

phosphorus present is unknown. According to the manufacturers, the extracts are likely to contain either tris(hydroxymethyl)phosphine oxide (THPO) (1067-12-5) (Figure I-3) or polymers of THPC (Baitinger, 2000; Martin, 1998). THPC and THPC-urea are considered unlikely to survive the oxidation step. Little is known about the toxicity of THPO and polymers of THPC (Bittner, 1999c; MacGregor et al., 1980). Additional information on the identity and toxicity of the chemicals migrating from THPC-treated fabrics is needed. Therefore, a quantitative assessment of the health risks associated with THPC-treated fabrics cannot be performed. However, an exposure assessment will be presented.

Like THPC, PA is a reactive FR chemical and migration was measured as total phosphorus. PA reacts covalently with cellulose fibers and durable press resins. From 20 to 28 percent of total phosphorus in the extracts was in the form of inorganic phosphorus (Cobb, 2000). The extracts contained multiple peaks that could not be resolved or quantified, but which might include PA. Thus, the chemical form of most of the total phosphorus present is unknown. Furthermore, there was insufficient information to calculate an acceptable daily intake (ADI) (Bittner, 1999b) or reference dose* (RfD) (NRC, 2000) PA, which does not satisfy the definition of “toxic” under the FHSA. Due to the lack of an ADI or RfD, an exposure assessment only will be presented for PA.

A substance that does not satisfy the FHSA definition of “toxic” cannot be considered “hazardous” under the FHSA. HBCD and PA do not satisfy the FHSA definition of “toxic.” Therefore, it is not necessary to perform exposure or risk assessments for HBCD and PA to determine whether products treated with these compounds are “hazardous substances.” However, exposure data for both compounds are available. Although the CPSC staff did not calculate an ADI value for HBCD, the NRC Subcommittee calculated an RfD. Thus, a risk assessment is presented for HBCD. Neither CPSC nor NRC derived an ADI or RfD for PA. However, such values might be derived in the future if additional toxicity data were to become available. Thus, an exposure assessment is presented here.

2. Exposure Routes and Scenarios

Exposure by all three possible routes—dermal, oral, and inhalation—was considered. Dermal exposure may occur through several scenarios (Table I-4). “Passive” dermal exposure may occur by contact of exposed skin to FR-treated fabric, such as when sitting on furniture (scenario D.1). Spilled liquids or cleaning agents may extract FR chemicals from the fabric and then deposit them on the fabric surface, leading to subsequent consumer exposure. Therefore, passive exposure may also occur after the furniture has been exposed to spilled liquids (D.2.a) or upholstery cleaners (D.2.b). “Active” dermal exposure may occur when cleaning up spilled liquids (D.3.a) or cleaning the furniture with upholstery cleaners (D.3.b). The spilled liquid or cleaner may extract FR chemical from the fabric, which then contacts the skin. Oral exposure may occur when children place FR-treated upholstery fabrics into their mouths (O.1). Inhalation exposure may occur through two scenarios. Semi-volatile FR’s may be emitted directly into indoor air (I.1). Fabric particles containing FR chemicals may be released into indoor air as the

* Both the acceptable daily intake (ADI) and reference dose (RfD) are estimates of the amount of a chemical a person can be exposed to on a daily basis over an extended period of time (up to a lifetime) with a negligible risk of suffering deleterious effects. The differences between the ADI and RfD are explained in the Discussion.

fabric is worn or ages (I.2). For five of the chemicals under consideration (see above), migration data were available for use in predicting dermal or oral exposure. Mathematical models were used to predict inhalation exposure (see Methodology).

In addition to the various exposure routes and scenarios, separate risk assessments were performed for adults and children. The risk assessment for children reflects differences in skin surface area, breathing rates, body weight, and behavior. Data on age-related differences in susceptibility to adverse health effects are unavailable for the FR's under consideration. In some cases, fabrics were subjected to accelerated aging or wear regimens prior to migration studies. These data were used to assess the effects of age and wear on exposure. The health endpoints considered are limited to chronic health effects, including cancer, reproductive/developmental effects, neurotoxicity, and other persistent adverse health effects.

Exposure from the three routes and various scenarios may be combined to estimate the total or aggregate exposure and risk. These were combined to form a series of reasonably foreseeable cases. The "basic" case (Table I-5), combines all scenarios; saline is used to model spills and aqueous cleaner to model the spot cleaning scenario. Oral exposure applies only to children. Direct exposure from cleaning applies only to adults. The "acidic spill" case is the same as the basic case, except that citric acid is used to model spills, as some beverages and foods are acidic. The "non-aqueous cleaner" case is the same as the basic case, except that a non-aqueous cleaner is used for the cleaning scenario. Finally, the "aged fabric" case is the same as the basic case, except that migration rates are adjusted for artificially aged or worn fabric.

3. Health Effects

This risk assessment is limited to the assessment of chronic health effects, including: carcinogenicity, neurotoxicity, reproductive/developmental toxicity, and chronic organ toxicity. Most of the FR chemicals exhibited low levels of acute toxicity. Chronic health effects, by their nature, are generally observed at lower levels than acute effects. All of the available toxicity data were considered in determining whether each FR chemical is toxic and in deriving ADI values. However, the number and quality of the studies comprising the database were dependent on the individual chemical (Table I-1).

AT caused systemic effects in animals when administered by the oral route (see Table I-2) (reviewed in Hatlelid, 1999a). When inhaled, AT dusts caused lung inflammation, fibrosis, and tumors in animals. Workers exposed to AT dusts at high levels developed pneumoconiosis. AT is considered to be probably toxic in humans, based on sufficient evidence of toxicity by the oral and inhalation routes in animals, and further supported by limited evidence of toxicity by inhalation in humans. Furthermore, AT is considered a probable carcinogen by inhalation in humans, based on sufficient evidence in animals. There was inadequate evidence of carcinogenicity in humans exposed by inhalation. The staff derived an oral ADI of 2.3 mg/kg-d, and an inhalation "ADI" of 9 ng/m³. A cancer potency estimate for inhalation was also derived (see Methodology).

DBDPO caused liver and thyroid effects in subchronic and lifetime feeding studies in rodents (reviewed in Bittner, 1999a). Thus, DBDPO is probably toxic in humans, based on

sufficient evidence in animals. The staff derived an oral ADI of 3.2 mg/kg-d (Bittner, 2001). In addition, DBDPO is considered a possible human carcinogen, based on limited evidence in animals. That DBDPO is possibly carcinogenic does not contribute to the finding that DBDPO is toxic under the FHSA.

There was limited evidence of liver toxicity, reproductive and developmental effects, and neurotoxicity in animals fed HBCD (reviewed in Hatlelid, 1999b). HBCD is considered possibly toxic in humans, based on limited evidence in animals. Thus, HBCD does not satisfy the FHSA definition of "toxic." This does not necessarily mean that HBCD is safe, only that there was insufficient data to support a finding of toxicity as defined under the FHSA. This conclusion could be changed if additional toxicity data became available. An ADI was not calculated, because HBCD does not satisfy the FHSA definition of toxic. The NRC Subcommittee derived an RfD of 0.2 mg/kg-d (NRC, 2000, p. 64).

There was inadequate evidence of toxicity in animals fed PA for up to 21 days (reviewed in Bittner, 1999b). Thus, PA does not satisfy the FHSA definition of toxic. However, the database on PA is very limited. This conclusion could be changed if additional toxicity data became available. There was insufficient information to derive an ADI or RfD.

There was sufficient evidence of liver toxicity and neurotoxicity in animals exposed to THPC (reviewed in Bittner, 1999c). Thus, THPC is probably toxic in humans, based on sufficient evidence in animals. In addition, THPC is a possible development toxicant in humans, based on limited evidence in animal studies. THPC is also acutely toxic. However, as discussed above, THPC is not present in detectable quantities in extracts of THPC-treated fabrics. The CPSC staff derived an oral ADI of 0.0027 mg/kg-d (Bittner, 2001).

CPE caused systemic effects in rats and maternal toxicity in rabbits (reviewed in Hatlelid, 1999c). Thus, CPE is probably toxic in humans, based on sufficient evidence in animals. The staff derived an oral ADI of 10 mg/kg-d (Bittner, 2001). CPE was also associated with minor developmental delays (reduced ossification and rib defects) in rabbits. Minor developmental delays or variations generally are not considered as providing sufficient evidence of toxicity. Therefore, CPE is considered a possible developmental toxicant, based on limited evidence in animals. That CPE is a possible developmental toxicant does not contribute to the finding that CPE is toxic under the FHSA.

EDHP was toxic to the liver and adrenal glands and led to reduced growth rates in animal studies (reviewed in Ferrante, 1999a). Thus, EDHP is probably toxic in humans, based on sufficient evidence of toxicity in animal studies. The staff derived an oral ADI value of 0.01 mg/kg-d.

TDCP was tested in a two-year feeding study in rats (reviewed in Ferrante, 1999b). In this study, TDCP induced liver carcinomas and adenomas, renal cortical tumors, adrenal tumors in females, and testicular interstitial tumors in males. Thus, TDCP is considered to be a probable human carcinogen, based on sufficient evidence in animals (tumors at multiple sites and at multiple doses). The CPSC staff derived a cancer potency estimate for TDCP (see

Methodology). TDCP is also acutely toxic in animals. The NRC Subcommittee derived an oral RfD of 0.005 mg/kg-d, based on testicular effects in male rats.

Table I-1. Availability of Toxicity Data on Flame Retardant Chemicals^a

Chemical/Class ^b	CAS no. ^c	Acute	Subchronic	Chronic	Repro/Dev	Neurotox ^d	Genotox	Human ^e
1 Antimony trioxide (AT)	1309-64-4	X	X	X	X	-	X	X
2 Decabromodiphenyl oxide (DBDPO)	1163-19-5	X	X	X	X	-	X	-
3 Hexabromocyclododecane (HBCD)	3194-55-6	X	X	X	X	-	X	-
	13674-84-5							
4 Tris(chloropropyl) phosphate (TCPP) (mixture of 4 isomers)	76649-15-5 76025-08-6 6145-73-9	X	-	-	-	-	X	-
5 Tris(1,3-dichloropropyl-2) phosphate (TDCP) Phosphonic acid, (3-[[hydroxymethyl] amino]-)	13674-87-8	X	X	X	X	X	X	-
6 3-oxopropyl-, dimethyl ester (PA) (Pyrovatex®)	20120-33-6	X	?	-	-	-	X	-
7 Tetrais (hydroxymethyl) phosphonium salts (THPX) (Proban®):								
Chloride salt (THPC)	124-64-1	X	X	X	X	?	X	-
Sulfate salt (THPS)	55566-30-8	X	X	X	?	?	X	-
Compound with urea (THPC-urea)		X	-	-	X	?	X	-
Polymer (THPOH/NH ₃)	27104-30-9	?	-	-	-	-	X	-
8 Organic phosphonates:								
Dimethyl phosphonate (DMHP)	868-85-9	X	X	X	X	X	X	-
Dimethyl methylphosphonate (DMMP)	756-79-6	X	X	X	X	X	X	-
Cyclic phosphonate esters (CPE) (mixture of monomer and dimer) (Antiblaze N/NT®) ^f	41203-81-0 42595-45-9	X	X	-	X	-	X	-
9 Aromatic phosphates:								
<i>t</i> -Butylphenyl diphenyl phosphate (BPDP)	56803-37-3	-	X	-	-	X	X	X
2-Ethylhexyl diphenyl phosphate (EHDP)	1241-94-7	X	X	X	X	X	X	-
Isodecyl diphenyl phosphate (IDDP)	29761-21-5	X	X	-	-	X	X	-
Phenol isopropylated phosphate (PIP)	86937-41-7	X	X	-	-	X	-	X
Santizer 141 (>90% EHDP)		X	X	-	X	X	X	-
Santizer 148 (>90% IDDP)		X	X	-	X	X	X	-

Table I-1. Availability of Toxicity Data on Flame Retardant Chemicals (continued)

Chemical/Class ^b	CAS no. ^c	Acute	Subchronic	Chronic	Repro/Dev	Neurotox ^d	Genotox	Human ^e
Santicizer 154 (TPP + BDP)		X	X	-	X	X	X	-
o-Tricresyl phosphate (o-TCP)		X	X	-	X	X	-	X
Tricresyl phosphate (TCP) (isomers)	1330-78-5	X	X	-	X	X	X	X
Triphenyl phosphate (TPP)	1145-86-6	X	X	-	X	X	X	X
10 Chlorinated paraffins	63449-39-8 + 20 others	X	X	X	X	-	X	-
11 Molybdates:								
Calcium molybdate	7789-82-4	X	X	-	-	-	-	-
Zinc molybdate	61583-60-6	X	-	-	-	-	-	-
12 Antimonates:								
Antimony Pentoxide	1314-60-9	X	-	-	-	-	X	-
Sodium antimonate	15432-85-6	X	-	-	-	-	-	-
13 Zinc borate (mixture of zinc oxide and boric anhydride)	1332-07-6	-	-	-	-	-	-	-
Zinc oxide	1314-13-2	X	X	-	X	X	X	X
Boric anhydride	1303-86-2	X	X	-	-	-	-	X
Boric acid	10043-35-3	X	X	X	X	-	X	X
14 Alumina trihydrate	21645-51-2	X	X	-	X	X	X	X
15 Magnesium hydroxide	1309-42-8	-	-	-	-	-	-	X
16 Ammonium polyphosphates (Antiblaize LR2 and Antiblaize LR4)	68333-79-9	X	-	-	-	-	X	-

Table I-1. Availability of Toxicity Data on Flame Retardant Chemicals (continued)

- ^a X indicates availability of data; ? limited data available; -, no data available.
- ^b These 16 flame retardant (FR) chemicals or chemical classes were proposed by the Fire Retardant Chemicals Association for use in upholstered furniture.
- ^c CAS no., Chemical Abstract Service number.
- ^d For neurotoxicity, a ? means that although a neurotoxicity test per se was not performed, neurotoxic effects were observed in other tests.
- ^e Human data includes clinical reports and epidemiological studies.
- ^f Monomer: phosphonic acid, methyl-, (5-ethyl-2-methyl-1,3,2-dioxaphosphorinan-5-yl)methyl methyl ester, P-oxide.
Dimer: phosphonic acid, methyl-, bis[(5-ethyl-2-methyl-1,3,2-dioxaphosphorinan-5-yl)methyl] ester, P,P'-oxide.

Table I-2. Toxicity Summary of Flame Retardant Chemicals

Chemical/Class ^a	Acute Toxicity ^b	Chronic Toxicity ^c	Endpoint ^d	NOAEL/LOAEL ^{e,f} (mg/kg-d)	UF ^f	ADI ^{g,h} (mg/kg-d)	
1 Antimony trioxide (AT)	Oral	B	O	230	100	2.3	
	Inhalation	B	C,O	(9 µg/m ³) L	1,000	(9 ng/m ³)	
2 Decabromodiphenyl oxide (DBDPO)		B	O	3,200 L	1,000	3.2	
3 Hexabromocyclododecane (HBCD)		B	R,D,N,O			ND ⁱ	
4 Tris(chloropropyl) phosphate (TCPP) (mixture of 4 isomers)	T	I				ND	
5 Tris(1,3-dichloropropyl-2) phosphate (TDCP)	T	B	C			ND	
6 Phosphonic acid, (3-[[hydroxymethyl] amino]-3-oxopropyl)-, dimethyl ester (PA) (Pyrovatex®)		I				ND	
7 Tetrakis (hydroxymethyl) phosphonium salts (THPX) (Proban®):	Chloride salt (THPC)	B	N,O	2.7 L	1,000	0.0027	
	Sulfate salt (THPS)	B	O	3.6	100	0.036	
	Compound with urea (THPC-urea)	B	D	50	100	0.5	
	Polymer (THPOH/NH ₃)	I				ND	
	Organic phosphonates:						
Dimethyl phosphonate (DMHP)	T	B	O	36	100	0.36	
Dimethyl methylphosphonate (DMMP)		B	R,O	180 L	1,000	0.18	
Cyclic phosphonate esters (CPE) (mixture of monomer and dimer) (Antiblaze N/NT®) ^g		B	O	1,000	100	10	
9 Aromatic phosphates:	t-Butylphenyl diphenyl phosphate (BPDP)	I				ND	
	2-Ethylhexyl diphenyl phosphate (EHDP)	B	O	100	100	1.0	
	Isodecyl diphenyl phosphate (IDDP)	C	O			ND	
	Phenol isopropylated phosphate (PIP)	C	N,O			ND	
	Santizer 141 (>90% EHDP)	T	B	O	100	100	1.0
	Santizer 148 (>90% IDDP)		B	O			0.01
	Santizer 154 (TPP + BPDP)		C	N,O			ND
	o-Tricresyl phosphate (o-TCP)		A	N			ND
	Tricresyl phosphate (TCP) (isomers)		B	R,N,O	50 L	1,000	0.05
	Triphenyl phosphate (TPP)		C	N,O			ND

Table I-2. Toxicity Summary of Flame Retardant Chemicals (continued)

Chemical/Class ^a	Acute Toxicity ^b	Chronic Toxicity ^c	Endpoint ^d	NOAEL/LOAEL ^e (mg/kg-d)	UF ^f	ADI ^{g,h} (mg/kg-d)
10 Chlorinated paraffins		B	C, D, O ⁱ	10	100	0.1
11 Molybdates:						
Calcium molybdate		C	O			ND
Zinc molybdate		C	O			ND
12 Antimonates:						
Antimony Pentoxide		I				ND
Sodium antimonate		I				ND
13 Zinc borate (mixture of zinc oxide and boric anhydride)	T	C	R, D, N, O			ND
14 Alumina trihydrate		A	N, O			45 (AI) ^j
15 Magnesium hydroxide		A	O			68 ^j
16 Ammonium polyphosphates (Antiblaze LR2 and Antiblaze LR4)		I				ND

^a These 16 flame retardant (FR) chemicals or chemical classes were proposed by the Fire Retardant Chemicals Association for use in upholstered furniture.

^b Acute toxicity as defined in FHSA regulations: T, toxic; H, highly toxic.

^c Chronic toxicity as defined under the FHSA and the CPSC chronic hazard guidelines: A, known to be toxic in humans; B, probably toxic in humans; C, possibly toxic in humans; I, insufficient data. Based on oral studies, except where indicated.

^d Chronic toxicity endpoint(s): C, cancer; D, developmental; N, neurotoxicity; R, reproductive; O, other (e.g., liver toxicity).

^e Doses are in mg/kg-d by the oral route, except where indicated. I indicates LOAEL.

^f ADI, acceptable daily intake; LOAEL, lowest observed adverse effect level; ND, not determined; NOAEL, no observed adverse effect level; UF, uncertainty factor.

^g Monomer: phosphonic acid, methyl-, (5-ethyl-2-methyl-1,3,2-dioxaphosphorinan-5-yl)methyl methyl ester, P-oxide.

^h Dimer: phosphonic acid, methyl-, bis[(5-ethyl-2-methyl-1,3,2-dioxaphosphorinan-5-yl)methyl] ester, P, P'-oxide.

ⁱ Toxicity depends on the chain length and level of chlorine substitution. Carcinogenicity was observed with a short-chain product.

^j Maximum therapeutic dose, as aluminum.

Maximum therapeutic dose.

Table I-3. FR chemical treatments on which exposure and/or risk assessments were performed.

FR chemical	Application method	Migration data available	Percutaneous absorption data available
Antimony trioxide (AT)	BC ^a	Y	N ^b
Cyclic phosphonate ester (CPE) ^c Both washed and unwashed fabrics	I ^d	N	Y
Decabromodiphenyl oxide (DBDPO)	BC	Y	Y
2-Ethylhexyl diphenyl phosphate (EHDP)	BC	N	N
Hexabromocyclododecane (HBCD)	BC	Y	Y
Phosphonic acid, (3-[[hydroxymethyl]amino]-3-oxopropyl)-, dimethyl ester (PA) ^e	I ^f	Y	N
Tetrakis (hydroxymethyl) phosphonium chloride (THPC) ^g	I ^f	Y	N
Tris (1,3-dichloropropyl-2) phosphate (TDCP)	BC	N	Y

^a BC, back-coated; I, immersion treated; N, no; Y, yes.

