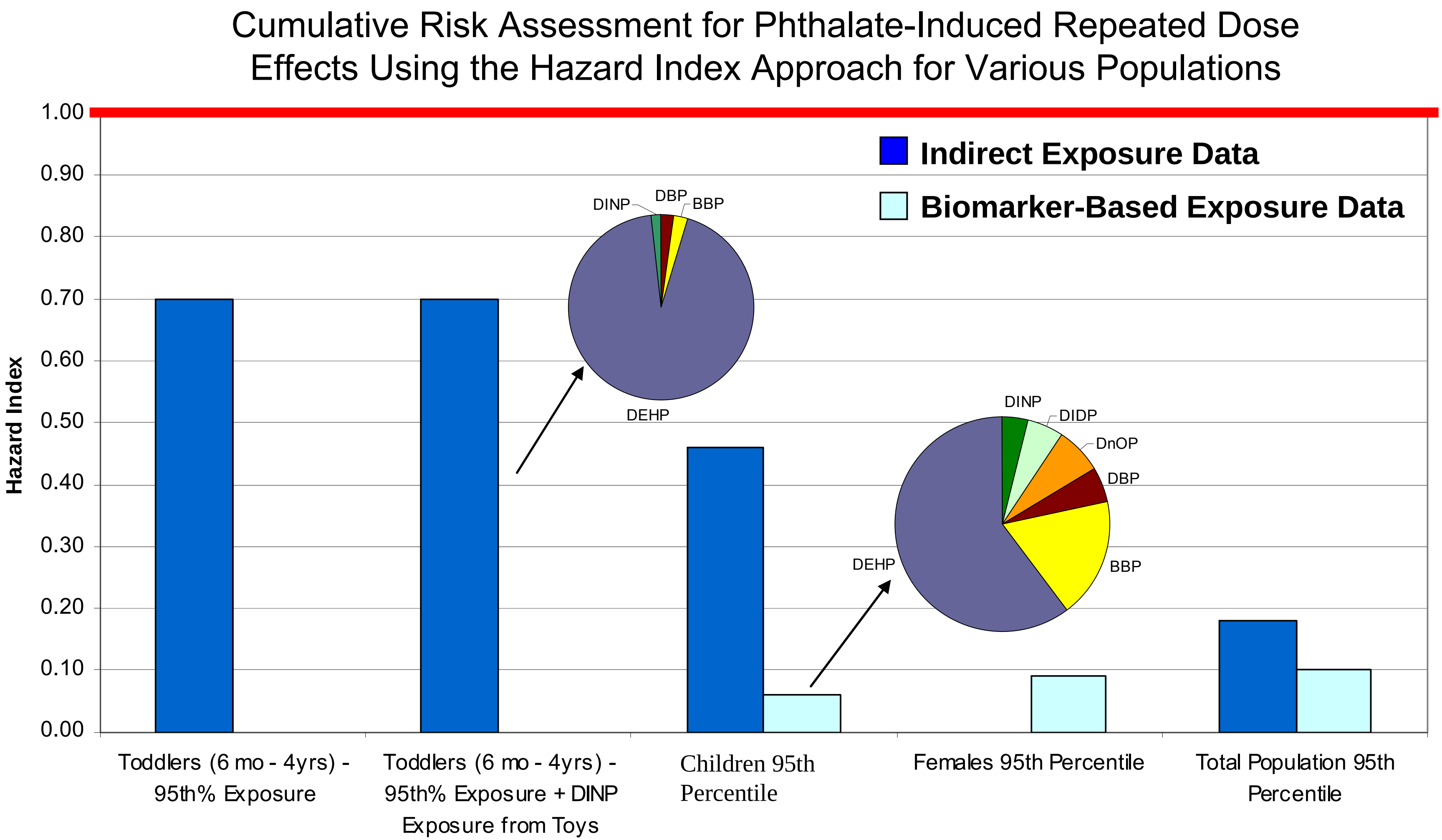
Repeated Dose Effects	**Toddlers (6 mo - 4yrs) 95th%**	**Toddlers (6 mo – 4yrs) 95th% + DINP Toy Exposure (12-23 mos.) 99th%[1]**	**Children 95th%[2]**	**Females (15 – 44yrs) 95th%**	**Total Population[3] 95th%**
Indirect Exposure	0.70	0.70	0.46		0.18
Biomarker-Based Exposure			0.07	0.1	0.09

[1]The 99th percentile estimated mean exposure to DINP is 1.5 μ g/kg/day for children aged 12- 23 months (Babich *et al.*, 2004). This exposure estimate has been added to the toddler (6 months – 4 yrs) DINP exposure estimate from Clark, 2009, unpublished data

[2] Children as defined by the CDC NHANES dataset are 6-11 yrs. Children as defined by the Clark, 2009, unpublished dataset are 5-11 yrs

[3] Total Population as defined by the CDC NHANES dataset is 6-60 yrs. Adult population as defined by the Clark, 2009, unpublished dataset are 20+ yrs

Summary of Results



Conclusions

- The HI approach provides a conservative determination of potential risk
  - Overestimates risks due to conservatism, design and assumptions
- For phthalate esters, no concern from cumulative risk assessment observed for repeated dose effects
  - Assessment conservative in that
    - + Default uncertainty factors accounted for in calculation
    - + Generally in rodents, MPOD for repeated dose effect is lower than that for other effects
    - + Effect not relevant to humans
    - + Use of indirect exposure artificially increases the exposure estimate; biomonitoring data is more representative of total exposure to phthalates including that from consumer products
- Exposure to DINP from mouthing of toys does not substantially impact HI

Backup

CHAP/CPSC History

- CPSC staff risk assessments in 1998 concluded that few, if any, children are at risk of organ toxicity from mouthing teethers, rattles and toys made of DINP-plasticized PVC
- CPSC recommended convening a CHAP to evaluate whether there are chronic hazards from exposure to DINP, and conducting additional studies to better define potential exposure to DINP
- In Nov 1998, NGOs submitted request to CPSC to ban PVC in products for children 5 years of age and under, and issue a national advisory on health risks of vinyl toys
- Commission voted to convene CHAP in Dec 1998
- CHAP met three times in 2000 and submitted a report in June 2001

Previous CHAP Determination

- Conclusions
  - Cancer/Tumors -- Although DINP is clearly carcinogenic in rodents inducing liver tumors in rats and mice of both sexes, kidney tumors in male rats and mononuclear cell leukemia in male and female rats, the human risk from cancer induced by DINP is **negligible or non-existent.**
    - + Liver cancer -- DINP causes liver cancer by a mechanism known as peroxisome proliferation. The peroxisome proliferator-activated receptor α (PPARα) mediated mechanism of hepatocarcinogenesis is pronounced in rodents, but believed not readily induced in humans, especially at the doses resulting from current use of consumer products.
    - + Kidney tumors -- The male rat kidney tumors were viewed as rat specific since they met the criteria for supporting an α2μ-globulin mechanism of action, a mechanism accepted as unique to male rats. They were not used to predict human risk.
    - + MNCL -- Mononuclear cell leukemia in Fischer 344 rats was viewed of questionable significance and was not used in human risk prediction.
    - + Spongiosis Hepatis (liver lesions) -- No observed adverse effect levels identified in laboratory animals exposed to DINP
  - Genotoxicity – DINP is **not genotoxic**.
    - + Majority of data indicate DINP is non-genotoxic, consistent with results from other peroxisome proliferators. Bacterial mutation assays, mammalian gene mutation, in vivo and in vitro cytogenetic assays, and in vitro analysis of unscheduled DNA synthesis in rat hepatocytes all show no evidence of mutagenicity or genotoxicity.
  - Reproductive/Development Toxicity – The risk to reproductive and developmental processes in humans due to DINP exposure is **extremely low or non-existent**.
    - + Large margin between dose to pregnant women and those expected to be without effect in the animal assays.

Previous CPSC Review

- CPSC conducted a state-of-the-art mouthing study and migration studies
- Using its exposure data and the very conservative Acceptable Daily Intake (ADI) from the DINP CHAP report, CPSC staff conducted a worst-case risk assessment
  - “With this worst case analysis, even the 99th percentile exposure would not exceed the acceptable daily intake (ADI)”
  - “The staff concluded that oral exposure to DINP from mouthing soft plastic toys, teethers, and rattles is not likely to present a health hazard to children. Since children mouth other children's products less than they do toys, teethers and rattles and since dermal exposure is expected to be minimal, staff does not believe that other children's products are likely to present a health hazard to children”
- The Commission voted to deny the petition to ban PVC in children’s products and issue a national advisory