^b Surrogate compounds or assumptions were used to estimate exposure when migration or percutaneous absorption data were not available.

^c Sold under the brand name Antiblaze N/NT[®].

^d Immersion treated with heat cure, also known as the Thermosol[®] process. Unbound CPE is generally removed by washing (scouring), but this step is sometimes omitted.

^e Sold under the brand name Pyrovatex[®].

^f These are reactive FR chemicals.

^g Mixture of THPC and THPC-urea. Sold under the brand name Proban CC[®].

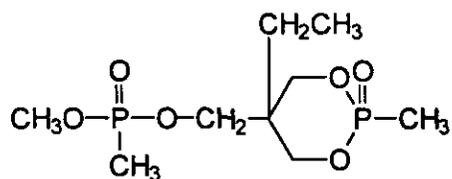
Table I-4. Exposure routes and scenarios under consideration.

Route	Scenario	No.	Fabric	Adults	Children
Dermal	<u>Passive Exposures</u>				
	Normal use	D.1	new	Y*	Y
			old	Y	Y
	Fabric exposed to spills	D.2.a	new	Y	Y
			old	Y	Y
	Fabric exposed to cleaners	D.2.b	new	Y	Y
			old	Y	Y
<u>Active Exposures</u>					
Spills	D.3.a	new	Y	Y	
		old	Y	Y	
Spot cleaning	D.3.b	new	Y	N	
		old	Y	N	
Oral	Mouthing	O.1	new	N	Y
			old	N	Y
Inhalation	<u>Vapor phase</u>	I.1	new	Y	Y
	<u>Particles</u>	I.2	new	Y	Y

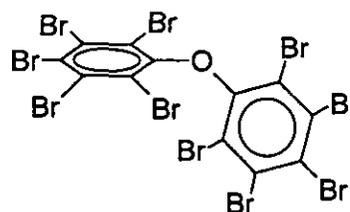
* Y, yes; N, no.

Table I-5. Combining risks from exposure scenarios.

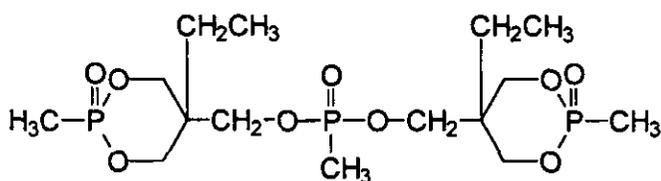
Case	Scenarios
Basic	Combines all scenarios. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.
Acid spill	Same as the basic case, but with citric acid to model spills.
Non-aqueous cleaner	Same as the basic case, but with non-aqueous cleaner.
Aged fabric	Same as the basic case, but adjusting for aged/worn fabric.



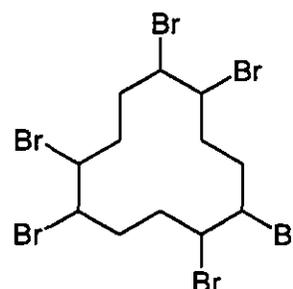
CPE Monomer



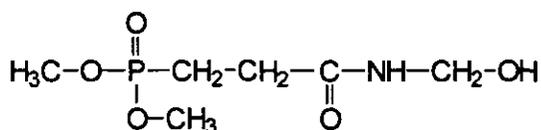
DBDPO



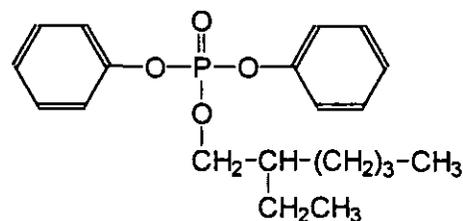
CPE Dimer



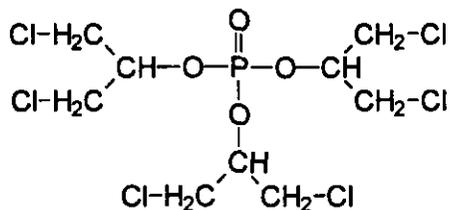
HBCD



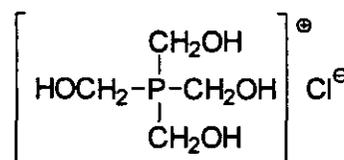
PA



EHDP



TDCP



THPC

Figure I-1. Chemical structures of organic flame retardant chemicals.

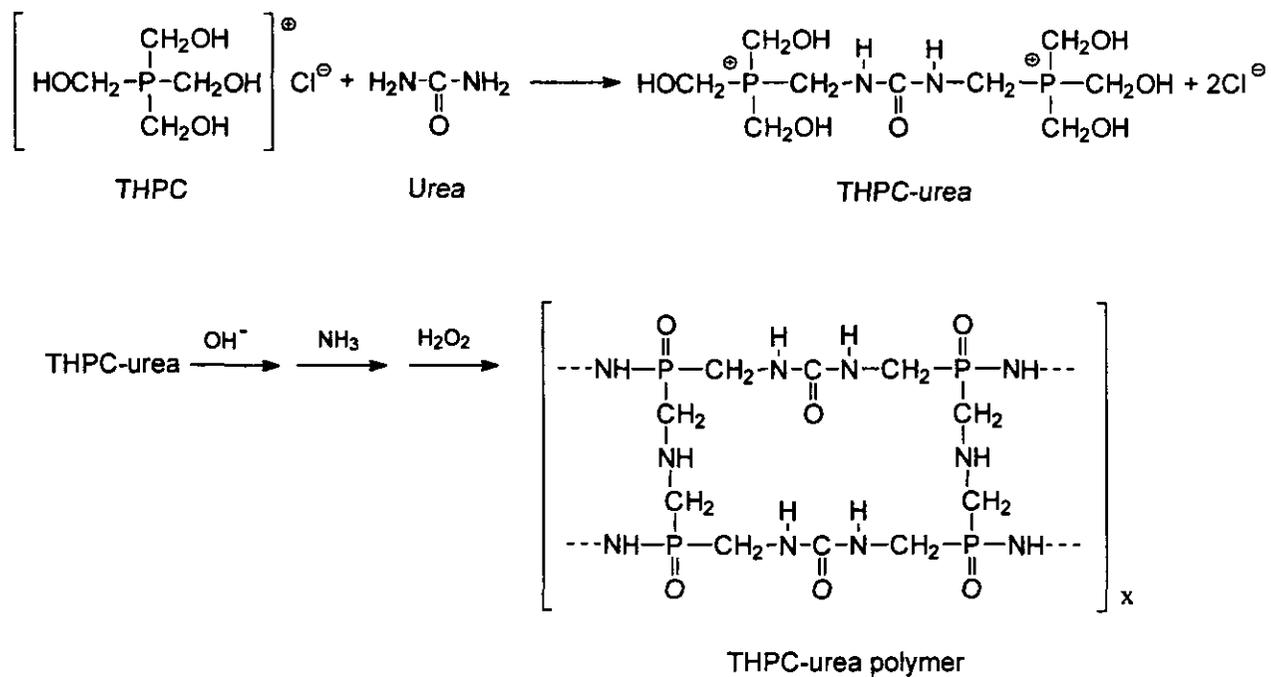


Figure I-2. Process for the Application of THPC and THPC-urea.

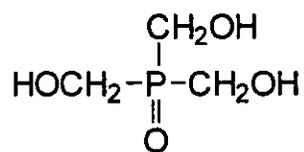


Figure I-3. Tris(hydroxymethyl) phosphine oxide (THPO).

II. Methodology

A. Hazard Identification

The CPSC staff completed toxicity reviews of the 16 FR chemicals or classes proposed by the FRCA for use in upholstered furniture. The toxicity reviews were performed in accordance with the CPSC chronic hazard guidelines (CPSC, 1992). The guidelines provide definitions of sufficient, limited, and inadequate evidence that are specific for carcinogenicity (*ibid.*, pp. 46635-46636), neurotoxicity (*ibid.*, pp. 46639-46641), and reproductive/developmental toxicity (*ibid.*, pp. 46642-46644). In general, "sufficient evidence in humans" means that there is sufficient evidence to establish a cause-and-effect relationship. In other words, there must be a statistically significant effect in well-designed epidemiological studies; no identified bias that can account for the association; and all possible confounding factors can be reasonably ruled out. "Sufficient evidence in animals" generally means that there is a statistically significant effect, at multiple doses, in multiple species or strains, or by different routes of administration, in well-designed studies. Limited evidence means that one of the criteria for sufficient evidence is not satisfied. Inadequate evidence means that two or more of the criteria for sufficient evidence are not satisfied.

A substance is classified as "known to be toxic" in humans only if there is sufficient evidence in humans. It is considered "probably toxic," if there is either limited evidence in humans or sufficient evidence in animals, and "possibly toxic" if there is either inadequate evidence in humans or limited evidence in animals (see page 3). A substance is considered "toxic" under the FHSA, if it is either known to be, or is probably, toxic in humans.

B. Dose Response Assessment

For non-cancer endpoints, an uncertainty factor approach was used to calculate an "acceptable daily intake" (ADI) value from the no observed adverse effect level (NOAEL) (CPSC, 1992, p. 46656). The default uncertainty factors include a factor of 10 to account for differences in sensitivity between individuals, and another factor of 10 for differences between animals and humans if animal data are used. In cases where a NOAEL has not been established, the ADI is calculated from the lowest observed adverse effect level (LOAEL) using an additional 10-fold uncertainty factor. Under the chronic hazard guidelines, additional uncertainty factors for extrapolating from subchronic to chronic exposure or to account for the lack of data for certain endpoints are not applied. Rather, ADI's are simply based on the available toxicity data.

Unit cancer risks (cancer potency estimates) were calculated for AT (inhalation of particles) and TDCP (oral route). Animal bioassay data were fitted to the multistage model using Global83 (Howe and Crump, 1983; Crump, 1984), as described in the chronic hazard guidelines (CPSC, 1992, p. 46654). The unit risk was based on the maximum likelihood estimate of extra risk, that is, the linear term (q_1) in the model. In cases (that is, AT) where q_1 was zero, the 95 percent upper confidence limit of risk (q_1^*) was used to ensure a linear dose response at low doses. Animal-to-human extrapolation was by the surface area correction. By this method, the unit risk is proportional to body weight to the two-thirds power. Thus, humans are roughly 6-fold more sensitive than rats. However, such an adjustment was not applied to AT, because

the dose is expressed as the concentration in air and AT is active at the site of contact (the lung) (CPSC, 1992, p. 46654).

In the case of AT, the unit risk is based on the incidence of lung tumors (adenoma and carcinoma) in rats (Watt, 1983). Doses were adjusted for the exposure regimen (6 hours per day, 5 days per week for 1 year) and it was assumed that the life span of the animals was 24 months (EPA, 1988, p. 1-5). Thus, the average exposure was calculated by:

$$C_{TWA} = C \cdot \frac{6}{24} \cdot \frac{5}{7} \cdot \frac{12}{24} \quad (2.1)$$

where: C_{TWA} , time-weighted average concentration, mg/m^3 ; and C , concentration in air, mg/m^3 .

The time-weighted average concentrations are as follows:

C (mg/m^3)	C_{TWA} (mg/m^3)
1.6	0.14
4.2	0.38

In the case of TDCP, the unit risk was based on the incidence of liver carcinoma and tumors of the renal cortex in males and females (Akzo Nobel, 1998). Benign interstitial cell tumors of the testes were not included (CPSC, 1992, p. 46636). Unit risks for liver and renal tumors were calculated separately and then combined, as described in the guidelines (CPSC, 1992, p. 46654). Males and females were combined, because their unit risks differed by less than a factor of two. Details such as mortality and body weight were not available. It was assumed that all animals (60 per dose/sex group) were at risk and that the doses were for a lifetime. An average body weight of 350 grams (EPA, 1988, p. 3-60) was assumed for the purpose of animal to human extrapolation.

C. Exposure and Bioavailability Assessment

1. Migration Studies

The total FR content in fabrics was measured by different methods, depending on the FR present. For AT, PA, and THPC, the fabric was digested in nitric acid. Elemental antimony (AT) or phosphorus (PA and THPC) was measured by inductively coupled plasma spectrometry (ICP) (Bhooshan and Cobb, 2000). For DBDPO and HBCD, fabric was extracted with either tetrahydrofuran (DBDPO) or acetonitrile (HBCD) and analyzed by high pressure liquid chromatography (HPLC).

The LSC staff used two methods to measure the migration of FR chemicals into liquid media (Bhooshan and Cobb, 2000). The methods are described briefly here. The first method is migration to filter paper. The filter paper method was used mainly to estimate dermal exposure

by liquid-mediated scenarios, such as may occur when liquids are spilled on upholstered furniture or when the furniture is cleaned (see below). A 38 mm x 38 mm sample of upholstery fabric was placed in a 400 mL beaker with the top or finished side facing upward. A 55 mm diameter piece of filter paper was placed on the fabric sample and saturated with 1.5 mL to 2.0 mL of solvent (either 0.9 percent saline, 5 percent citric acid, an aqueous upholstery cleaner, or methyl chloroform). The solvent was allowed to evaporate overnight, and then the amount of FR chemical migrating into the filter paper was measured. The filter paper was analyzed as described above. The process was repeated for a total of five extractions on the same fabric sample, and the results combined. The migration is calculated as the fraction of total FR chemical in the filter paper.

The second migration method is referred to as the “head-over-heels” method. The head-over-heels method was used to estimate oral exposure to children from the mouthing of upholstery fabrics. It is essentially the same method used previously to estimate exposure to phthalates in children’s products made from polyvinyl chloride, such as pacifiers, teethingers, and toys (CPSC, 1998; EU, 1998). A 38 mm x 38 mm sample of upholstery fabric was placed in a screw cap bottle with 25 mL of 0.9 percent saline. The bottle was placed in a special apparatus where it was rotated at 60 rpm for 30 minutes. The process was repeated a total of three times and the saline solutions were combined. The saline was analyzed as described above. The average migration rate was calculated as mg/cm²-h.

2. Equations for Predicting Exposure and Dose

This section presents the equations for predicting exposure and dose. More detailed explanations and derivations of the equations are provided in Appendix A.

a. Dermal Exposure

To estimate dermal exposure, it will be assumed that an external liquid phase facilitates the transfer of FR chemical from the fabric to the skin (NRC, 2000, p. 38). During normal use, that is, while sitting on furniture, perspiration is assumed to be the liquid phase. Depending on the circumstances, the liquid phase may be body fluids, spilled beverages, or liquid cleaners. The average daily dose (ADD) from dermal exposure was calculated by:

$$ADD_{D,x} = \frac{L \cdot M_L \cdot F_F \cdot A_S \cdot k_T \cdot T \cdot N}{W} \quad (2.2)$$

where: $ADD_{D,x}$, average daily dose from dermal exposure by scenario x , mg/kg-d; L , FR chemical loading, mg/cm²; M_L , fraction of FR chemical that migrates into the liquid phase; F_F , fraction of liquid phase remaining in the fabric after the bulk liquid is removed, unitless; A_S , skin surface area exposed, cm²; k_T , percutaneous absorption rate, h⁻¹; T , exposure duration, h; N , number of exposures per day, d⁻¹; and W , body weight, kg.

The fraction of FR chemical migrating into the liquid phase (M_L) was obtained by the static “filter paper” method described above (under C.1. Migration Studies) (Bhooshan and Cobb, 2000). M_L is assumed to equal the fraction of FR chemical in the filter paper after the solvent

evaporates. Isotonic saline was used as a surrogate for perspiration or spilled neutral pH beverages. Five percent citric acid solution was used as a surrogate for acidic beverages or foods. An aqueous (i.e., water-based) upholstery cleaner (referred to as “cleaner 1” in Bhooshan and Cobb, 2000) was used as the “aqueous cleaner.” This product is believed to be typical of upholstery cleaners sold to consumers. Methyl chloroform was used to represent dry cleaning type upholstery cleaners and is referred to as “non-aqueous cleaner.”

The fraction of liquid phase remaining in the fabric after the bulk liquid is removed, F_F , applies to passive exposure scenarios involving spills and cleaning. This variable accounts for the fact that spills and cleaning solutions are generally removed by blotting with a paper towel or sponge. For other scenarios (normal use and active exposures), F_F is set equal to one.

Certain active dermal exposure scenarios, such as active exposure to spilled liquids or cleaning agents, occur intermittently. That is, they are not daily occurrences. The present risk assessment is concerned with chronic health effects, which are generally based on chronic or subchronic animal studies. Therefore, for certain non-cancer effects, it may be appropriate to average these intermittent exposures over longer time periods. The average daily dose (ADD) may be adjusted as follows:

$$ADD_{TW,i} = \frac{ADD_i \cdot N_A}{T_A} \quad (2.3)$$

where: $ADD_{TW,i}$, time-weighted average daily dose from the i -th scenario, mg/kg-d; ADD_i , average daily dose from the i -th scenario, mg/kg-d; N_A , the of days that the exposure takes place during the averaging period, d; and T_A , averaging period, d.

The time-weighted ADD was calculated for scenarios D.3.a and D.3.b. and was used in place of the one-day ADD.

b. Oral Exposure

Oral exposure may occur when children mouth FR-treated upholstery fabrics (O.1). The “head-over-heels” method was used to estimate the migration rate of FR chemicals into a saliva simulant (see above, C.1, Migration Studies). The ADD from oral exposure was calculated by:

$$ADD_{O.1} = \frac{k_H \cdot A_F \cdot T \cdot N}{W} \quad (2.4)$$

where: $ADD_{O.1}$, average daily dose from scenario O.1, mg/kg-d; k_H , migration rate, mg/cm²-h, as measured by the head-over-heels method (Bhooshan and Cobb, 2000); A_F , fabric area, cm²; T , exposure duration, h; N , number of exposures per day, d⁻¹; and W , body weight, kg.

c. Inhalation Exposure

Inhalation exposure may occur through two scenarios. Semi-volatile FR's may be emitted directly into indoor air (I.1). In addition, fabric particles containing FR chemicals may be released into indoor air as the fabric is worn or ages (I.2). A simple one-zone mass balance model may be used to predict the concentration of FR chemicals in indoor air (NRC, 1981). The steady-state pollutant concentration in indoor air is given by:

$$C_A = \frac{S}{V(ACH + k_D)} \quad (2.5)$$

where: C_A , concentration of particle-bound FR chemical in indoor air, mg/m^3 ; S , source strength, mg/h ; V , room volume, m^3 ; ACH , air infiltration rate, h^{-1} ; and k_D , decay rate, h^{-1} .

In the case of vapor phase emissions, the decay rate (k_D) was assumed to be zero. In other words, sink effects were not considered. The source strengths (mass emitted per unit time) of vapor phase or particulate emissions from building or furnishing materials are typically derived from emission rates (source strength per unit area) measured in chambers. However, such data are not available for FR chemical-treated fabrics. Therefore, mathematical models were used to estimate emission rates. Different models were used for vapor phase and particulate emissions.

Vapor phase FR chemicals (I.1). The ADD from inhalation exposure to vapor phase FR chemicals was calculated by:

$$ADD_{I,1} = \frac{S \cdot I \cdot T \cdot N}{ACH \cdot V \cdot W} \quad (2.6)$$

where: $ADD_{I,1}$, average daily dose from exposure scenario I.1, $\text{mg}/\text{kg}\cdot\text{d}$; S , source strength, mg/h ; I , average inhalation rate, m^3/h ; T , exposure duration, h ; N , number of exposures per day, d^{-1} ; ACH , air infiltration rate, h^{-1} ; V , room volume, m^3 and W , body weight, kg .

The source strengths (mass emitted per unit time) of volatile chemical emissions from building or furnishing materials are typically derived from emission rates (source strength per unit area) measured in small chambers. However, such data are not available for FR chemical-treated fabrics. Therefore, emission rates will be predicted by the use of a mathematical model essentially similar to that described by the National Research Council (NRC, 2000, p.p. 44-49). The model does not allow for absorption of the FR chemical by surfaces in the room or reactive decay processes. The source strength for vapor phase FR chemical was calculated by:

$$S_V = \frac{C_{Sat}}{\frac{1}{ACH \cdot V} + \frac{H}{F_A \cdot A_F \cdot D_{Air}}} \quad \text{For } T_{Max} \geq Y_F \text{ years} \quad (2.7)$$

$$S_V = \left(\frac{T_{Max}}{Y_F} \right) \left(\frac{C_{Sat}}{\frac{1}{ACH \cdot V} + \frac{H}{F_A \cdot A_F \cdot D_{Air}}} \right) \quad \text{For } T_{Max} < Y_F \text{ years}$$

where:

$$T_{Max} = \frac{10,000 \cdot L \cdot H}{C_{Sat} \cdot D_{Air} \cdot 8,766} \left(1 + \frac{F_A \cdot A_F \cdot D_{Air}}{ACH \cdot V \cdot H} \right) \quad (2.8)$$

and where: S_V source strength for vapor phase FR chemical; C_{Sat} , saturation concentration of the FR chemical in air, mg/m^3 ; ACH , air infiltration rate, h^{-1} ; V , room volume, m^3 ; H , boundary layer thickness, that is, layer of air immediately over the fabric where transfer from the solid phase to vapor phase occurs, m ; F_A , fraction of fabric that is exposed to air, unitless; A_F , fabric area, m^2 ; and D_{Air} , diffusivity of the FR chemical in air, m^2/h ; T_{max} , maximum time that the steady-state FR concentration in air could be maintained, years; L , FR chemical loading, mg/cm^2 ; 10,000 is to convert from mg/cm^2 to mg/m^2 ; and Y_F , average lifetime of upholstered furniture, years.

This model is algebraically equivalent to the model used by NRC. It has been rearranged to separate the source strength expression from the mass-balance model. This was done, in part, to allow the use of alternative source strength models or the eventual use of empirically derived source strengths in future risk assessments (see Discussion).

Particle-bound FR chemical (I.2). The ADD from inhalation exposure to particle-bound FR chemicals was calculated by:

$$ADD_{12} = \frac{S_p \cdot I \cdot T \cdot N}{W \cdot V (ACH + k_D)} \quad (2.9)$$

where: $ADD_{1,2}$, average daily dose from scenario I.2, $\text{mg}/\text{kg}\cdot\text{d}$; S_p , source strength of particle-bound FR chemical, mg/h ; I , average inhalation rate, m^3/h ; T , exposure duration, h ; N , number of exposures per day, d^{-1} ; W , body weight, kg ; V , room volume, m^3 ; ACH , air infiltration rate, h^{-1} ; and k_D , particle deposition rate, h^{-1} .

The model used to estimate the source strength for particle-bound FR chemical is essentially similar to the model used by the NRC (NRC, 2000, pp. 42-44). The source strength for particle-bound FR chemicals was calculated by:

$$S_p = 10,000 \cdot L \cdot A_F \cdot F_W \cdot k_R \quad (2.10)$$

where: 10,000 cm²/m² is to convert from mg/cm² to mg/m²; L, FR chemical load, mg/cm²; A_F, fabric area, m²; F_W, fraction of the fabric area subjected to heavy wear; and k_R, fabric particle release rate, h⁻¹.

In the case of AT, which acts directly on the respiratory tract, it is more convenient to use the average daily exposure (ADE), rather than the ADD. The ADE for inhalation exposure to particle-bound FR chemicals was calculated by:

$$ADE = \frac{C_{AP} \cdot T \cdot N}{24} \quad (2.11)$$

where: ADE, time-weighted average daily exposure, mg/m³; C_{AP}, airborne particle-bound FR concentration, mg/m³; T, exposure duration, h; N, number of exposures per day, d⁻¹; and 24, the number of hours per day.

D. Risk Assessment

1. Non-Cancer Endpoints

The potential risk from non-cancer endpoints is evaluated by calculating the hazard index (HI), which is the ratio of the ADD to the acceptable daily intake (ADI), that is:

$$HI_i = \frac{ADD_i}{ADI} \quad (2.12)$$

where: HI_i, hazard index from exposure scenario i, unitless; ADD_i, average daily dose from exposure scenario i, mg/kg-d; and ADI, acceptable daily intake, mg/kg-d.

When the HI is greater than one, the product or exposure scenario under consideration is considered to present a hazard to consumers. Dermal exposure estimates include an adjustment for bioavailability, that is, the percutaneous absorption rate (see above). The ADI values are generally based on bioassays in which animals are exposed orally. Therefore, a route-to-route adjustment was applied for dermal exposures. Thus, the HI for dermal exposure was calculated by (Babich, 1989, p. 21):

$$HI_{D,j} = \frac{ADD_{D,j}}{ADI \cdot B} \quad (2.13)$$

where: HI_{D,j}, hazard index for dermal exposure by the D.j scenario, unitless; ADD_{D,j}, average daily dose from the dermal scenario D.j, mg/kg-d; ADI, acceptable daily intake, mg/kg-d; and B, bioavailability in the oral bioassay from which the ADI is derived, that is the fraction of the oral dose that is absorbed, unitless.

In the case of AT, which acts directly on the respiratory tract, exposure was expressed as the average airborne concentration (mg/m^3), rather than the average daily intake ($\text{mg}/\text{kg}\cdot\text{d}$) (see above). In such cases, an "inhalation ADI" (ADI_I) was calculated. In this case, the HI is calculated by:

$$HI_{I,j} = \frac{ADE_{I,j}}{ADI_I} \quad (2.14)$$

where: $HI_{I,j}$, hazard index for exposure scenario I,j , unitless; $ADE_{I,j}$, time-weighted average daily exposure from scenario I,j , mg/m^3 ; and ADI_I , "inhalation ADI," mg/m^3 .

Exposures to the same chemical from different scenarios were combined by summing the HI values from different scenarios, where appropriate:

$$HI_{Total} = \sum_i HI_i \quad (2.15)$$

where: HI_{Total} , hazard index summed over different scenarios; and HI_i , hazard index from scenario i .

2. Cancer Risk

The lifetime average daily dose (LADD) was calculated from the ADD as follows:

$$LADD_i = \frac{ADD_i \cdot N_y \cdot Y}{365.25 \cdot Y_E} \quad (2.16)$$

where: $LADD_i$, lifetime average daily dose from the i -th scenario; ADD_i , average daily dose from the i -th scenario, $\text{mg}/\text{kg}\cdot\text{d}$; N_y , number of days per year that the product is used or that the exposure scenario occurs, d/y ; Y , number of years of exposure, y ; 365.25, number of days per year, d/y ; Y_E , average life expectancy, y .

The lifetime individual excess cancer risk was calculated by:

$$R_i = Q \cdot LADD_i \quad (2.17)$$

where: R_i , lifetime individual excess cancer risk from the i -th scenario; Q , unit cancer risk, or cancer potency, $(\text{mg}/\text{kg}\cdot\text{d})^{-1}$; and $LADD_i$, lifetime average daily dose from the i -th scenario, $\text{mg}/\text{kg}\cdot\text{d}$.

Dermal exposure estimates include an adjustment for bioavailability, that is, the percutaneous absorption rate (see above). Thus, the cancer risk from dermal exposures was calculated by (Babich, 1989, p. 21):

$$R_{D,j} = \frac{Q \cdot LADD_{D,j}}{B} \quad (2.18)$$

where: $R_{D,j}$, lifetime individual excess cancer risk from the D.j scenario; Q, unit cancer risk, or cancer potency, $(\text{mg}/\text{kg}\cdot\text{d})^{-1}$; $LADD_{D,j}$, lifetime average daily dose from the D.j scenario, $\text{mg}/\text{kg}\cdot\text{d}$; and B, bioavailability in the oral bioassay from which the unit risk is derived, that is the fraction of the oral dose that is absorbed, unitless.

In the case of AT, in which the cancer risk was based on the airborne concentration, the lifetime average daily exposure (LADE) was calculated by:

$$LADE_{I,j} = \frac{ADE_{I,j} \cdot N_Y \cdot Y}{365.25 \cdot Y_E} \quad (2.19)$$

where: $LADE_{I,j}$, lifetime average daily exposure from scenario I.j, mg/m^3 ; $ADE_{I,j}$, average daily exposure from scenario I.j, mg/m^3 ; N_Y , number of days per year that the product is used, d/y; Y, number of years of exposure, y; 365.25, number of days per year, d/y; Y_E , average life expectancy, y.

Then, the lifetime individual excess cancer risk is:

$$R_{I,j} = Q_I \cdot LADE_{I,j} \quad (2.20)$$

where: $R_{I,j}$, lifetime individual excess cancer risk from scenario I.j; Q_I , unit cancer risk, or cancer potency, by the inhalation route, $(\text{mg}/\text{m}^3)^{-1}$; and $LADE_{I,j}$, lifetime average daily exposure by scenario I.j, mg/m^3 .

The risks from exposures to the same chemical from different scenarios were combined by summing the risks from each scenario, where appropriate:

$$R_{Total} = \sum_i R_i \quad (2.21)$$

where: R_{Total} , individual excess cancer risk summed over different scenarios; and R_i , individual excess cancer from scenario i .

E. Input Parameters

This section lists and describes the sources of the input parameters used to calculate exposure and risk.

1. General Parameters

General input parameters are those that are applicable to multiple exposure scenarios (Table II-1). The average lifetime of a suite of upholstered furniture, 15 years, was estimated by

industry representatives (NRC, 2000; Ray, 2000). The average number of years of exposure to upholstered furniture is not necessarily the same as the average product life. Consumers are exposed to upholstered furniture for virtually their entire lives. A consumer may be exposed to several different types of furniture, which may be treated with different FR chemicals or none at all. For the purpose of risk assessment, it was assumed that adults are exposed to the same FR treatments for a lifetime, 75 years, which is the most conservative approach.

For children, an exposure duration of two years, the first two years of life, was assumed. Children are most likely to place objects in their mouths between 3 months and 12 months of age; mouthing activity declines significantly by 24 months of age (Greene, 1998; Groot et al., 1998). Mouthing activity is one of the principal quantifiable differences between children and adults that may affect exposure to FR chemicals. The surface area and respiration rate, relative to body weight, are also different in children. Birth to two years is the period when the differences between children and adults are greatest.

In calculating cancer risks, which depend on cumulative exposure, the cancer risk in adults represents the risk from a lifetime of exposure, 75 years. The cancer risk in children represents the contribution to the lifetime risk from exposure during the first two years of life. Because only children are exposed by mouthing, the risk from this route of exposure may be added to the risk in adults to obtain the true lifetime risk. However, among the FR chemicals considered herein, only TDCP is a systemic carcinogen. The estimated cancer risk from mouthing was negligible in comparison to the lifetime cancer risk in adults (see Results, under TDCP).

The body weight for adults is the mean body weight (EPA, 1997a). The body weight for children is the mean weight for children from 3 to 12 months old (Green, 1998). The average life expectancy is from the EPA "Exposure Factors Handbook" (EPA, 1997a, p. 8-1).

2. Chemical-Specific Parameters

The chemical-specific parameters are those which are unique to a given chemical. They include the physico-chemical, toxicological, and product application properties. Due to the number of chemicals, the tables listing the chemical-specific parameters are divided into two parts. The first part of each table (Tables II-2a, II-3a, and II-4a) includes the chemicals for which LSC developed migration data—AT, DBDPO, HBCD, PA, and THPC. The second part (Tables II-2b, II-3b, and II-4b) includes chemicals for which migration data are not available—CPE, EDHP, and TDCP. CPE is considered in two forms, washed and unwashed. Fabrics are generally washed (scoured) to remove excess CPE, although this step is sometimes omitted. Omitting the wash step increases the bioavailability of the CPE (Albright and Wilson, 1998a; Maibach, 1979; Ulsamer et al., 1980). THPC and PA are reactive FR's. Because phosphorus was used as a surrogate for THPC and PA in the migration studies, the identity of the species in the liquid phase is unknown. Only exposure assessments were done for THPC and PA.

a. Physico-Chemical Properties

Most of the physico-chemical properties (Table II-2a, b) are from the CPSC staff toxicity reviews (Bittner, 1999a, b, c; Ferrante, 1999a, b; Hatlelid, 1999a, b, c), which generally obtained

information from the Hazardous Substances Data Bank (HSDB) or other secondary sources. The vapor pressure of THPC is for an 80% aqueous solution (as cited in NRC, 2000). The vapor pressure of CPE was unavailable. Dimethyl phosphonate is a structurally related compound with a lower molecular weight (110) and a vapor pressure of 4.52 (as cited in Hatlelid, 1999c). Therefore, a vapor pressure of 4.5 torr was assumed for CPE. Values for the saturation concentration in air (C_{Sat}) were calculated from the vapor pressure.* The diffusivity in air (D_{air}) was estimated from the molecular weight (Schwope et al., 1989, p. 81).

b. FR Chemical Loading Rates

FR chemical loading rates (L) for AT, DBDPO, HBCD, PA, and THPC are average values from the 8 fabric samples tested in the migration studies conducted by LSC (Bhooshan and Cobb, 2000, Table 1) (see Table II-2a). The fabrics tested included two each treated with DBDPO, HBCD, PA, and THPC. DBDPO and HBCD are used in combination with AT. Thus, there were four AT-treated fabrics. Mean FR loads are used. AT levels are given as antimony. Most authors report CPE, PA, and THPC levels as the weight percent of phosphorus present (e.g., Albright and Wilson, 1998a; Bhooshan and Cobb, 2000). These were converted to the amount of FR chemical. In Tables II-2a and II-2b, the phosphorus content is given in parentheses. The FR loading for CPE (Table II-2b) is from information provided by the manufacturer (Albright and Wilson, 1998a). The loading rate for EHDP is the same value that the NRC report used for tricresyl phosphate (TCP) (NRC, 2000, p. 409). The loading rate for TDCP is the value used by NRC (NRC, 2000, p. 379).

c. FR Chemical Migration

Measurements of FR chemical migration in liquid media (M_L and k_H) were performed by LSC for fabrics treated with AT, DBDPO, HBCD, PA, and THPC (Bhooshan and Cobb, 2000). M_L is a unitless factor that represents the fraction of FR chemical migrating into various liquid media in a static test, that is, the filter paper method (see above). M_L is used to estimate dermal exposure resulting from spilled liquids or cleaning agents. Isotonic saline was used as a surrogate for perspiration and neutral spilled liquids or foods. Five percent citric acid solution was used as a surrogate for acidic beverages or foods. A water-based upholstery cleaner and methyl chloroform were used for the cleaning scenarios. The water-based cleaner is a typical upholstery cleaner sold to consumers. Methyl chloroform is used as a surrogate for dry cleaning solvents. Many of these tests resulted in non-detectable migration. Therefore, the data reported by LSC were reanalyzed by substituting one-half the limit of detection (LOD) for each non-detect. Tests were performed on two fabrics treated with each FR chemical, except AT, for which there were four. The values represent the average from tests on at least 2 fabrics (Table II-3a).

The migration of PA and THPC was measured as the loss of total phosphorus from the fabric (Bhooshan and Cobb, 2000). Some of the phosphorus in saline extracts of PA- and THPC-treated fabrics was inorganic phosphate (Cobb, 2000). Specifically, extracts from the two

* $C_{\text{Sat}} = (\text{MW} \times \text{VP} \times 1000) / (\text{R} \times \text{T})$, where: C_{Sat} , saturation concentration in air, mg/m³; MW, molecular weight, g/mol; VP, vapor pressure, torr; R, gas constant, 62.4 torr-L/mol-°K; T, temperature, 298 °K; and 1000 is to convert from grams to milligrams.

PA-treated fabrics tested contained either 20 or 28 percent inorganic phosphate. Extracts from the two THPC-treated fabrics contained either 5 or 17 percent inorganic phosphate. The fraction of inorganic phosphate was subtracted from the migration measurements (M_L) reported by Bhooshan and Cobb, except in cases where the extent of migration was below the LOD.

The parameter k_H ($\text{mg}/\text{cm}^2\text{-h}$) is the migration rate into saline, as measured by the "head-over-heels" method. This parameter is used to estimate oral exposure due to mouthing by children. As with M_L , each value is the average of tests on at least 2 fabrics. The migration of inorganic phosphate was subtracted from the k_H values reported by Bhooshan and Cobb, as described above for M_L .

LSC also investigated the effect of age and wear on FR chemical migration (Bhooshan and Cobb, 2000). At least one fabric for each FR chemical was subjected to an accelerated aging process, then subjected to the same set of migration tests as the untreated fabrics. One fabric containing AT and DBDPO was also subjected to an accelerated mechanical wear process. Generally, the rate or extent of migration increased by an average of about 2-fold (range, 0.3 to 3.3), as compared to new fabric. Therefore, for the purpose of risk assessment, it will be assumed that the migration rate (k_H) and extent of migration (M_L) are doubled in aged or worn fabric.

Fabrics treated with CPE, EHDP, or TDCP were unavailable for testing (Table II-3b). HBCD was used as a surrogate for EHDP and TDCP, because these three chemicals are hydrophobic and the same application method is being modeled. For CPE, which is water soluble, it was assumed that the unbound fraction was available for migration into saline and aqueous upholstery cleaner. Thus, the fraction migrated (M_L) was assumed to equal one-half of the unbound fraction (F_U). This estimate was applied only for saline and the aqueous upholstery cleaner. The hourly migration rate (k_H) in the head over heels test was assumed to equal one-half of the unbound fraction (F_U , unitless) times the FR chemical load (L , mg/cm^2).

d. Toxicological Parameters

The toxicological values include the acceptable daily intake (ADI), cancer potency, oral bioavailability, and percutaneous absorption rates (Table II-4a,b). ADI values are generally from the CPSC staff toxicity reviews. The ADI's for AT, CPE, DBDPO, EHDP, and THPC have been revised (Bittner, 2001).

A two-year feeding study in rats was performed for TDCP (Biodynamics, 1981). However, only a summary of this study was available to the CPSC staff (reviewed in Ferrante, 1999b). The summary included data on the incidence of tumors, but no data on non-cancer endpoints were provided. Therefore, CPSC did not calculate an ADI for the non-cancer effects of TDCP (Ferrante, 1999b). However, the NRC Subcommittee on Flame Retardant Chemicals, which had access to the complete report (NRC, 2000, p. 377), calculated a reference dose (RfD)* of 0.005 $\text{mg}/\text{kg-d}$ for TDCP, based on a LOAEL of 5.0 $\text{mg}/\text{kg-d}$ for testicular effects in male rats

* Both the acceptable daily intake (ADI) and reference dose (RfD) are estimates of the amount of a chemical a person can be exposed to on a daily basis over an extended period of time (up to a lifetime) with a negligible risk of suffering deleterious effects. The differences between the ADI and RfD are explained in the Discussion.

(testicular atrophy and decreased seminal vesicle secretory product). The NRC RfD of 0.005 mg/kg-d will be used in place of an ADI to calculate the HI. The CPSC staff calculated the cancer potency, based on the risk of tumors in the liver and renal cortex in rats (see above).

Applying the default CPSC uncertainty factors to the LOAEL of 5.0 mg/kg-d would result in an ADI of 0.005 mg/kg-d, which is similar to the RfD calculated by the NRC Subcommittee. Furthermore, because a linear dose response is assumed (CPSC, 1992; NRC, 2000), cancer is a more sensitive endpoint than the non-cancer testicular effects. Therefore, the fact that CPSC did not have access to the data on non-cancer effects does not affect the conclusions of this risk assessment.

The CPSC staff did not calculate an ADI for HBCD because, under the CPSC chronic hazard guidelines (CPSC, 1992), HBCD is regarded as possibly toxic in humans based on limited evidence of toxicity in animals (Hattelid, 1999b; Bittner, 2001). However, the NRC Subcommittee (NRC, 2000, p. 64) calculated a RfD of 0.2 mg/kg-d, based on liver effects in rats (Zeller and Kirsch, 1969). The NRC RfD of 0.2 mg/kg-d will be used in place of an ADI to calculate the HI. The CPSC staff reviewed the same study as NRC (Zeller and Kirsch, 2000), but concluded that it did not support the finding that HBCD is probably toxic under the FHSA.