What's New Since Then

- 2003 National Toxicology Program's Center for the Evaluation of Risks to Human Reproduction determined
  - DINP presents "minimal concern" for both developmental and reproductive adverse effects
  - DIDP presents "minimal concern" for developmental adverse effects and "negligible concern" for reproductive adverse effects
- 2003 European Chemical Bureau's Risk Assessment report concluded "no risk reduction required" for DINP & DIDP
- 2004 Organization for Economic Cooperation and Development's HPV program concludes HMW phthalates are "low priority for further work"
- 2005 US CDC study showed that the general population has low ppb levels of DINP metabolites in urine
  - Recent research shows ppb levels for both DINP and DIDP metabolites - indicative of exposure well within safe limits
- 2006 Oslo-Paris North-East Atlantic Commission for protection of the marine environment concludes "DINP and DIDP are not PBT substances and "there is no indication of potential for endocrine disruption"
- Toxicological Literature search by ExxonMobil Biomedical Sciences (EMBSI) since 2002 indicates no new science to shift the opinion on DINP or DIDP
  - 31 studies on HMW phthalate (DINP/DIDP/DPHP) toxicology. References submitted by ExxonMobil to CPSC on Jan 12, 2009.
- 2009 "Review of Recent Scientific Data on DINP and Risk Characterization for its Use in Toys and Childcare Articles" completed by EMBSI
  - Submitted to European Commission for re-evaluation of the DINP/DIDP Toy Restriction
  - Report clearly demonstrates that there is an adequate margin of safety for DINP in toys that can be mouthed to support lifting toy restriction
- 2009 Successful REACH registration of DIDP
- 2010 "DINP Carcinogenicity Hazard Assessment" completed by ExxonMobil Biomedical Sciences (EMBSI)
  - Submitted to California Office of Environmental Health Hazard Assessment (OEHHA) for consideration in determining potential Prop 65 listing.
  - Listing consideration based on observance of tumors in rodents treated with high doses of DINP; however, robust database demonstrates rodent tumors are not relevant to humans.
- 2010 Successful REACH registration of DINP

Not all Phthalates are the same

EU Evaluation of Plasticizers and PVC Toys

Restricted in all toys and childcare articles

Restricted in toys and childcare articles which can be placed in the mouth

No restrictions and no EU regulatory evaluations for use in toys

Evaluations	LMW phthalates Category 2 CMR	LMW phthalates Category 2 CMR	HMW phthalates	Other HMW phthalates	Linear phthalates	Other phthalates	Other plasticisers
Plasticisers	DEHP DBP BBP	DIBP DIHP	DINP DIDP	DPHP	Linear 810 Linear 911	BINP DOTP	DOA, DINA, DIDA, DOZ, DOS, Dibenzoates, Monobenzoates, Alkylsulphonic ester of phenol, TBC, TEC DINCH, Polymerics, Triglyceride esters of castor oil
EU Risk Assessment	Yes	No	Yes	No	No	No	No
EU Classification review	Yes - CMR Cat 2	Yes - CMR Cat 2	Yes - Not classified	No	No	No	No
Regulatory safety evaluation for use in toys	Yes	No	Yes	No	No	No	No
Restrictions in all toys and childcare articles	Yes - all toys and childcare articles	Yes (Toy Safety Directive)	No	No	No	No	No
Restrictions in toys and childcare articles that can be placed in the mouth	Yes	Yes	Yes	No	No	No	No

-The table shows commercial plasticisers which are used in PVC toys, have the potential to be used in PVC toys, or which have been restricted in PVC toys.

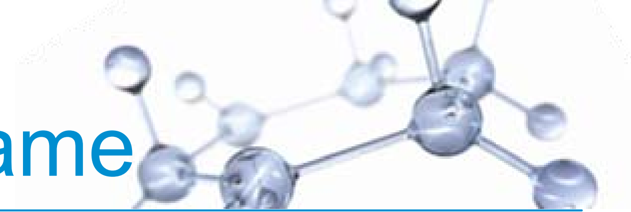
-Not all of the substances with the potential to be used are necessarily used in PVC toys.

-The table also shows the Cateogry 2 CMR phthalates.

ExxonMobil

Taking on the world's toughest energy challenges.

Not all phthalates are the same



Low molecular weight

DEHP, BBP, DBP

C4 to C8 alcohol + Phthalic Acid

Cat 2 Reproductive Agents

Risk reduction required

REACH Candidate List

Restricted in all toys and childcare

Articles pending scientific review

High molecular weight

DINP & DIDP

C9 & C10 Alcohol + Phthalic Acid

✓ Not CMR

✓ Not classified and labelled

✓ No risk reduction required

✓ Not endocrine disrupters

✓ REACH Registered

Temporary restriction in toys that can be placed in the mouth and childcare articles pending scientific review