The CPSC staff did not calculate an ADI for PA, because there was inadequate evidence of toxicity in animals (Bittner, 1999b). Therefore, PA cannot be classified regarding its potential for toxicity in humans. The NRC Subcommittee did not calculate a RfD (NRC, 2000, p. 301). Thus, only an exposure assessment will be performed for PA.

Certain exposure scenarios—direct exposure to spilled liquids or upholstery cleaner—occur intermittently. Because the ADI's are generally based on chronic or subchronic studies, it is unlikely that a one-time exposure exceeding the ADI would have the same effects as daily exposure. An averaging period of one year has been suggested for subchronic effects (Thompson, 1999). For the purpose of the present risk assessment, an averaging time of one year (365 days) will be assumed for non-cancer risks. An averaging time of 75 years (a lifetime) is assumed for estimating cancer risk.

Cancer potency estimates were calculated for AT and TDCP, using the default methods outlined in the CPSC chronic hazard guidelines (CPSC, 1992) (see above). Generally, this involves linear extrapolation from high-to-low doses, and the surface area correction for animal to human extrapolation. For TDCP, the separate potency estimates for liver and renal cortex tumors were added together to give a potency estimate for both sites combined, as described in the chronic hazard guidelines (CPSC, 1992).

Exposure of rats to AT in the form of airborne particles resulted in tumors only at the site of exposure (lung) (reviewed in Hattelid, 1999a). Thus, cancer estimates will be made only for inhalation exposure to particles. Furthermore, the cancer potency is based on the concentration in air and the surface correction was not applied (see above).

Estimates of oral bioavailability, where such data were available, are from the CPSC toxicity reviews. When data were not available, the default value of 1 was assumed. The oral

bioavailability was used only for route to route correction in estimating the risk from dermal exposures (see above, equations (2.13) and (2.18)).

Percutaneous absorption data are from a variety of sources. The available studies involved the application of the FR chemical either as a pure compound or with the FR dissolved in a volatile solvent. First-order kinetics was assumed in calculating the absorption rate or transfer coefficient (k_T) (Scheuplein and Ross, 1974) from the percentage of the applied dose absorbed at a given time, generally 24 hours.

Percutaneous absorption data for DBDPO, HBCD, and TDCP are from *in vitro* studies with hairless mouse skin and flow-through diffusion cells (Hughes, 2000). ^{14}C -Labeled compounds were applied to the skin by solvent deposition. The percentage absorption at 24 hours, including compound in both the receptor fluid and skin, was determined. With TDCP, the greater portion of absorbed compound was in the receptor fluid, the converse being true with HBCD and DBDPO. Percutaneous absorption of TDCP ranged from 73 percent to 85 percent of the applied dose, depending on the applied dose (0.014 to 0.14 $\mu\text{g}/\text{cm}^2$). These values combine FR compound in the receptor fluid and bound within the skin. Based on 85 percent absorption at 24 hours, the percutaneous absorption rate for TDCP is 0.08 h^{-1} . HBCD absorption ranged from 3 percent to 6 percent at applied doses ranging from 0.2 to 2 $\mu\text{g}/\text{cm}^2$. Based on 6 percent absorption at 24 hours, the percutaneous absorption rate for HBCD is 0.003 h^{-1} .

Absorption of DBDPO was highly dependent on the applied dose: about 2 percent absorption was observed at 60 nanomoles (nmol) (90 $\mu\text{g}/\text{cm}^2$), 3 percent at 30 nmol (45 $\mu\text{g}/\text{cm}^2$), and 20 percent at 6 nmol (9 $\mu\text{g}/\text{cm}^2$) (Hughes, 2000). Therefore, different percutaneous absorption rates may be derived, depending on the amount of DBDPO in contact with the skin. A value of 20 percent absorption may be applied when there is less than or equal to 9 $\mu\text{g}/\text{cm}^2$ of DBDPO in contact with the skin. This is equivalent to a percutaneous absorption rate of 0.01 h^{-1} . Three percent absorption may be applied when greater than 9 $\mu\text{g}/\text{cm}^2$ DBDPO is in contact with the skin. This is equivalent to 0.001 h^{-1} .

DBDPO was present in fabrics at an average loading rate (L) of 3.1 mg/cm^2 (Table II-2a). DBDPO migration was greatest with methyl chloroform; the fraction of DBDPO in the liquid phase (M_L) was 0.012 (Table II-3a). Multiplying 3.1 mg/cm^2 by 0.012 gives 0.037 mg/cm^2 or 37 $\mu\text{g}/\text{cm}^2$. The amount of DBDPO contacting the skin is greater than 9 $\mu\text{g}/\text{cm}^2$. DBDPO migration with upholstery cleaner is less than with methyl chloroform, but greater than the other solvents. The extent of migration with upholstery cleaner is only 7.5×10^{-5} ; the amount of DBDPO in contact with the skin is 2.3×10^{-4} mg/cm^2 or 0.23 $\mu\text{g}/\text{cm}^2$, which is less than 9 $\mu\text{g}/\text{cm}^2$. Therefore, a percutaneous absorption rate of 0.001 h^{-1} was used with non-aqueous cleaner, and 0.01 h^{-1} was used in all other cases.

Maibach studied the percutaneous absorption of CPE in monkeys (Maibach, 1979). Pure CPE was applied to the abdominal skin of 3 monkeys at a rate of 42 $\mu\text{g}/\text{cm}^2$. An average of 10.4 percent of the applied dose was recovered in the urine over the course of 8 days. The report did not specify whether the CPE remained on the skin for 8 days, or it was removed after 24 hours, as is frequently done in experiments of this type. Assuming that the CPE was applied for only 24 hours, P_{24} is 0.10 and k_T is about 0.005 h^{-1} .

Percutaneous absorption data for EHDP were not available. Percutaneous absorption data for *o*-tricresyl phosphate (TCP) were used as a surrogate for EHDP. These two compounds are members of the same chemical class (aromatic phosphates) and have roughly similar physico-chemical properties. When pure TCP was applied to the skin of male felines *in vivo*, 73 percent of the applied dose of pure TCP was absorbed in 12 hours (as cited in Ferrante, 1999a, p. 24).

Percutaneous absorption data also were unavailable for AT, PA, and THPC. The rate of percutaneous absorption depends, in part, on the K_{ow} and molecular weight. PA and THPC have log K_{ow} values and molecular weights that are roughly comparable to those of CPE (Table II-2b). For the purpose of the present risk assessment, it will be assumed that 10 percent of PA and THPC are absorbed in 24 hours, as observed with CPE (Maibach, 1979).

Inorganic compounds are generally absorbed at low, but detectable rates (reviewed in EPA, 1992; see also Hughes et al., 1995; Rahman and Hughes, 1994; Rahman et al., 1994). Therefore, for the purpose of the present risk assessment, it will be assumed that 5 percent of AT is absorbed in 24 hours.

3. Scenario-Specific Parameters

Scenario-specific parameters are those which are related to the manner in which consumers use and interact with upholstered furniture. In general, these parameter values depend on the specific scenarios (see Introduction).

a. Dermal Exposure

The parameters used for estimating dermal exposure are summarized in Table II-5.

Passive Dermal Exposure (Scenarios D.1, D.2.a, and D.2.b).* Passive dermal exposure involves direct contact of the skin with upholstery fabric, such as when sitting on the furniture. The CPSC Division of Human Factors estimated the skin surface area in contact with upholstered furniture based on the assumption that the consumer is lying on a sofa and wearing a short-sleeved shirt and short pants (Smith, 2000). This results in an average skin surface area of approximately 0.25 m² (2500 cm²) for adults and 0.05 m² (500 cm²) for children (Table II-5). Based on compiled activity data (EPA, 1997b), Human Factors estimated a total exposure duration of approximately 4 hours per day for both children and adults (Smith, 2000). The total daily exposure duration of 4 hours is likely to be divided among several events (Smith, 2000). However, only the total number of hours per day is needed to estimate exposure by the methods used in this risk assessment.

Passive dermal exposure also occurs when spilled liquids (D.2.a) or cleaning products (D.2.b) dry on the fabric surface. Dried liquids may deposit extracted FR chemical on the fabric surface, where subsequent passive exposure may occur when a consumer sits on the upholstered furniture.

* The scenario designations are described in Table 2 in section I.D, Scope of the Risk Assessment.

Even if the spilled liquid or cleaning agent is dried with a towel, a portion of the liquid is likely to remain. Thus, passive exposure to spilled liquids or cleaners depends, in part, on the fraction of total liquid that is retained by the fabric after the liquid is blotted dry (F_F). LSC performed experiments in which the fabrics used for the migration studies were saturated with saline solution and then blotted dry (Bhooshan, 2000). On average, the fraction of retained liquid was 0.4. Most of these fabrics were cellulosic, which tend to absorb and retain more water than synthetic fibers. Therefore, this may overestimate exposure for non-cellulosic fabrics exposed to aqueous solvents or for cellulosic fabrics exposed to non-aqueous solvents.

Spills are likely to be frequent occurrences, but they probably do not involve the entire piece of furniture. Thus, at any given time, it is likely that a portion of a piece of furniture has been exposed to spilled liquids. For the purpose of the present risk assessment, it will be assumed that passive exposure from spilled liquids (D.2.a) and cleaning (D.2.b) is continuous, and that the entire surface of the furniture is affected by both events. Thus, it was assumed that the skin surface area and exposure duration for each of these scenarios is the same as for the normal use scenario (D.1). It will be further assumed that exposure from all of the passive dermal exposure pathways (D.1, D.2.a, and D.2.b) is additive. In reality, it is unlikely that an entire piece of furniture is subject to spills. Cleaning an entire piece of furniture is possible, although it is believed to be infrequent (see below). Therefore, the assumption that all of the furniture is affected by both spills and cleaning, as well as the assumption that the processes are additive, clearly will tend to overestimate exposure.

Active Dermal Exposure (D.3.a and D.3.b). Active dermal exposure occurs when spilled liquids or cleaning agents facilitate the migration of FR chemicals from upholstery fabric to the skin. In the direct liquid mediated exposure scenarios, consumers are directly exposed to either spilled liquid (D.3.a) or liquid cleaner (D.3.b) that contains extracted FR chemical. While cleaning may range from spot cleaning to cleaning an entire suite of furniture, only spot cleaning is modeled herein. For both spills and spot cleaning, it was assumed that one side of one hand is directly exposed to the liquid. For adults, the mean surface area of the hands is about 0.084 m^2 in men and 0.075 m^2 in women (EPA, 1997a, Table 6-4). Averaging the two values and dividing by four, gives a surface area for one side of one adult hand of about 0.02 m^2 (200 cm^2). For children up to two years old, the hands are about 5.5 percent of the total body surface area. This was obtained by averaging the fraction of the total surface area for the hands in children <1 year old and from 1 to 2 years old (EPA, 1997a, Table 6-8). Data on the total body surface area of children under 2 years old were not available. Therefore, the 15th percentile values for boys and girls from 2 to 3 years old were averaged (EPA, 1997b, Tables 6-6 and 6-7). Multiplying 5.5 percent by the total body surface area results in a surface area for both sides of the hands of 0.03 m^2 (300 cm^2). Dividing by four gives the surface area for one side of one hand, 75 cm^2 .

Little information is available regarding the frequency of spills or the use of upholstery cleaners. Consumers are reported to use spot cleaners an average of 16 times per year (EPA, 1997b, as cited in Smith, 2000), although this is not limited to upholstered furniture. For the purpose of risk assessment, it will be assumed that spills occur monthly and that spilled liquid is in contact with the skin for one-tenth of an hour. Furthermore, it will be assumed that consumers use spot cleaners on upholstered furniture for one-tenth of an hour each month, as well. Only adults are assumed to spot clean furniture. Both adults and children are assumed to be exposed

to spills: adults by cleaning up the spills and children by contacting the spilled liquid. It is further assumed that active exposure from spills (D.3.a) and spot cleaning (D.3.b) occur independently, but that the resulting exposures are additive. Active exposure from spills and spot cleaning were assumed to be in addition to passive exposures (D.1, D.2.a., and D.2.b).

As discussed earlier, the averaging time for estimating the ADD for non-cancer endpoints is assumed to be one year. Thus, these intermittent exposures are averaged over the course of a year. Consequently, the number of days exposed per each averaging period (T_A) is the same as the number of exposures per year (N_Y).

b. Oral Exposure

Oral exposure is assumed to occur when children mouth portions of upholstery fabric. The parameters used to estimate oral exposure are summarized in Table II-6. The fabric surface area is the value previously used by CPSC staff to estimate exposure from the mouthing of teething toys, and pacifiers (CPSC, 1983; CPSC, 1998). Based on an observational study of children's mouthing activity, mouthing of objects other than toys and parts of the anatomy is expected to occur frequently, but for a limited duration (Smith and Kiss, 1998; Smith, 2000). HF staff estimates that one-year-olds would mouth such objects up to 9 times per hour, but for a total duration of no more than 1.4 minutes per hour (Smith, 2000). If the child is awake for 10 ¼ hours per day (Nelson, 1996), this results in a total of 15 minutes per day.

c. Inhalation Exposure

Parameters specific to inhalation exposure are listed in Table II-7. This risk assessment considers exposure to both vapor phase and particle-bound FR chemicals. Inhalation rates are long-term average values; the adult value is the average for men and women (EPA, 1997a). The room volume was calculated for a 140 m² (1500 ft²) ranch house, assuming 2.4 m (8 ft) high ceilings (DOE, 1992; Traynor et al., 1989). The air infiltration rate is the median value for all seasons and all regions in the U.S. (Koontz and Rector, 1993).

The furniture fabric areas were measured from a typical suite of furniture including a sofa, love seat, and chair. Only fabric directly exposed to air was measured. The fraction of the fabric area that is subject to heavy wear is used to estimate the release of airborne particles. It was assumed that 50 percent of the horizontal surface area is subject to heavy wear, which leads to a worn fraction of 0.15.

The exposure duration (16 h/d) is from compiled data, and represents the amount of time that consumers spend in their residences each day (EPA, 1997b, p. 15-7) (Table II-7). The number of exposures per day is assumed to be one, although the total exposure duration may be divided among several events. This does not affect the exposure estimates. The number of days per year exposed is assumed to be 365.

The particle release rate is based on the assumption that the worn fraction is released over the lifetime of the furniture (15 years) and that 1 percent of the released particles are of respirable size (NRC, 2000, p. 43). This results in a particle release rate of 8×10^{-8} per hour.

Multiplying this by the worn fraction and converting to a daily rate gives a value of 2.9×10^{-7} per day, which is roughly similar to the value assumed by NRC, 2.3×10^{-7} per day, (NRC, 2000, p. 43). The particle decay rate is a published value for particles 1 to 5 μm in diameter (EPA, 1997b, Table 17-3).

The boundary layer thickness is required to estimate vapor phase exposure for immersion-treated fabrics; this is the same value used by NRC (NRC, 2000, p. 46). The fraction of fabric or polymer exposed to air, F_A , is required to estimate exposure to vapor phase FR chemicals. For most fabrics, a value of 1 was used, because only fabric exposed to air was included when the fabric surface area was measured. For back-coated fabrics, the use of a value of 1 was based on the assumption that the polymer and the fabric would not impede the migration of FR chemical from the polymer to the air. This probably tends to overestimate exposure for back-coated fabrics. However, values less than 1 were used for PA and THPC, which are reactive chemicals. Most of the PA or THPC present in fabric is covalently bound to the fabric fiber or present as a polymer. The extent of migration into saline (M_L) in the filter paper assay (Table II-3a) was used to estimate the fraction of PA and THPC that is not covalently bound and, therefore, "exposed" to air. Saline is an appropriate solvent for these chemicals, because they are water-soluble.

Table II-1. General input parameters.

	Parameter	Adults	Children	Reference
Y _F ,	Furniture lifetime, years	15	15	NRC, 2000; Ray, 2000
Y _i ,	Years of product exposure, years	75	2	Assumed ^a
Y _E ,	Life expectancy, years	75	75	EPA, 1997a, p. 8-1
W,	Body weight, kg	72	15	EPA, 1997a, Table 7-2 Greene, 1998

^a A lifetime of exposure is assumed for adults. For children, the time from birth to age 2 is the period during which mouthing activity is most frequent.

Table II-2a. Physico-chemical properties and FR chemical loading rates—chemicals with migration data.

Parameter	AT	DBDPO	HBCD	PA	THPC
MW, Molecular weight, g/mol ^a	291.5	960.0	641.7	211.2	190.6
VP, Vapor pressure, torr ^a	1x10 ⁻¹⁰	3.5x10 ⁻⁸	4.7x10 ⁻⁷	17.25	1 ^b
C _{Sat} , Saturation concentration in air, mg/m ^{3d}	1.6x10 ⁻⁶	1.8x10 ⁻³	1.6x10 ⁻²	2.0x10 ⁵	1x10 ⁴
D _{Air} , Diffusivity in air, m ² /h ^e	NA ^c	0.008	0.01	0.017	0.017
Log K _{ow} , Log octanol: water partition coefficient, unitless ^a	NA	6.265	5.6	-1.68	-1.15
Application method ^f	BC	BC	BC	IR	IR
L, FR chemical load, as: ^g					
Percent (w/w) ^h	Sb	DBDPO	HBCD	PA (P)	THPC (P)
mg/cm ² ^h	2.3	6.5	9.2	9.3 (1.4)	13 (2.1)
	1.0	3.1	3.4	2.4 (0.36)	4.5 (0.73)

^a Physico-chemical properties are generally from secondary sources reviewed in the CPSC staff toxicity reviews.

^b As cited in NRC, 2000.

^c NA, not applicable.

^d Calculated from the vapor pressure and molecular weight.

^e Calculated from the molecular weight, as described in Schwowe et al., 1989, p. 81.

^f BC, back-coated; IH, immersion treated with heat cure; IR, immersion treatment with reactive chemical.

^g Loading data are from Bhooshan and Cobb, 2000. AT levels are given as the antimony content. PA and THPC levels are given as the chemical weight, with phosphorus content in parentheses.

^h The number in parentheses is the phosphorus content.

Table II-2b. Physico-chemical properties and FR chemical loading rates—chemicals without migration data.

Parameter	CPE ^a		EHDP	TDCP
	washed	unwashed		
MW,	286.2	286.2	362.4	430.9
VP,	4.5 ^c	4.5 ^c	1x10 ⁻⁵	0.01
C _{Sat} ,	6.9x10 ⁻⁴	1.5x10 ⁻⁴	1.2	230
D _{Air} ,	0.014	0.014	0.013	0.012
Log K _{ow}	-1.07	-1.07	5.73	3.76
Log octanol: water partition coefficient, unitless ^b				
Application method ^f	IH	IH	BC	BC
L, FR chemical load, as: ^g				
Percent (w/w)	CPE (P) 0.9 (0.2)	CPE (P) 1.8 (0.4)	EHDP 10	TDCP 10.5
mg/cm ²	0.4 (0.1)	0.8 (0.2)	4.8	5.0

^a CPE-treated fabrics are generally washed (scoured) to remove excess CPE, although this step is sometimes omitted, which may affect exposure.

^b Physico-chemical properties are generally from secondary sources reviewed in the CPSC staff toxicity reviews.

^c Assumed. The vapor pressure of dimethyl phosphonate, a related compound, is 4.5 (see text).

^d Calculated from the vapor pressure and molecular weight.

^e Calculated from MW as described in Schwobe et al., 1989, p. 81.

^f BC, back-coated; IH, immersion treated with heat cure; IR, immersion treatment with reactive chemical.

^g Loading rates are assumed. CPE is from (Albright and Wilson, 1998a); the numbers in parentheses are the phosphorus content. EHDP is the value NRC used for o-tricresyl phosphate, a related compound (NRC, 2000, p. 409). TDCP is from (NRC, 2000, p. 409).

Table II-3a. FR chemical migration—chemicals with migration data. ^a

Parameter	AT	DBDPO	HBCD	PA ^b	THPC ^b
M _L Fraction in liquid phase, unitless:					
Saline	1.0x10 ⁻³	2.0x10 ^{-5c}	6.0x10 ⁻⁴	6.6x10 ⁻²	2.7x10 ⁻²
Citric acid	2.0x10 ⁻¹	2.0x10 ⁻⁵	2.3x10 ⁻⁴	2.1x10 ⁻¹	2.7x10 ⁻²
Upholstery cleaner ^d	9.1x10 ⁻⁴	7.5x10 ⁻⁵	2.6x10 ⁻⁴	6.7x10 ⁻²	2.4x10 ⁻²
Methyl chloroform	1.0x10⁻⁴	1.2x10 ⁻²	1.3x10 ⁻¹	6.1x10⁻⁴	3.2x10⁻⁴
K _H Head-over-heels migration rate, mg/cm ² -h ^a	1.2x10 ⁻³	6.2x10 ⁻⁴	1.3x10 ⁻²	8.5x10 ⁻²	3.0x10 ⁻²

^a Migration data (M_L and K_H) are from Bhooshan and Cobb, 2000.

^b The fraction of inorganic phosphate (Cobb, 2000) was subtracted, except when the migration was below the detection limit (LOD).

^c Values in bold indicate that some or all measurements were below the LOD. Measurements below the LOD are assumed to equal one-half the LOD.

^d Referred to as cleaner 2 in Bhooshan and Cobb, 2000.

Table II-3b. FR chemical migration—chemicals without migration data.

Parameter	CPE ^a washed	CPE ^a unwashed	EHDP ^b	TDCP ^b
M _L , Fraction in liquid phase, unitless:				
Saline	2.5x10 ⁻²	2.5x10 ⁻¹	6.0x10 ⁻⁴	6.0x10 ⁻⁴
Citric acid	NA ^c	NA	2.3x10 ⁻⁴	2.3x10 ⁻⁴
Upholstery cleaner ^d	2.5x10 ⁻²	2.5x10 ⁻¹	2.6x10 ⁻⁴	2.6x10 ⁻⁴
Methyl chloroform	NA	NA	1.3x10 ⁻¹	1.3x10 ⁻¹
k _H , Head-over-heels migration rate, mg/cm ² -h	2.5x10 ⁻³	2.5x10 ⁻²	1.3x10 ⁻²	1.3x10 ⁻²

^a Migration values for CPE are assumed to equal the unbound fraction (see text). CPE-treated fabrics are generally washed (scoured) to remove excess CPE, although this step is sometimes omitted, which may affect exposure.

^b Values for EHDP and TDCP (M_L and k_H) are assumed to be equal to those for HBCD.

^c NA, not applicable.

^d Referred to as cleaner 2 in Bhooshan and Cobb, 2000.

Table II-4a. Toxicological parameters for FR chemicals—chemicals with migration data.

Parameter	AT	DBDPO	HBCD	PA	THPC
Critical toxic endpoint(s)/sites: ^a Oral exposure	systemic effects	liver	liver	NA ^b	liver, neurotoxicity
Inhalation exposure	lung cancer, fibrosis)	NA	NA	NA	NA
ADI, Acceptable daily intake, mg/kg-d	2.3 ^c	3.2 ^d	0.2 ^e	NA	0.0027
ADI _I , Inhalation ADI, mg/m ³	9x10 ⁻⁶ ^c	NA	NA	NA	NA
T _A , Averaging time, days ^f	365	365	365	365	365
Q, Cancer potency, (mg/kg-d) ⁻¹	NA	NA	NA	NA	NA
Q _I , Inhalation cancer potency, (mg/m ³) ⁻¹	0.51 ^c	NA	NA	NA	NA
B, Oral bioavailability, unitless	0.03	0.01	1 ^g	1 ^g	1 ^g
k _T , Dermal absorption rate ^h , h ⁻¹	0.002 ^f	0.01 or 0.001 ⁱ	0.003	0.004 ^f	0.004 ^f

^a Toxicity information is from the CPSC staff toxicity reviews, except where indicated.

^b NA, not applicable or data not available.

^c As antimony.

^d Revised value (Bittner, 2001).

^e The substance is not considered "toxic" under the FHSA. The RfD calculated by NRC is used (NRC, 2000, p. 64).

^f Assumed value (Thompson, 1999). Applies to non-cancer endpoints.

^g Default value.

^h Calculated from P₂₄ assuming first-order kinetics.

ⁱ A percutaneous absorption rate of 0.001 h⁻¹ was used at applied doses ≥ 9 μg/cm² (0.009 mg/cm²) (see text).

Table II-4b. Toxicological parameters for FR chemicals—chemicals without migration data.

Parameter	CPE	EHDP	TDCP
Critical toxic endpoint(s)/sites: ^a			
Oral exposure	systemic effects	organ toxicity (liver, adrenal)	cancer (liver, renal cortex)
Inhalation exposure	NA ^b	NA	NA
ADI, Acceptable daily intake, mg/kg-d	10 ^c	1.0	0.005 ^d
ADI _i Inhalation ADI, mg/m ³	NA	NA	NA
T _A , Averaging time, days ^e	365	365	365
Q, Cancer potency, (mg/kg-d) ⁻¹	NA	NA	6.2x10 ⁻³
Q _i , Inhalation cancer potency, (mg/m ³) ⁻¹	NA	NA	NA
B, Oral bioavailability, unitless	1 ^f	1 ^f	1 ^f
k _T , Dermal absorption rate ^g , h ⁻¹	0.005	0.1 ^h	0.08

^a Toxicity information is from the CPSC staff toxicity reviews, except where indicated.

^b NA, not applicable or data not available.

^c Revised value (Bittner, 2001).

^d CPSC did not calculate an ADI for non-cancer effects. The RfD calculated by NRC is used (NRC, 2000, p. 377).

^e Assumed value.

^f Default value.

^g Calculated from P₂₄ assuming first-order kinetics.

^h o-Tricresyl phosphate is used as a surrogate compound.

Table II-5. Dermal Exposure Parameters.

Parameter	Exposure Scenario					
	D.1 ^a	D.2.a	D.2.b	D.3.a	D.3.b	
A _s , Skin surface area, cm ² ^b	Adults	2500	2500	200	200	
	Children	500	500	75	NA	
F _F , Residual fraction in fabric, unitless ^c	1.0	0.4	0.4	1.0	1.0	
T _i , Exposure duration, h ^b	4	4	4	0.1	0.1	
N _i , Exposures per day, d ⁻¹ ^b	1	1	1	1	1	
N _y , Days per year exposed, d/y ^b	365	365	365	12	12	
T _A , Averaging time, days ^d	NA ^e	NA	NA	365	365	
N _A , Days exposed per unit T _A , days ^f	NA	12	126	NA	NA	

^a The scenario designations are described in Table I-2 in section I.D, Scope of the Risk Assessment.

^b Estimated. See text for estimation.

^c Bhooshan, 2000.

^d Assumed. See text for explanation.

^e NA, not applicable.

^f This is the same as days per year exposed, N_y, because the averaging time is one year.

Table II-6. Oral Exposure Parameter.

Parameter	Value	Source
k_H , Migration rate, mg/cm ² -h	see Table II-3	Bhooshan and Cobb, 2000
A_F , Fabric surface area, cm ²	11	CPSC, 1983
T , Exposure duration, h	0.25 ^a	Smith and Kiss, 1998; Smith, 2000
N , Exposures per day, d ⁻¹	1 ^a	Smith, 2000
N_Y , Days per year exposed, d/y	365	Assumed

- ^a Observations suggest that there would be multiple exposures, with a total mouthing time of up to 1.4 minutes per waking hour. The total of 0.25 hours (15 minutes) is based on 10.25 waking hours (Nelson, 1996).

Table II-7. Inhalation Exposure Parameters.

Parameter	Value	Source	
I,	Inhalation rate, m ³ /h	EPA, 1997a, Table 5-23	
	Adults	0.55	
	Children	0.28	
V,	Room volume, m ³	DOE, 1992; Traynor et al., 1989	
ACH,	Air infiltration rate, h ⁻¹	Koontz & Rector, 1993	
A _F ,	Fabric area, total, m ²	Measured	
A _{FH} ,	Horizontal	3.0	
A _{FV} ,	Vertical	7.0	
T,	Exposure duration, h	EPA, 1997b, p. 15-17	
N,	Exposures per day, d ⁻¹	EPA, 1997b, p. 15-17	
N _Y ,	Days per year exposed, d/y	Assumed	
<u>Particles (I.2)</u>			
F _W	Worn fraction, unitless	0.15	Assumed (see text)
k _R ,	Particle release rate, h ⁻¹	1x10 ⁻⁷	Assumed
k _D ,	Particle decay rate, h ⁻¹	0.5	EPA, 1997b, Table 17-13
<u>Vapor Phase (I.1)</u>			
F _A	Fraction exposed to air, unitless:		
	PA	0.03	Fraction migrating into saline
	THPC	0.086	Fraction migrating into saline
	All others	1	Assumed highest possible value
H,	Boundary layer thickness, m	0.01	NRC, 2000, p. 46

III. Results

Aggregate doses and risks were calculated for several different “cases.” The cases are described in Table I-3. Each case represents the aggregate time-averaged exposure (ADD or LADD) and associated risk (HI or cancer risk) from all three routes of exposure and a total of seven different scenarios. Tables III-1 and III-3 to III-10 give the ADD’s and HI’s for the various cases. The ADD’s and HI’s for the basic case are also given by the route of exposure and exposure scenario. The same applies for the LADD’s and cancer risks for TDCP in Table II-11. This information shows which of the various routes and scenarios contribute significantly to the aggregate risk. However, this level of detail is not included for the acidic spill, non-aqueous cleaner, or aged furniture cases. Table III-2 gives the estimated cancer risk associated with the inhalation of particles containing AT.

A. Chemicals with Migration Data

1. Antimony Trioxide (AT)

AT is generally applied in a back-coating, where it is used only in combination with other FR chemicals, mainly DBDPO and HBCD. AT is not acutely toxic. However, AT is considered “toxic” under the FHSA, based on sufficient evidence of chronic toxicity in animals (Hattelid, 1999a). AT caused systemic effects following subchronic oral exposure in several animal species. The CPSC staff derived an ADI of 2.3 mg/kg-d. Furthermore, inhalation of AT dusts caused fibrosis in guinea pigs and rats. AT also caused lung tumors in two of three studies in rats. For inhalation exposure to AT dusts, the CPSC staff derived an acceptable exposure level of 9×10^{-6} mg/m³ (Hattelid, 1999a) and a cancer unit risk of $0.51 \text{ (mg/m}^3\text{)}^{-1}$ (see Methodology).

AT-treated fabric samples were available for testing by LSC, which conducted studies of liquid-mediated migration applicable to dermal and oral exposures (Bhooshan and Cobb, 2000). Percutaneous absorption data were not available. ADD estimates for dermal scenarios are based on an assumed percutaneous absorption rate of 5 percent in 24 hours.

The “basic case” represents the aggregate exposure and risk from all three routes of exposure and a total of seven different pathways. The HI for the basic case (systemic effects) was 0.007 in adults and in children (Table III-1). In all cases, the HI for systemic effects was less than 1.0. When AT-treated fabrics were exposed to citric acid, the extent of migration was higher than with other treatments (see Table II-3a). Thus, the HI for the acidic spill case was 0.33. In adults, dermal exposure was the primary route of exposure; the contribution to systemic exposure from inhalation of particles was negligible. In children, oral exposure from mouthing contributed two percent of the total risk (HI).

AT is also considered toxic by inhalation. The inhalation hazard index for non-cancer effects was 0.26 in both adults and children (Table III-2). The lifetime individual excess cancer risk was estimated to be 1.2 per million in adults and 0.03 per million in children. The risk estimate in children represents the contribution to the total lifetime risk from exposure during the first two years of life. In other words, the 1.2 per million risk in adults includes the 0.03 per million risk in children.

It does not appear that AT would present a hazard to consumers due to either dermal or oral exposure. However, the inhalation of particles containing AT is a possible concern. There is considerable uncertainty in the estimated exposure to airborne particles, which was based entirely on mathematical models. Although the HI for inhalation is only 0.26, the true HI could be higher or lower. The estimated cancer risk is about one-in-a-million, but this estimate is subject to the same uncertainties. Cancer risks greater than one-in-a-million are considered to be hazardous, as defined by the FHSA (CPSC, 1992). Data on exposure to airborne particles containing AT are needed to determine whether AT could present a risk to consumers.

2. Decabromodiphenyl oxide (DBDPO)

DBDPO is generally applied in a back-coating, where it is used in combination with AT. DBDPO is not acutely toxic. However, DBDPO is considered “toxic” under the FHSA, based on sufficient evidence of chronic toxicity in animals (reviewed in Bittner, 1999a). DBDPO caused effects in the liver following subchronic or chronic oral exposure in rats and mice. The CPSC staff derived an ADI of 3.2 mg/kg-d. The percutaneous absorption rate was measured *in vitro* (Hughes, 2000). DBDPO-treated fabric samples were available for testing by LSC, which conducted studies of liquid-mediated migration applicable to dermal and oral exposures (Bhooshan and Cobb, 2000). In many cases, migration of DBDPO was below the detection limit. In these cases, one-half the limit of detection was used to estimate exposure. With the filter paper method, which is used to estimate dermal exposure, migration was not-detectable with saline, citric acid, or aqueous cleaner as the solvent (see Table II-3a). Detectable migration occurred with methyl chloroform (non-aqueous cleaner). Migration was also detectable with the head-over-heels method, which was used to estimate oral exposure.

The HI for the basic case was 0.008 in adults and in children (Table III-3). The HI was less than 1.0 in all cases. When DBDPO-treated fabrics were exposed to methyl chloroform, the extent of migration was higher than with other treatments (see Table II-3a). Thus, the HI for the non-aqueous cleaner case was 0.07. In adults and children, dermal exposure was the primary route of exposure; the contribution from inhalation of particles was negligible. In children, oral exposure from mouthing contributed less than 1 percent of the total risk (HI). It does not appear that DBDPO would present a hazard to consumers.

3. Hexabromocyclododecane (HBCD)

HBCD is generally applied in a back-coating, where it is used in combination with AT. HBCD is not acutely toxic. Furthermore, HBCD did not satisfy the definition of chronic toxicity under the FHSA, because there was only limited evidence of toxicity in animals (Hatlid, 1999b). However, the NRC calculated an RfD of 0.2 mg/kg-d, based on liver effects in a 13-week study in rats (NRC, 2000, p.64). The percutaneous absorption rate was measured *in vitro* (Hughes, 2000). HBCD-treated fabric samples were available for testing by LSC, which conducted studies of liquid-mediated migration applicable to dermal and oral exposures (Bhooshan and Cobb, 2000). As with DBDPO, the migration rates used to estimate dermal exposure were below the detection limit with saline, citric acid, and aqueous cleaner. Detectable

migration was observed with non-aqueous cleaner. Detectable migration also was observed in the test used to estimate oral exposure.

The HI for the basic case was 0.007 in adults and 0.020 in children (Table III-4). The HI was less than 1.0 in all cases. When HBCD-treated fabrics were exposed to methyl chloroform, the extent of migration was higher than with other treatments (see Table II-3a). Thus, the HI for the non-aqueous cleaner case was 0.37. As with DBDPO, dermal exposure was the primary route of exposure to adults; the contribution from inhalation was negligible. In children, oral exposure from mouthing is predicted to be the primary route of exposure, contributing about 60 percent of the total risk (HI). Nevertheless, the HI in children ranged from 0.020, for the basic case, to 0.37, if non-aqueous cleaners are used. It does not appear that HBCD would present a hazard to consumers.

4. Phosphonic Acid, (3-{{[Hydroxymethyl]Amino}}-3-Oxopropyl)-, Dimethyl Ester (PA)

PA is a reactive FR treatment that is used with cellulosic fabrics. It reacts with the cellulose fibers and/or permanent press resins. PA did not satisfy the definition of “toxic” under the FHSA (Bittner, 1999b). The NRC concluded that there were insufficient data to calculate an RfD for PA (NRC, 2000, p. 301). Therefore, only exposure estimates are presented here. PA-treated fabric samples were available for testing by LSC, which conducted studies of liquid-mediated migration applicable to dermal and oral exposures (Bhooshan and Cobb, 2000). LSC measured the migration of total phosphorus from PA-treated fabrics. LSC also measured the percentage of total phosphorus that was in the form of inorganic phosphate (Cobb, 2000). The fraction of inorganic phosphate was subtracted from the total phosphorus, except in cases where the extent of migration was below the LOD (see Methodology). Exposure estimates were then calculated as mg PA/kg-day. Percutaneous absorption data were not available. ADD estimates for dermal scenarios are based on an assumed absorption rate of 10 percent in 24 hours.

The ADD for the basic case was 0.16 mg/kg-d (as PA) in adults and 0.17 mg/kg-d in children (Table III-5). The extent of migration was somewhat greater with citric acid (see Table II-3a). Thus, the ADD for the acidic spill case was 0.24. In adults, dermal exposure was the primary route of exposure; inhalation of vapors accounted for only 0.02 percent of the ADD. In children, oral exposure from mouthing contributed 9 percent of the total exposure.

PA did not satisfy the definition of “toxic” under the FHSA and, therefore, it is not considered “hazardous” under the FHSA. Thus HI values were not calculated.

5. Tetrakis(Hydroxymethyl)Phosphonium Chloride (THPC)

THPC is a reactive FR treatment that is used with cellulosic fabrics. THPC and THPC-urea polymerize within the cellulose fibers. THPC-treated fabric samples were available for testing by LSC, which conducted studies of liquid-mediated migration applicable to dermal and oral exposures (Bhooshan and Cobb, 2000). LSC measured the migration of total phosphorus from THPC-treated fabrics. LSC also measured the percentage of total phosphorus that was in the form of inorganic phosphate (Cobb, 2000). The fraction of inorganic phosphate was

subtracted from the total phosphorus, except in cases where the extent of migration was below the LOD (see Methodology). Exposure estimates were calculated as mg THPC/kg-day. Both THPC and THPC-urea are considered “toxic” under the FHSA, based on sufficient evidence of chronic toxicity in animals (Bittner, 1999c). However, THPC was not detected in the extracts from the LSC migration studies (Cobb, 2000). The principal chemical species present in the extracts and their possible toxic effects are unknown. Therefore, an exposure assessment only is presented for THPC. Percutaneous absorption data were not available. ADD estimates for dermal scenarios are based on an assumed absorption rate of 10 percent in 24 hours.

The ADD for the basic case was 0.12 mg/kg-d (as THPC) in adults and children (Table III-6). The extent of migration did not vary substantially among the aqueous solvents, but was somewhat lower with non-aqueous cleaner. It should be noted that the extent of migration declined significantly following successive extractions (see Discussion). Inhalation exposure contributed only 0.4 percent of the aggregate ADD in adults and children. Oral exposure from mouthing contributed about 5 percent of the aggregate ADD in children.

It cannot be determined whether THPC-treated fabric may present a hazard to consumers. Additional information is needed on the identity and toxicity of the chemical species migrating from THPC-treated fabrics. However, if the compounds in the extracts were as toxic as THPC, then THPC-treated fabrics would likely present a hazard to consumers, as the predicted ADD's (0.09 to 0.17 mg/kg-d) were greater than the ADI for THPC (0.0027 mg/kg-d).

B. Chemicals without Migration Data

1. Cyclic Phosphonate Esters (CPE)

CPE may be applied by various methods. However, for use in apparel, it is generally applied to synthetic fabrics by immersion and heat curing, which fixes a portion of the CPE within the fibers. Unbound CPE may be removed by washing the treated fabric, but this step is sometimes omitted. Thus, risk assessments for both washed and unwashed fabrics are presented here. CPE is not acutely toxic. However, CPE is considered “toxic” under the FHSA, based on sufficient evidence of chronic toxicity in animals (reviewed in Hatlelid, 1999c). CPE caused systemic effects following subchronic oral exposure in various species. The CPSC staff derived an ADI of 10 mg/kg-d (Bittner, 2001). The percutaneous absorption rate was measured *in vivo* in monkeys (Maibach, 1979). CPE-treated fabric samples were not available for testing by LSC. To estimate exposure, it was assumed that unbound CPE would be available for migration into aqueous solvents, because CPE is water soluble.

With washed fabrics, the HI for the basic case was 0.001 in adults and in children (Table III-7). The HI was less than 1.0 in all cases. In adults, inhalation of vapor phase CPE contributed about 2 percent of the (HI). In children, inhalation exposure and mouthing each contributed about 4 percent of the total risk (HI).

With unwashed fabrics, the HI for the basic case was 0.025 in adults and children (Table III-8). Again, the HI was less than 1.0 in all cases. Therefore, it does not appear that CPE would present a hazard to consumers.

2. 2-Ethylhexyl Diphenyl Phosphate (EHDP)

It is anticipated that EHDP, an aromatic phosphate, would be applied in a back-coating. EHDP is not acutely toxic. However, EHDP is considered “toxic” under the FHSA, based on sufficient evidence of chronic toxicity in animals (reviewed in Ferrante, 1999a). The CPSC staff derived an ADI of 1.0 mg/kg-d, based on histopathological effects in multiple organs in rats fed EHDP for 90 days (Bittner, 2001). EHDP-treated fabric samples were not available for testing by LSC. HBCD was used as a surrogate compound for estimating dermal and oral exposure. Percutaneous absorption data were not available. *o*-Tricresyl phosphate (*o*-TCP) was used as a surrogate compound for estimating percutaneous absorption. Dermal absorption of *o*-tricresyl phosphate was measured *in vivo* in felines (as cited in Ferrante, 1999a).

The HI for the basic case was 0.066 in adults and 0.069 in children (Table III-9). The HI was less than 1.0 in all but one case. When HBCD-treated fabrics were exposed to methyl chloroform, the extent of migration was considerably higher than with other treatments (see Table II-3a). The same is expected to be true for EHDP, which is also hydrophobic. Thus, the HI for the non-aqueous cleaner case was 3.5 in adults. This means that the aggregate exposure may exceed the ADI when the fabric is exposed to non-aqueous cleaners. No other HI's were greater than 1.0. Therefore, it appears that EHDP would not present a hazard under most conditions. However, it could present a hazard if the fabric were cleaned with dry cleaning fluid. It should also be noted that the dermal and oral exposure assessments and dermal bioavailability estimate are based on a surrogate compounds (HBCD and *o*-TCP, respectively). The actual exposure to EHDP and the resulting HI could be lower or higher.

3. Tris(1,3-dichloropropyl-2) Phosphate (TDCP)

TDCP could be applied by a variety of methods. This risk assessment assumes that TDCP would be applied in a back-coating. TDCP is considered to be acutely toxic under the FHSA (reviewed in Ferrante, 1999b). Furthermore, TDCP is considered to be a chronic toxicant under the FHSA, based on sufficient evidence of carcinogenicity in animals. TDCP caused tumors in the liver and renal cortex in rats. The CPSC staff derived a cancer potency estimate of $6.2 \times 10^{-3} \text{ (mg/kg-d)}^{-1}$ (see Methodology). The CPSC did not derive an ADI for the non-cancer effects of TDCP. However, the NRC Subcommittee derived an RfD of 0.005 mg/kg-d, based on testicular effects in male rats. TDCP-treated fabric samples were not available for testing by LSC. HBCD was used as a surrogate compound for estimating dermal and oral exposure. The percutaneous absorption rate of TDCP was measured *in vitro* (Hughes, 2000).

The HI for the basic case was 11 in adults and 12 in children (Table III-10). The HI for the non-aqueous cleaner case was 590 in adults, which is due to the increased migration of HBCD (used as a surrogate for estimating exposure) with methyl chloroform (see Table II-3a). The same is expected to be true for TDCP, which is also hydrophobic. In adults, inhalation of vapor phase TDCP contributed about 5 percent of the total risk (HI). In children, inhalation and mouthing contributed about 11 percent and 54 percent, respectively, of the total risk (HI). HI values for the various cases range from 11 to 590. This means that the aggregate exposure is predicted to exceed the ADI under all conditions of exposure.

TDCP is also carcinogenic in animals. For the basic case, the individual excess cancer risk was estimated to be 340 per million in adults and 9.8 per million in children (Table III-11). The risk estimate in children represents the contribution of exposures during the first two years of life. For the non-aqueous cleaner case, the cancer risk was as great as 18,000 per million. A substance or product is considered to present a hazard to consumers when the cancer risk exceeds one per million (CPSC, 1992). It should be noted that in adults, the estimated cancer risk from either dermal exposure (330 per million) or inhalation exposure (17 per million) alone exceeds the one-in-a-million level.

Cancer risks are considered to be cumulative. Because only children are exposed by mouthing, the risk from this route, 0.39 per million could be added to the lifetime risk for adults, 340 per million for the basic case. However, the contribution from oral exposure, in this case, is small in comparison to the total risk.

TDCP exposure is predicted to present a hazard to consumers for both cancer and non-cancer effects. However, the estimated dermal and oral exposures are based on a surrogate compound (HBCD). The estimated inhalation exposure is based entirely on mathematical models. Experimental measurements of TDCP migration and emissions of vapor phase TDCP are needed to verify these conclusions. The actual exposure to TDCP and the resulting risks could be lower or higher.

C. Effects of Age and Wear

LSC studied the effect of fabric age and wear on the liquid-mediated migration of FR chemicals (Bhooshan and Cobb, 2000). At least one fabric for each FR chemical was subjected to an accelerated aging process, then subjected to the same set of migration tests as the untreated fabrics. One fabric containing AT and DBDPO was also subjected to an accelerated mechanical wear process. Generally, the rate or extent of migration increased by an average of about 2-fold (range, 0.3 to 3.3), as compared to new fabric. Therefore, for the purpose of risk assessment, it was assumed that the migration rate (k_H) and extent of migration (M_L and M_U) are doubled in aged and worn fabric. Therefore, based on LSC's tests, the effect of age and wear on exposure is relatively small. There were no cases (see above) where these effects changed the overall conclusion of whether a given FR treatment would present a hazard to consumers.

Table III-1. Aggregate non-cancer risks (systemic effects) from exposure to antimony trioxide (AT).

Case, route, scenario	Adults		Children	
	ADD ^{a, b}	HI	ADD	HI
<u>Basic case</u> ^c	4.9x10 ⁻⁴	0.007	6.9x10 ⁻⁴	0.007
Dermal	4.9x10 ⁻⁴	0.007	4.7x10 ⁻⁴	0.007
Passive, normal use	2.8x10 ⁻⁴	4.0x10 ⁻³	2.7x10 ⁻⁴	3.9x10 ⁻³
Passive, spill	1.1x10 ⁻⁴	1.6x10 ⁻³	1.1x10 ⁻⁴	1.5x10 ⁻³
Passive, cleaning	1.0x10 ⁻⁴	1.5x10 ⁻³	9.7x10 ⁻⁵	1.4x10 ⁻³
Active, spill	1.8x10 ⁻⁸	2.6x10 ⁻⁷	3.3x10 ⁻⁸	4.8x10 ⁻⁷
Active, spot cleaning	1.7x10 ⁻⁸	2.4x10 ⁻⁷	0	0
Oral	0	0	2.2x10 ⁻⁴	1.0x10 ⁻⁴
Inhalation	4.5x10 ⁻⁷	1.9x10 ⁻⁷	1.1x10 ⁻⁶	4.6x10 ⁻⁷
Vapor phase	1.5x10 ⁻⁸	6.5x10 ⁻⁹	3.6x10 ⁻⁸	1.6x10 ⁻⁸
Particles	4.3x10 ⁻⁷	1.9x10 ⁻⁷	1.1x10 ⁻⁶	4.6x10 ⁻⁷
<u>Acidic spill</u>	2.3x10 ⁻²	0.33	2.2x10 ⁻²	0.31
<u>Non-aqueous cleaner</u> ^d	4.0x10 ⁻⁴	0.006	6.1x10 ⁻⁴	0.006
<u>Aged fabric</u>	7.0x10 ⁻⁴	0.010		

^a ADD, average daily dose, mg antimony per kg/d; HI, hazard index.

^b ADD estimates are based on an assumed value for percutaneous absorption.

^c Cases and scenarios are described in the Introduction. The basic case combines all scenarios, except full cleaning. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.

^d Migration into non-aqueous cleaner was below the detection limit. One-half the detection limit was used to calculate dermal exposure.

Table III-2. Cancer and non-cancer risks from inhalation exposure to antimony trioxide- (AT-) containing particles.

Population	Non-cancer effects			Cancer	
	ADE ^a	HI _i	LADE ^b	Risk per ^c million	
Adults	2.4x10 ⁻⁶	0.26		2.4x10 ⁻⁶	1.2
Children	2.4x10 ⁻⁶	0.26		6.3x10 ⁻⁸	0.03

^a ADE average daily exposure, mg antimony per m³ in air (mg/kg-d); HI_i, hazard index for inhalation exposure.

^b LADE, lifetime average daily exposure, mg antimony per m³ (mg/m³) in air.

^c Lifetime individual excess cancer risk from exposure to airborne AT-containing particles.

Table III-3. Aggregate non-cancer risks (liver effects) from exposure to decabromodiphenyl oxide (DBDPO).

Case, route, scenario	Adults		Children	
	ADD ^a	HI	ADD	HI
<u>Basic case</u> ^b	2.6x10 ⁻⁴	0.008	3.8x10 ⁻⁴	0.008
Dermal ^c	2.5x10 ⁻⁴	0.008	2.4x10 ⁻⁴	0.008
Passive, normal use	8.6x10 ⁻⁵	0.003	8.3x10 ⁻⁵	0.003
Passive, spill	3.4x10 ⁻⁵	0.001	3.3x10 ⁻⁵	0.001
Passive, cleaning	1.3x10 ⁻⁴	0.004	1.2x10 ⁻⁴	0.004
Active, spill	5.7x10 ⁻⁹	1.8x10 ⁻⁷	1.0x10 ⁻⁸	3.2x10 ⁻⁷
Active, spot cleaning	2.1x10 ⁻⁸	6.6x10 ⁻⁷	0	0
Oral	0	0	1.1x10 ⁻⁴	3.6x10 ⁻⁵
Inhalation	1.1x10 ⁻⁵	3.5x10 ⁻⁶	2.7x10 ⁻⁵	8.6x10 ⁻⁶
Vapor phase	9.9x10 ⁻⁶	3.1x10 ⁻⁶	2.4x10 ⁻⁵	7.6x10 ⁻⁶
Particles	1.3x10 ⁻⁶	4.2x10 ⁻⁷	3.3x10 ⁻⁶	1.0x10 ⁻⁶
<u>Acidic spill</u> ^c	2.6x10 ⁻⁴	0.008	3.8x10 ⁻⁴	0.008
<u>Non-aqueous cleaner</u>	2.2x10 ⁻³	0.07	2.2x10 ⁻³	0.07
<u>Aged fabric</u>	4.2x10 ⁻⁴	0.01		

^a ADD, average daily dose, mg/kg-d; HI, hazard index.

^b Cases and scenarios are described in the Introduction. The basic case combines all scenarios, except full cleaning. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.

^c Migration into saline, citric acid, and aqueous cleaner was below the detection limit. One-half the detection limit was used to calculate dermal exposure in these cases.

Table III-4. Aggregate non-cancer risks (liver effects) from exposure to hexabromocyclododecane (HBCD).

Case, route, scenario	Adults		Children	
	ADD ^a	HI	ADD	HI
<u>Basic case</u> ^b	1.3x10 ⁻³	0.007	3.9x10 ⁻³	0.020
Dermal ^c	1.3x10 ⁻³	0.007	1.3x10 ⁻³	0.006
Passive, normal use	8.5x10 ⁻⁴	0.0043	8.2x10 ⁻⁴	0.0041
Passive, spill	3.4x10 ⁻⁴	0.0017	3.3x10 ⁻⁴	0.0016
Passive, cleaning	1.5x10 ⁻⁴	7.4x10 ⁻⁴	1.4x10 ⁻⁴	7.1x10 ⁻⁴
Active, spill	5.6x10 ⁻⁸	2.8x10 ⁻⁷	1.0x10 ⁻⁷	5.0x10 ⁻⁷
Active, spot cleaning	2.4x10 ⁻⁸	1.2x10 ⁻⁷	0	0
Oral	0	0	2.4x10 ⁻³	0.012
Inhalation	1.5x10 ⁻⁶	7.4x10 ⁻⁶	2.7x10 ⁻⁴	1.3x10 ⁻³
Vapor phase	6.8x10 ⁻⁹	3.4x10 ⁻⁸	2.7x10 ⁻⁴	1.3x10 ⁻³
Particles	1.5x10 ⁻⁶	7.3x10 ⁻⁶	3.6x10 ⁻⁶	1.8x10 ⁻⁵
<u>Acidic spill</u> ^c	1.1x10 ⁻³	0.006	3.7x10 ⁻³	0.019
<u>Non-aqueous cleaner</u>	7.5x10 ⁻²	0.37	7.5x10 ⁻²	0.37
<u>Aged fabric</u>	1.8x10 ⁻³	0.009		

^a ADD, average daily dose, mg/kg-d; HI, hazard index.

^b Cases and scenarios are described in the Introduction. The basic case combines all scenarios, except full cleaning. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.

^c Migration into saline, citric acid, and aqueous cleaner was below the detection limit. One-half the detection limit was used to calculate dermal exposure in these cases.

Table III-5. Aggregate exposure to phosphonic acid, (3-{{[hydroxymethyl]amino}-3-oxopropyl)-, dimethyl ester (PA).^a

Case, route, scenario	Adults		Children	
	ADD ^{b, c, d}	HI ^e	ADD	HI
<u>Basic case</u> ^f	0.16	ND ^g	0.17	ND
Dermal	0.16	ND	0.15	ND
Passive, normal use	0.088		0.084	
Passive, spill	0.035		0.034	
Passive, cleaning	0.036		0.034	
Active, spill	5.8x10 ⁻⁶		1.0x10 ⁻⁵	
Active, spot cleaning	5.9x10 ⁻⁶		0	
Oral	0	0	0.016	ND
Inhalation	4.0x10 ⁻⁵	ND	9.9x10 ⁻⁵	ND
Vapor phase	3.9x10 ⁻⁵		9.6x10 ⁻⁵	
Particles	1.0x10 ⁻⁶		2.5x10 ⁻⁶	
<u>Acidic spill</u>	0.24	ND	0.24	ND
<u>Non-aqueous cleaner</u> ^h	0.12	ND	0.13	ND
<u>Aged fabric</u>	0.23	ND		

^a HI values were not calculated because PA does not satisfy the FHSA definition of "toxic" and there were insufficient data to calculate an ADI.

^b ADD, average daily dose, mg/kg-d; HI, hazard index..

^c ADD estimates are based on an assumed value for percutaneous absorption.

^d ADD estimates represent total organic phosphate, as mg PA/kg-day. Inorganic phosphate was subtracted from the total phosphate found in extracts of PA-treated fabrics before converting to mg PA (see Methodology).

^e There is no ADI for PA, because it does not satisfy the definition of "toxic" under the FHSA. Therefore, the HI cannot be calculated.

^f Cases and scenarios are described in the Introduction. The basic case combines all scenarios, except full cleaning. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.

^g ND, not done.

^h Migration into non-aqueous cleaner was below the detection limit. One-half the detection limit was used to calculate dermal exposure.

Table III-6. Aggregate exposure to reaction products of tetrakis (hydroxymethyl) phosphonium chloride (THPC).^a

Case, route	Adults		Children	
	ADD ^{b, c, d}	HI ^b	ADD	HI
<u>Basic case</u> ^e	0.12	ND ^f	0.12	ND
Dermal	0.12	ND	0.11	ND
Passive, normal use	0.068		0.065	
Passive, spill	0.027		0.026	
Passive, cleaning	0.024		0.023	
Active, spill	4.4x10 ⁻⁶		8.0x10 ⁻⁶	
Active, spot cleaning	3.9x10 ⁻⁶		0	
Oral	0	ND	5.5x10 ⁻³	ND
Inhalation	2.1x10 ⁻⁴	ND	5.2x10 ⁻⁴	ND
Vapor phase	2.1x10 ⁻⁴		5.2x10 ⁻⁴	
Particles	1.9x10 ⁻⁶		4.7x10 ⁻⁶	
<u>Acidic spill</u>	0.12	ND	0.12	ND
<u>Non-aqueous cleaner</u> ^g	0.095	ND	0.097	ND
<u>Aged fabric</u>	0.17	ND		

- ^a Fabrics are generally treated with a mixture of THPC and THPC-urea, which react to form a polymer. Migration was measured as the loss of total phosphorus from the fabric. The principal chemical species present in the extracts are unknown. However, THPC was not detected in the extracts (see text). The HI was not calculated, because the principal chemical species present in the extracts have not been identified
- ^b ADD, average daily dose, mg/kg-d; HI, hazard index.
- ^c ADD estimates are based on an assumed value for percutaneous absorption.
- ^d ADD estimates represent total organic phosphate, as mg THPC/kg-day. Inorganic phosphate was subtracted from the total phosphate found in extracts of THPC-treated fabrics before converting to mg THPC (see Methodology).
- ^e Cases and scenarios are described in the Introduction. The basic case combines all scenarios, except full cleaning. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.
- ^f ND, not done.
- ^g Migration into non-aqueous cleaner was below the detection limit. One-half the detection limit was used to calculate dermal exposure.

Table III-7. Aggregate non-cancer risks (systemic effects) from exposure to cyclic phosphonate esters (CPE) – washed fabric.^{a, b}

Case, route	Adults		Children	
	ADD ^c	HI ^c	ADD	HI
<u>Basic case</u> ^d	0.013	0.001	0.013	0.001
Dermal	0.013	0.001	0.012	0.001
Passive, normal use	6.9x10 ⁻³	6.9x10 ⁻⁴	6.7x10 ⁻³	6.7x10 ⁻⁴
Passive, spill	2.8x10 ⁻³	2.8x10 ⁻⁴	2.7x10 ⁻³	2.7x10 ⁻⁴
Passive, cleaning	2.8x10 ⁻³	2.8x10 ⁻⁴	2.7x10 ⁻³	2.7x10 ⁻⁴
Active, spill	4.6x10 ⁻⁶	4.6x10 ⁻⁷	8.2x10 ⁻⁷	8.2x10 ⁻⁸
Active, spot cleaning	4.6x10 ⁻⁷	4.6x10 ⁻⁸	0	0
Oral	0	0	4.6x10 ⁻⁴	4.6x10 ⁻⁵
Inhalation	2.2x10 ⁻⁴	2.2x10 ⁻⁵	5.3x10 ⁻⁴	5.3x10 ⁻⁵
Vapor phase	2.2x10 ⁻⁴	2.2x10 ⁻⁵	5.3x10 ⁻⁴	5.3x10 ⁻⁵
Particles	1.7x10 ⁻⁷	1.7x10 ⁻⁸	4.2x10 ⁻⁷	4.2x10 ⁻⁸
<u>Acidic spill</u>	ND ^e	ND	ND	ND
<u>Non-aqueous cleaner</u>	ND	ND	ND	ND
<u>Aged fabric</u>	0.018	0.002		

^a Exposure estimates assume that the treated fabric was washed to remove unbound CPE.

^b Treated fabric samples were not available for testing by CPSC. Exposure estimates are based on the loss of total phosphorus when treated apparel fabrics are laundered (Albright and Wilson, 1998c).

^c ADD, average daily dose, mg/kg-d; HI, hazard index.

^d Cases and scenarios are described in the Introduction. The basic case combines all scenarios, except full cleaning. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.

^e ND, not done.

Table III-8. Aggregate non-cancer risks (systemic effects) from exposure to cyclic phosphonate esters (CPE) – unwashed fabric.^{a, b}

Case, route	Adults		Children	
	ADD ^c	HI ^c	ADD	HI
<u>Basic case</u> ^d	0.25	0.025	0.25	0.025
Dermal	0.25	0.025	0.24	0.024
Passive, normal use	0.14	0.014	0.13	0.013
Passive, spill	0.056	0.006	0.053	0.005
Passive, cleaning	0.056	0.006	0.053	0.005
Active, spill	9.1x10 ⁻⁶	9.1x10 ⁻⁷	1.6x10 ⁻⁵	1.6x10 ⁻⁶
Active, spot cleaning	9.1x10 ⁻⁶	9.1x10 ⁻⁷	0	0
Oral	0	0	0.005	0.0005
Inhalation	4.4x10 ⁻⁴	4.4x10 ⁻⁵	1.1x10 ⁻³	1.1x10 ⁻⁴
Vapor phase	4.4x10 ⁻⁴	4.4x10 ⁻⁵	1.1x10 ⁻³	1.1x10 ⁻⁴
Particles	3.5x10 ⁻⁷	3.5x10 ⁻⁸	8.4x10 ⁻⁷	8.4x10 ⁻⁸
<u>Acidic spill</u>	ND ^e	ND	ND	ND
<u>Non-aqueous cleaner</u>	ND	ND	ND	ND
<u>Aged fabric</u>	0.36	0.036		

- ^a Exposure estimates assume that the treated fabric was NOT washed to remove unbound CPE.
- ^b Treated fabric samples were not available for testing by CPSC. Exposure estimates assume that unbound CPE is available for migration into aqueous solutions (Albright and Wilson, 1998a; Maibach, 1979; Ulsamer et al., 1980).
- ^c ADD, average daily dose, mg/kg-d; HI, hazard index. ND, not done.
- ^d Cases and scenarios are described in the Introduction. The basic case combines all scenarios, except full cleaning. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.
- ^e ND, not done.

Table III-9. Aggregate non-cancer risks (systemic effects) from exposure to 2-ethylhexyl diphenyl phosphate (EHDP).^a

Case, route	<u>Adults</u>		<u>Children</u>	
	ADD ^b	HI ^b	ADD	HI
<u>Basic case</u> ^c	0.066	0.066	0.069	0.069
Dermal	0.063	0.063	0.060	0.060
Passive, normal use	0.040	0.040	0.038	0.038
Passive, spill	0.016	0.016	0.015	0.015
Passive, cleaning	0.0069	0.007	0.0067	0.007
Active, spill	2.6x10 ⁻⁶	2.6x10 ⁻⁶	4.7x10 ⁻⁶	4.7x10 ⁻⁶
Active, spot cleaning	1.1x10 ⁻⁶	1.1x10 ⁻⁶	0	0
Oral	0	0	0.0024	0.002
Inhalation	2.6x10 ⁻³	2.6x10 ⁻³	6.4x10 ⁻³	6.4x10 ⁻³
Vapor phase	2.6x10 ⁻³	2.6x10 ⁻³	6.4x10 ⁻³	6.4x10 ⁻³
Particles	2.1x10 ⁻⁶	2.1x10 ⁻⁶	5.1x10 ⁻⁶	5.1x10 ⁻⁶
<u>Acidic spill</u>	0.056	0.056	0.060	0.060
<u>Non-aqueous cleaner</u>	3.5	3.5^d	3.4	3.4
<u>Aged fabric</u>	0.089	0.089		

^a Treated fabric samples were not available for testing by CPSC. HBCD was used as a surrogate compound to estimate dermal and oral exposure to EHDP (see text). Percutaneous absorption data were not available. *o*-Tricresyl phosphate was used as a surrogate compound to estimate percutaneous absorption (see text).

^b ADD, average daily dose, mg/kg-d; HI, hazard index.

^c Cases and scenarios are described in the Introduction. The basic case combines all scenarios, except full cleaning. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.

^d Values in bold indicate HI values greater than one.

Table III-10. Aggregate non-cancer risks (testicular effects) from exposure to tris(1,3-dichloropropyl-2) phosphate (TDCP).^a

Case, route, scenario	Adults		Children	
	ADD ^b	HI ^b	ADD	HI
<u>Basic case</u> ^c	0.055	11 ^d	0.059	12
Dermal	0.052	10	0.050	10
Passive, normal use	0.033	6.7	0.032	6.4
Passive, spill	0.013	2.7	0.013	2.6
Passive, cleaning	0.0058	1.2	0.0055	1.1
Active, spill	2.2x10 ⁻⁶	4.4x10 ⁻⁴	3.9x10 ⁻⁶	7.9x10 ⁻⁴
Active, spot cleaning	9.5x10 ⁻⁷	1.9x10 ⁻⁴	0	
Oral	0	0	0.0024	0.48
Inhalation	2.7x10 ⁻³	0.55	6.7x10 ⁻³	1.3
Vapor phase	2.7x10 ⁻³	0.55	6.7x10 ⁻³	1.3
Particles	2.2x10 ⁻⁶	4.3x10 ⁻⁴	5.3x10 ⁻⁶	0.001
<u>Acidic spill</u>	0.047	9.4	0.052	10
<u>Non-aqueous cleaner</u>	2.9	590	2.8	570
<u>Aged fabric</u>	0.074	15		

^a Treated fabric samples were not available for testing by CPSC. HBCD was used as a surrogate to estimate dermal and oral exposure to TDCP (see text).

^b ADD, average daily dose, mg/kg-d; HI, hazard index.

^c Cases and scenarios are described in the Introduction. The basic case combines all scenarios, except full cleaning. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.

^d Values in bold indicate HI values greater than one.

Table III-11. Aggregate cancer risks from exposure to tris(1,3-dichloropropyl-2) phosphate (TDCP).^a

Case, route, scenario	Adults		Children	
	LADD ^b	Risk per ^c million	LADD	Risk per million
<u>Basic case</u> ^d	0.055	340 ^e	1.6x10 ⁻³	9.8
Dermal	0.052	330	1.3x10 ⁻³	8.3
Oral	0	0	6.4x10 ⁻⁵	0.39
Inhalation	2.7x10 ⁻³	17	1.8x10 ⁻⁴	1.1
<u>Acidic spill</u>	0.047	290	1.4x10 ⁻³	8.5
<u>Non-aqueous cleaner</u>	2.9	18,000	0.075	470
<u>Aged fabric</u>	0.055	340		

^a Treated fabric samples were not available for testing by CPSC. HBCD was used as a surrogate for exposure to TDCP (see text).

^b LADD, lifetime average daily dose, mg/kg-d.

^c Lifetime individual excess cancer risk.

^d Cases and scenarios are described in the Introduction. The basic case combines all scenarios, except full cleaning. Uses saline to model spills and aqueous cleaner to model spot cleaning scenario. Oral exposure applies only to children. Direct exposure from spot cleaning applies only to adults.

^e Values in bold indicate lifetime individual excess cancer risks greater than one-in-a-million.

IV. Discussion

A. Assumptions and Limitations

The purpose of the present risk assessment is to predict consumer exposure to FR chemicals used in residential upholstered furniture and the risk of chronic health effects associated with that exposure. The hazard identification and dose response assessment were based primarily on animal studies. Only chronic health effects were considered. The exposure assessment was accomplished by evaluating a series of dermal, oral, and inhalation exposure scenarios. Input data for the exposure assessment included migration (leaching) data, *in vivo* or *in vitro* percutaneous absorption data, and assumptions regarding consumer behavior. Due to the complexity of the exposure assessment, only point estimates of exposure were calculated. However, a variety of exposure scenarios were included. All of the steps of the risk assessment were in accordance with the CPSC chronic hazard guidelines (CPSC, 1992). As with any risk assessment, there are many assumptions, limitations, and sources of uncertainty. These are discussed below, along with recommendations for reducing the uncertainty in future iterations of this risk assessment.

Risk assessment is an iterative process. The NRC used available information to estimate risk and, therefore, had very little data to estimate exposure. The present risk assessment incorporates new data on liquid-mediated migration and percutaneous absorption. These data were used to estimate dermal and oral exposure and internal dose. However, a number of significant data gaps remain. Migration data are only available for 5 FR treatments. There are still no data on inhalation exposure. Due to the lack of chemical-specific migration data for some FR chemicals, it was necessary to use data from chemicals with similar physico-chemical properties to estimate exposure. In some cases, assumptions regarding percutaneous absorption were made. In addition, data on carcinogenicity, teratogenicity, or neurotoxicity were not available for all chemicals. Therefore, due to the presence of these data gaps, additional data to fill these gaps may alter some of the conclusions of this report. In addition to providing a quantitative risk assessment, this risk assessment describes a general approach that manufacturers may use to assess the potential risks of FR chemicals in upholstered furniture.

In effect, two different exposure/risk assessments are presented here. The first includes FR treatments for which migration data were available—AT, DBDPO, HBCD, PA, and THPC. The second includes FR treatments for which migration data were not available—CPE, EHDP, and TDCP. Migration data were used to predict dermal and oral exposure. In most cases, dermal exposure was predicted to be the primary route of exposure; inhalation of particles was predicted to be significant for AT. When migration data were not available, surrogate compounds or other methods were used to estimate dermal and oral exposure. Furthermore, it should be noted that there were some chemicals for which percutaneous absorption data were not available, including AT, PA, and THPC. In these cases, percutaneous absorption rates were assumed based on data obtained with surrogate compounds with similar physico-chemical properties. No data relating to the inhalation of vapors or particles were available; all inhalation exposure estimates were derived from mathematical models.

1. Toxicity of FR Chemicals

The HS staff reviewed all the available toxicity data on 16 FR chemicals or chemical classes. In all, over 30 individual compounds were studied. For some FR's—such as AT, DBDPO, and some aromatic phosphates—the database is fairly extensive (Table I-1). In some cases—such as PA, tris(chloropropyl) phosphate, and some inorganic compounds—very few toxicity studies are available. When there was evidence of toxicity, the staff classified each chemical as either “known,” “probably,” or “possibly” toxic in humans, as defined in the CPSC chronic hazard guidelines (CPSC, 1992). Any chemical that is either “known” or “probably” toxic in humans is considered “toxic” under the FHSA. 16 CFR 1500.3 (c) (2)(ii). A number of chemicals did not satisfy the FHSA definition of “toxic.” This does not necessarily mean that they are “safe” or “non-toxic.” It may mean that insufficient testing has been done to determine whether the chemical is “toxic.” Indeed, virtually all chemicals are toxic at some dose or under some set of circumstances. The FHSA does not define “non-toxic” or “safe.” It only defines what is toxic or hazardous. Furthermore, the FHSA does not require any specific battery of tests for chronic hazards, nor does it provide for pre-market registration or approval of chemicals or consumer products.

FR chemicals that are “toxic” need to be evaluated in a quantitative risk assessment to determine whether they are “hazardous” under the FHSA. This report assesses the potential exposures and/or risks associated with a subset of the 16 chemicals or classes reviewed for toxicity, which are considered the most likely to be used in upholstered furniture.

2. Dermal Exposure and Bioavailability

Dermal exposure was modeled as a liquid-mediated process. For example, when sitting on upholstered furniture, a layer of perspiration is regarded as an external liquid phase that extracts FR chemical from the fabric and brings it into contact with the skin where it can be absorbed. The NRC Subcommittee employed a similar model (NRC, 2000, p. 38). However, the amount of external liquid phase, that is, perspiration, that is present during reasonably foreseeable use may not be sufficient to facilitate the transfer of FR chemical from fabric to skin. Therefore, the use of the liquid-mediated transfer model may overestimate exposure under typical use conditions.

On the other hand, one can imagine other processes that would allow for dermal exposure to occur. Ulsamer et al. (1978) applied FR-treated fabric to the skin of laboratory animals. Dermal absorption of the FR chemical, tris(2,3-dibromopropyl)phosphate (Tris), was observed, although the extent of absorption was greater when the fabric was saturated with saline or urine. Essentially similar results were obtained with pesticide-treated fabrics applied to human skin *in vitro* (Wester et al., 1996). Therefore, unmediated transfer of chemical from fabric to the skin may be possible. Thus, one might question whether liquid-mediated transfer is the appropriate method to model migration of hydrophobic compounds such as DBDPO or HBCD, which have low water solubility. In such cases, an unmediated transfer process might be more important. However, the presence of an external aqueous phase facilitated the transfer of both hydrophobic (Tris) (Ulsamer et al., 1978) and water soluble (glyphosate) chemicals (Wester et al., 1996).

Thus, it appears that liquid-mediated transfer is an appropriate model for both hydrophobic and hydrophilic compounds.

LSC developed a laboratory method to estimate liquid-mediated dermal exposure (Bhooshan and Cobb, 2000). In several cases, the amount of FR chemical in the liquid phase was below the analytical LOD. In these cases, one-half the LOD was used to estimate dermal exposure. Migration of DBDPO and HBCD was only detectable when methyl chloroform (non-aqueous cleaner) was the solvent. Migration of AT, PA, and THPC was non-detectable with methyl chloroform, but measurable with the other solvents. Hydrophobic compounds such as DBDPO and HBCD have very low solubility in water. For example, the water solubility of DBDPO is less than 0.1 parts per billion (as cited in Bittner, 1999a). In comparison, the LOD was 0.06 parts-per-million (Bhooshan and Cobb, 2000). Therefore, using one-half the LOD to estimate migration probably leads to overestimates of dermal DBDPO or HBCD exposure for scenarios involving aqueous solvents. Furthermore, the LSC method combines 5 consecutive extractions from the same fabric sample. This will also lead to conservative exposure estimates, that is, estimates that tend to overestimate exposure.

Others have measured dermal exposure and bioavailability from treated fabrics by an alternative method. Fabric treated with radiolabeled chemicals was applied directly to the skin of a laboratory animal and the rate of percutaneous absorption measured (e.g., Maibach, 1979; Ulsamer et al., 1978). The fabric was either kept dry or else saturated with an appropriate liquid. One investigator applied the treated fabric to human skin *in vitro* (Wester et al., 1996). Data are not available to compare this method with the methods in the present risk assessment.

Estimates of percutaneous absorption were generally based on studies in which the FR chemical was applied to the skin either as the pure compound or else in a volatile vehicle (Maibach, 1979; Hughes, 2000), as opposed to studies with aqueous solutions. This was mainly for practical reasons. Such data were already available for CPE (Maibach, 1979). DBDPO, HBCD, and TDCP are not water-soluble and, therefore, it would not have been practical to conduct studies using aqueous solutions. Furthermore, applying pure compound to the skin is recommended for hydrophobic compounds such as DBDPO and HBCD (Bronaugh and Stewart, 1984). However, this approach does not account for the effect of the vehicle on percutaneous absorption. Percutaneous absorption depends, in part, on the partitioning of the compound between the vehicle and the skin (Wester and Maibach, 1983). Thus, absorption of hydrophobic compounds is generally greater from aqueous vehicles. The dermal dose of hydrophobic compounds such as DBDPO, HBCD, and TDCP could be underestimated under scenarios involving aqueous media—saline, citric acid, or aqueous cleaner. As described above, an alternative method of estimating dermal exposure and bioavailability is to treat fabric with radiolabeled FR's and apply it directly to the skin of laboratory animals.

Percutaneous absorption of DBDPO, HBCD, and TDCP was measured *in vitro* (Hughes, 2000). A significant portion of the applied dose was bound within the skin and could not be removed by washing. Roughly equal amounts of TDCP were found in the skin and in the receptor fluid. Much greater amounts of DBDPO and HBCD were found in the skin than in the receptor fluid. This is expected for hydrophobic compounds tested *in vitro*, because they do not partition very well into the receptor fluid (Bronaugh and Stewart, 1984). It has been shown that

a particular hydrophobic compound diffuses from the stratum corneum into the dermis, where it may be absorbed (Yourick et al., 2000). Thus, for the purpose of risk assessment, it was assumed that the compound in the skin would eventually be absorbed *in vivo* (Bronaugh and Collier, 1991). It is possible that DBDPO could be absorbed very slowly from the stratum corneum, because it has an exceptionally high molecular weight (960) and it appeared to bind tightly to the skin (Hughes, 2000). Therefore, the assumption that any FR chemical found in the stratum corneum would be absorbed could overestimate absorption of the hydrophobic compounds, DBDPO, HBCD, and TDCP (Wester and Maibach, 1983). This source of uncertainty could be resolved by performing *in vivo* studies.

In both the *in vitro* and *in vivo* studies of percutaneous absorption, the absorption rate was reported as the percentage of the applied dose absorbed. The percentage absorption depends on the applied dose, decreasing as the applied dose (mg/cm^2) increases. In the case of TDCP, the applied dose in the *in vitro* experiments ranged from 0.014 to 0.14 $\mu\text{g}/\text{cm}^2$ (Hughes, 2000). The estimated TDCP dose on the skin was on the order of 1 $\mu\text{g}/\text{cm}^2$ for most scenarios and about 1,000 $\mu\text{g}/\text{cm}^2$ for scenarios involving non-aqueous cleaner (data not shown).^{*} Thus, the estimated applied dose in the risk assessment is greater than the applied dose in the percutaneous absorption studies and, therefore, the dermal TDCP dose may be overestimated.

With HBCD, the applied dose in the *in vitro* experiments ranged from 0.2 to 2 $\mu\text{g}/\text{cm}^2$ (Hughes, 2000). The estimated dermal dose in the risk assessment, was roughly 1 $\mu\text{g}/\text{cm}^2$ for most scenarios, and about 1,000 $\mu\text{g}/\text{cm}^2$ for scenarios involving non-aqueous cleaner (data not shown). Thus, the applied doses in the risk assessment and in the percutaneous absorption studies are roughly equal, except for scenarios involving non-aqueous cleaner. The dermal dose of HBCD could be overestimated in the scenarios involving non-aqueous cleaner.

Absorption of DBDPO was highly dependent on the applied dose: about 2 percent absorption was observed at 60 nanomoles (90 $\mu\text{g}/\text{cm}^2$), 3 percent at 30 nmol (45 $\mu\text{g}/\text{cm}^2$), and 20 percent at 6 nmol (9 $\mu\text{g}/\text{cm}^2$) (Hughes, 2000). Therefore, different percutaneous absorption rates were derived, depending on the amount of DBDPO in contact with the skin. A value of 20 percent absorption was applied when less than or equal to 9 $\mu\text{g}/\text{cm}^2$ of DBDPO was estimated to be in contact with the skin. Three percent absorption was applied when greater than 9 $\mu\text{g}/\text{cm}^2$ DBDPO was estimated to be in contact with the skin. This approach tends to minimize errors relating to the effect of applied dose on percentage absorption.

The percutaneous absorption of radiolabeled CPE was studied in rhesus monkeys (Maibach, 1979). CPE excretion in urine (as radiolabel) was measured over a period of 8 days. This methodology could underestimate percutaneous absorption of CPE if a significant portion of the CPE were retained in the tissues, which were not examined.

The percutaneous absorption rates used in this risk assessment were obtained from animal studies or from *in vitro* studies with animal skin. Human skin is generally less permeable than rodent skin, by up to 10-fold (Wester and Maibach, 1983). No attempt was made to adjust for these species to species differences. Therefore, the use of animal data may lead to overestimates

^{*} The dose of FR chemical on the skin is an intermediate calculation, which was not reported in the Results.

of dermal absorption and the resulting risk. However, three compounds (DBDPO, HBCD, and TDCP) were tested *in vitro* with full thickness mouse skin (Hughes, 2000). The use of full thickness mouse skin tends to minimize the difference between mouse and human skin, because a compound need only penetrate to the upper dermis to be absorbed into the circulation (Bronaugh and Stewart, 1984). Furthermore, neonatal skin is more permeable than adult human skin (Wester and Maibach, 1983). Therefore, it seems reasonable to use the animal data, even if it tends to overestimate bioavailability and risk.

Three passive exposure scenarios were used to estimate dermal exposure: normal use, spilled beverages or foods, and cleaning. The normal use scenario estimates the exposure that may occur when bare skin contacts FR-treated upholstered furniture fabric. The spill and cleaning scenarios estimate the additional exposure that may occur when spilled beverages or cleaning agents extract FR chemical which is deposited on the fabric surface when the liquid dries. FR chemical deposited on the fabric probably does not remain indefinitely. Rather, it may be worn away, removed by cleaning, or absorbed by consumers contacting the furniture. However, the rates at which these processes occur are unknown. Therefore, in the absence of data, the most conservative assumption was used, that is, that FR chemical deposited by spills or cleaning is always present.

In combining these passive scenarios, it was assumed that the entire surface of any piece of furniture has been exposed both to spilled liquids and cleaner for the entire lifetime of the product. It is unlikely that the entire surface is exposed to spilled liquids. It is expected that some consumers would clean their furniture themselves or have it cleaned, but this is probably infrequent. Professional cleaners recommend that consumers have their furniture cleaned every 18 to 24 months (as cited in Smith, 2000). However, the actual frequency of full cleaning is probably less, and not all consumers would have their own furniture cleaned. Therefore, combining the exposures resulting from these three scenarios may be regarded as very conservative, that is, leading to the overestimation of dermal exposure.

To be sure, a more realistic scenario would be to apportion the exposure duration, frequency of exposure, and/or skin surface area among the different passive scenarios. However, no data are available that would provide a basis for doing so. For example, any assumptions regarding how much time one sits on furniture that was previously exposed to spills, or what portion of skin contacts the spill area, would be arbitrary. Therefore, in the absence of data, the most conservative assumption was used, that is, that the passive scenarios occur simultaneously every time a consumer contacts upholstered furniture.

3. Oral Exposure

Oral exposure was estimated by means of the “head-over-heels” method that was developed to estimate exposure to phthalate esters in children’s products, such as pacifiers and teethers (EU, 1998). A limited number of validation studies have been performed for PVC products (CPSC, 1998; EU, 1998). To our knowledge, this is the first instance in which this method was applied to fabric samples. This method has not been validated for fabrics. If more appropriate methods are developed in the future, they should be applied. At this time, however,

the head-over-heels is the used method used by CPSC for assessing oral exposure from children's mouthing activity.

4. Inhalation Exposure

Inhalation exposure was estimated for both particle-bound and vapor phase FR chemicals. Assessments for both inhalation scenarios were based entirely on mathematical models. The model used to predict the emission rates (source strengths) for particulate emissions is essentially similar to the model used by the NRC Subcommittee (NRC, 2000). The model assumes that a certain portion of the fabric is eroded over the life of the furniture, resulting in the release of airborne particles. A certain portion of the airborne particles is assumed to be of respirable size. The particles are assumed to be released at a constant rate and they are expected to be uniform with respect to FR content. In the absence of any data, this model provides a sense of whether exposure to particles could plausibly lead to unacceptable exposure levels.

Exposure to particle-bound FR's was a significant source of risk only for AT. This portion of the risk assessment could be improved by obtaining data on airborne AT levels in homes with treated furniture or by conducting appropriate laboratory experiments. Possibly, fabric samples could be placed in a chamber and subjected to a mechanical wear process. The concentration of FR-containing particles of various size ranges in air would then be measured, and the emission rate calculated. The CPSC staff subjected a back-coated fabric to an accelerated mechanical wear process (Tao et al., 2000). After 200,000 cycles, the AT content of the fabric was not significantly reduced.

A mathematical model described in the NRC report was used to predict source strengths for vapor phase FR chemicals. The model predicts emissions from the basic principles of diffusion (Fick's law) and the physico-chemical properties of the FR's, but does not account for sink effects, which would tend to reduce exposure. We are not aware of any data validating the model for this application. Measurements of emissions from treated fabrics, either in a chamber or in residences, would be needed to confirm the source strengths and FR levels predicted by this model. The model does not account for the loss of vapor phase FR due to absorption by other furnishings (e.g., carpet, wall-coverings, draperies) or chemical decay. These assumptions tend to overestimate exposure to vapor phase FR chemical.

The NRC model was used for both immersion-treated and back-coated fabrics. With immersion-treated fabrics, at least a portion of the FR chemical is on the fabric surface. In the case of back-coated fabrics, however, migration of the FR chemical through the polymeric back coating would be an additional step that might tend to slow the release of FR chemical into the air. The same situation would also apply to the portion of CPE that is fixed within the fabric fibers (Albright and Wilson, 1998c; Maibach, 1979). However, attempts to model back-coated fabrics with the EPA polymer migration model AMEM (Schwope et al., 1989) resulted in estimated exposures that were only slightly less than (within a factor of 2 or 3) those predicted by the NRC model (data not shown). AMEM is a more complicated model requiring a greater number of additional input parameters, many of which are poorly defined. Therefore, rather than introducing additional sources of uncertainty, the NRC model was used for all fabrics. It was generally assumed that all of the FR chemical load on a given fabric was exposed to air. The

only exceptions were PA and THPC, which are covalently bound. Only the portion of PA or THPC that could be extracted was considered to be exposed to air. For back-coated fabrics, it was assumed that 100 percent of the back-coating was exposed to air. The assumptions that 100 percent of the FR chemical is exposed to air and that the back-coating and fabric do not slow the migration of FR chemical tend to overestimate exposure to vapor phase FR chemical. These assumptions all tend to overestimate exposure to vapor phase chemicals.

The source strengths for particulate and vapor phase emissions were entered into a one-zone mass balance model (NRC, 1981). NRC used more conservative assumptions for the amount of FR-treated furniture present, room volume, and air infiltration rate (see below). The present risk assessment applied more typical values for these particular parameters. This resulted in significantly lower exposure estimates for particle-bound AT, for example, as compared to the NRC study. The present risk assessment assumed a volume of 340 m³, which is equivalent to a typical, 140 m² (1500 ft²) ranch home, and a typical whole-house air infiltration of 0.5 h⁻¹ (Koontz and Rector, 1993). It was also assumed that consumers spend an average of 16 hours per day in their homes (EPA, 1997b, p. 15-17). This approach predicts the overall average of particle-bound or vapor phase FR chemical in the home. In reality, these concentrations will be greater in rooms with upholstered furniture, such as the living room, than in rooms with little or no upholstered furniture, such as the kitchen or bedroom. If the interior doors are open or if a forced-air heating-air conditioning system is operating, then the difference in concentrations between rooms may be as little as 20 percent. Even if the concentration gradients are significant, consumers will most likely divide their time among different rooms. The use of a multi-zone model would require assumptions for interzone air exchange rates and the number of hours consumers spend in different rooms, which would introduce additional sources of uncertainty into the exposure and risk estimates. Therefore, for the purpose of this risk assessment, the staff assumed that an average indoor concentration would be adequate for estimating exposure and risk.

5. Risk Assessment

Certain exposure scenarios—direct exposure to spilled liquids or the use of upholstery cleaner for spot cleaning—occur intermittently. Because the ADI's are generally based on chronic or subchronic studies, it is unlikely that a one-time exposure exceeding the ADI would have the same effects as daily exposure. An averaging period of one year has been suggested for subchronic effects (Thompson, 1999). Therefore, an averaging time of one year was assumed for non-cancer endpoints. Exposures were averaged over a lifetime for estimating cancer risk. However, the use of a shorter averaging time may be appropriate for certain endpoints, such as developmental effects or acute toxicity. However, the active dermal exposures were generally quite low (see Results). None of the ADI's or RfD's in this assessment was based on developmental effects. Acute toxicity was not considered.

A route-to-route correction was applied for dermal exposures (Equations 2.13 and 2.18). The ADI values were generally based on oral studies in animals. Some compounds, such as AT and DBDPO, are poorly absorbed by the oral route (Hatlid, 1999a; Bittner, 1999a). Therefore, the absorbed dose is significantly less than the applied dose. ADI's are based on the applied dose. Adjustments for bioavailability are generally not necessary when performing an oral risk

assessment, because both the exposure estimate and the ADI are based on applied doses. Thus, the relative absorption in animals and humans, or among exposure routes, is generally more relevant to assessing risk than an absolute measure of absorption (CPSC, 1992, p. 46650). Whether 100 percent or 10 percent of the substance is absorbed is not critical, provided that the extent of absorption is about equal in animals and humans. Many risk assessments, including the CPSC risk assessment on FR chemicals, assume that absorption is equal in humans and animals.

Unlike oral exposure estimates, dermal exposure estimates generally include a bioavailability adjustment, that is, percutaneous absorption. Thus, the dermal dose estimate is an absorbed dose. Consequently, the ADI, which is an applied dose, must be adjusted. When the oral bioavailability is low, this adjustment increases the estimated dose (ADD or LADD) and its associated risk (HI or cancer risk). When the oral bioavailability is 100 percent, there is no effect on the estimate of dose or risk.

Oral bioavailability estimates were only available for AT and DBDPO. The route-to-route adjustment makes the estimated risk (HI) from dermal exposure more conservative for AT and DBDPO, as compared to the NRC report (see below). For the other FR chemicals, the default assumption (100 percent bioavailability) was applied, which is the same as making no adjustment.

No adjustments were made for inhalation exposure, because inhalation bioavailability estimates were generally not available. In estimating the contribution of inhalation exposure to total exposure, the default assumption of 100 percent bioavailability was applied.

B. Comparison with the NRC Report

The National Research Council (NRC) recently released a report on the “Toxicological Risks of Selected Flame-Retardant Chemicals” (NRC, 2000). Although the overall conclusions of the NRC report are generally similar to the present risk assessment, there are some differences with respect to scope and methodology that, in some cases, affect the conclusions. Some of the specific differences are discussed below. In comparing risk assessment methodologies, it is important to consider the overall risk assessment process and not to simply focus on the differences in individual steps.

The NRC report covered all 16 FR chemicals or chemical classes proposed by the FRCA. Chemical classes, such as aromatic phosphates and organic phosphonates, were represented by the most toxic member of the class. The present risk assessment includes 8 chemicals that are a subset of the 16 chemicals/classes proposed by FRCA and that the CPSC staff considers the most likely to be used in upholstered furniture. Four of these 8 chemicals are already in use in the U.K. and, therefore, are almost certain to be used in the U.S. if the draft standard is adopted. The CPSC staff believes that the remaining 4 are also very likely to be used, based on information provided by their manufacturers.

The greatest difference between the NRC and CPSC risk assessments is that the NRC Subcommittee had virtually no migration data from which to estimate exposure. They were forced to rely more heavily on mathematical models and assumptions. The CPSC staff recently

conducted migration studies on upholstery fabric samples representing five different FR chemical treatments. These data were used to estimate dermal and oral exposure (Bhooshan and Cobb, 2000). The CPSC staff also obtained percutaneous absorption data for three FR chemicals (Hughes, 2000). The availability of migration and percutaneous absorption data improve the risk assessment for FR chemicals, because dermal exposure is the principal exposure route for most of these chemicals. These data were not yet available when the NRC report was completed.

Exposure estimates in both risk assessments required estimates for a number of other parameters. In estimating inhalation exposures, NRC assumed a relatively large area of FR-treated fabric (30 m^2) in a small room (30 m^3), with a relatively low air infiltration rate (0.25 h^{-1}) (NRC, 2000, p. 42). The present risk assessment assumed 10 m^2 of fabric (equivalent to a sofa, love seat, and chair), in a typical 1500 ft^2 ranch style home, with a median air infiltration rate of 0.5 h^{-1} (see above). However, NRC assumed 8 hours/day of exposure, while CPSC assumed 16 hours/day. The net result of these differences is that CPSC calculated lower estimates of exposure and risk for inhalation exposures. For example, NRC estimated a greater HI (1.2) for the inhalation of AT-containing particles than CPSC (0.26). NRC also estimated a greater lifetime cancer risk for AT (1.7×10^{-4}) than CPSC (1.2×10^{-6}). NRC used an RfC that was 20-fold greater than the CPSC ADI (see below), although the cancer potencies were roughly similar. As with any risk assessment, any one approach is not necessarily superior to the alternatives. In this particular case, the lack of empirical data on the release of AT particles leads to greater uncertainty than any assumptions regarding room size, air infiltration, or exposure duration.

For estimating dermal exposure, NRC estimated that 2200 cm^2 of skin was in contact with furniture fabric for 6 hours/day, whereas CPSC estimated that 2500 cm^2 of skin would be in contact with furniture for 4 hours/day. However, CPSC applied additional dermal exposure scenarios—passive and active exposure to spills and cleaners. In estimating oral exposure, NRC assumed that a child would mouth 50 cm^2 of fabric for up to 1 hour per day. In contrast, the CPSC staff estimates that a child would mouth 11 cm^2 of fabric, the same surface area used for teething rings and toys, for no greater than 15 minutes per day, based on observational studies of children (Smith, 2000; Smith and Kiss, 1998).

The ADI values and cancer unit risks derived by the CPSC staff frequently differed somewhat from the RfD's and cancer potency estimates derived in the NRC report. These differences are generally due to differences between the CPSC chronic hazard guidelines and the EPA guidelines, which the NRC employed. The differences in risk assessment methodology among federal agencies are generally minor and have been discussed elsewhere (Babich, 1998; CPSC, 1992; Rhomberg, 1997). The major differences are highlighted below. To calculate the cancer unit risk, CPSC uses the maximum likelihood estimate of cancer risk, provided that the dose response is linear at low doses, whereas EPA prefers the upper bound. Under the CPSC guidelines, when there is more than one significantly responding tumor site (e.g., TDCP), the unit risk for each site is calculated separately and then added. The EPA guidelines allow the calculation of cancer potency based on total tumors.

The NRC and CPSC used roughly similar cancer potency factors for AT, $0.71 \text{ (mg/kg-d)}^{-1}$ and $0.51 \text{ (mg/kg-d)}^{-1}$, respectively. However, the NRC derived a significantly greater potency factor for TDCP. The NRC Subcommittee based their potency estimate ($0.06 \text{ (mg/kg-d)}^{-1}$) on

the incidence of benign interstitial cell tumors of the testes, but added that the relevance of these tumors to humans is debatable (NRC, 2000, p. 378). The CPSC potency estimate (6.2×10^{-3}) was based on the combined risk of liver carcinoma and tumors of the renal cortex in both males and females.

Both CPSC and EPA use an uncertainty factor approach to derive ADI or RfD values, respectively, for non-cancer endpoints. CPSC and EPA both use uncertainty factors to account for differences in sensitivity between individuals and differences between animals and humans. An additional uncertainty factor is applied if a NOAEL has not been established and a LOAEL must be used. The two agencies differ in that CPSC does not use additional uncertainty factors for extrapolating from subchronic to chronic exposure or for inadequacy of the data base. This is because the FHSA only defines what is toxic; it does not define non-toxic or safe. Therefore, ADI's are simply based on the available toxicity data. CPSC and NRC generally used the same studies to derive ADI and RfD or RfC values.

CPSC derived an ADI for inhalation exposure to AT particles of $0.009 \times 10^{-3} \mu\text{g}/\text{m}^3$, based on a LOAEL of $9 \times 10^{-3} \text{mg}/\text{m}^3$ and a net uncertainty factor of 1,000. In comparison, the EPA reference concentration (RfC) of $0.2 \mu\text{g}/\text{m}^3$, which the NRC Subcommittee employed, is 20-fold greater. In deriving the RfC, EPA used a benchmark dose instead of the LOAEL and used a mathematical model to adjust for animal-to-human differences (reviewed in Hatlelid, 1999a). While the CPSC ADI is more conservative than the RfC that the NRC used, the NRC used relatively conservative assumptions for estimating exposure. The net effect of these methodological differences is that the NRC estimated a 5-fold greater HI for exposure to AT particles (1.2) than CPSC (0.26).

There were a few other differences in methodology, as compared to the NRC report. The present risk assessment includes four additional scenarios that may contribute to dermal exposure—active (direct) exposure from spills and spot cleaning, and passive (indirect) exposure from spills and cleaning. These scenarios are not included in the NRC assessment. CPSC also applied a route-to-route correction for dermal exposure, which tends to increase the estimated risk from dermal exposure (discussed above).

The two risk assessments used different approaches to estimate percutaneous absorption. NRC used permeability coefficients (K_p values) (cm/h) obtained with aqueous solutions of the FR chemical to calculate the rate of percutaneous absorption. When appropriate K_p estimates were not available, which was frequently the case, a correlation was used to estimate K_p values from the octanol-water partition coefficient and molecular weight of the FR chemical (EPA, 1992; Potts and Guy, 1992). In the present risk assessment, dermal absorption studies with pure FR compounds applied to the skin were used to estimate the rate of percutaneous absorption as a transfer coefficient (k_t), which is the percent of applied chemical absorbed per hour (Scheuplein and Ross, 1974). When data were not available for a specific compound, compounds with similar octanol-water partition coefficients and molecular weights were used as surrogates. This approach was used partly because data with pure compounds were available for some FR's and partly because some FR's (e.g., DBDPO and HBCD) are not water soluble (see above). CPSC also applied a route-to-route correction for dermal exposure, which tends to increase the estimated risk from dermal exposure (discussed above).

The CPSC staff agrees with the NRC conclusion that additional data on inhalation exposure to AT-containing particles is needed to determine whether this route of exposure may be hazardous. Both risk assessments also agree that DBDPO, HBCD, and PA are not likely to present a hazard. Both risk assessments also agree that TDCP might be a hazard, but that exposure data are needed. However, the CPSC staff concluded that additional data are needed to assess THPC, while NRC concluded that THPC would present a minimal risk. NRC's conclusion is based, in part, on the assumption that exposure to THPC would be negligible because THPC polymerizes (NRC, 2000, pp. 433-434). However, migration studies performed by CPSC, which were unavailable to the NRC Subcommittee, indicate that significant migration of THPC by-products may occur. The NRC did not estimate risks for CPE and EHDP.

C. Conclusions

1. Specific FR Treatments

Migration data were available for 5 chemicals—AT, DBDPO, HBCD, PA, and THPC. Based on this risk assessment, the CPSC staff concludes that CPE, DBDPO, HBCD, and PA would not present a hazard to consumers, as defined under the FHSA (Table IV-1). The estimated HI values for DBDPO and HBCD are all less than unity under all exposure conditions. HBCD and PA did not satisfy the FHSA definition of toxic. However, the database for PA is limited. The staff also concludes that EHDP probably would not present a hazard, unless the fabric is exposed to dry cleaning solvents. Additional data are needed for AT to determine whether exposure to AT particles in air may be hazardous. TDCP is possibly hazardous; dermal, oral, and inhalation exposure data are needed. Additional data are needed to determine whether THPC may present a hazard.

AT. Exposure to AT by the dermal and oral routes was at acceptable levels. HI values for systemic effects ranged from 0.006 for the non-aqueous cleaner case to 0.33 for the acidic spill case, using very conservative exposure assumptions (Table III-1). Except for the acidic spill case, the HI is quite small. It should be noted that, while migration data were obtained for AT, percutaneous absorption data were not available. The staff concludes that AT does not present a hazard by the dermal or oral route. However, additional data are needed to assess the potential risks from inhalation of AT-containing particles. This conclusion is essentially similar to the conclusion reached by the NRC Subcommittee (NRC, 2000).

The HI for inhalation of particles was 0.26, which is acceptable (Table III-2). However, the inhalation exposures were estimated from mathematical models. Given the uncertainty in this approach, the true HI could be considerably higher or lower. The estimated lifetime excess cancer risk for inhalation of particles (1.2 per million) was about one-in-a-million for adults. Cancer risks greater than one-in-a-million are considered to be hazardous under the FHSA (CPSC, 1992, p. 46656). Again, the true risk could be higher or lower. Therefore, data on the release of airborne particles containing AT would be needed to develop a more reliable estimate of exposure. If the predicted levels are accurate, then AT would present a minimal hazard to consumers. The level of airborne, particle-phase AT predicted by the model—about 4 ng/m³—is roughly comparable to AT levels in ambient air in Washington, DC—1 to 3 ng/m³ (ATSDR,