6

Safety, health and environmental aspects of flame retardants

M. M. HIRSCHLER, GBH International, USA

DOI: 10.1533/9780857098931.1.108

Abstract: This chapter investigates the following issues: (a) key types of flame retardants; (b) toxic effects of smoke on fire victims and effects of flame retardants on smoke toxicity and fire safety; (c) acute effects of individual components of smoke on fire victims; (d) individual potential acute (short-term) and chronic (long-term) toxicity of major individual flame retardants; (e) effects of flame retardants on the environment and (f) life cycle analysis on the generation of toxic combustion products and environmental damage when comparing flame-retarded products with products that are not flame retarded and have high flammability. The work demonstrates that the use of flame retardants presents an important benefit to society and the environment via the improvement of fire safety. Undoubtedly not all flame retardants ever developed or about to be developed are safe from all points of view, but the use of appropriate scientific knowledge and the regulatory environment can effectively ensure that if any specific material is shown to be unsafe it is kept away from consumers. In summary, this survey shows that the appropriate use of flame retardants, as a class, effectively provides improved fire safety via lowering the probability of ignition, the heat released and the amounts of smoke, combustion products and dangerous environmental toxicants. In consequence the use of flame retardants increases the available time for escape from a fire.

Key words: fire, fire and the environment, fire retardants, fire safety, flame retardants, life cycle analysis, safety, smoke toxicity, toxicity.

6.1 Introduction

The use of flame retardants is one of the most common strategies used to improve the fire performance of textile (and other) materials.[1,2] One of the first known uses of flame retardants was by the Montgolfier brothers in France in 1783 to coat their 'lighter-than-air balloons' with 'alum' to reduce fire hazard.[3] Even earlier than that, the first patent for a flame retardant was granted in Britain to Obadiah (or Jonathan) Wyld in 1735 (British Patent

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

551[4]), for a mixture of alum, vitriol and borax, to be used for textile and paper pulp. Even in ancient times, the Egyptians discovered that a solution of alum made wood burn more slowly and, during the siege of Pyraeus (Greece, ca. 90–80 BC) alum was known to have been used to protect the storming towers against fire.

In modern times it is now known that seven elements are critical for imparting improved fire properties to both natural polymers (e.g., cotton or wool) and synthetic polymers, whether the end product is a textile, an elastomer/rubber or a solid plastic. Those seven heteroelements are: chlorine, bromine, aluminum, boron, phosphorus, nitrogen and antimony. Other elements are also useful, mainly as complements, particularly fluorine (rarely used in flame retardants) and sulfur. With the increased sophistication and improved technology, it is now clear that the flame-retardant effect does not come just from the key heteroelements used but also from the structure of the flame retardant, since different materials containing identical fractions of the same heteroelement are very likely to have vastly different effectiveness. Moreover, additivity and synergy often exists between individual heteroelements, whether they are contained in the same flame retardant or in different ones.

Flame retardants can be classified as additive or as reactive. Additive flame retardants are added to a material or a product without bonding or reacting with the product, whereas reactive flame retardants are chemically reacted into the raw materials used to make the final material or product to be commercialized. They can also be used as co-additives (or synergists) to improve on the effectiveness of the primary flame retardant or the inherent polymeric structure or as fillers (to decrease the amount of combustible material, or fuel, available for burning).

Several issues need to be addressed:

- the key types of flame retardants, particularly for textiles (see Section 6.2);
- the overall toxic effects of smoke on fire victims and the immediate effects of flame retardants on the toxicity of the smoke generated by a material containing such additives (i.e., the smoke toxicity of flame retardants and of the associated smoke; see Section 6.3);
- the acute effects of individual components of smoke on fire victims (see Section 6.4);
- the individual potential acute and chronic toxicity of flame retardants on their own, such as during manufacture, during disposal or as a result of their migration into the environment (i.e., the inherent short-term and long-term health effects of the flame retardants; see Section 6.5);
- the effects of flame retardants on the environment, such as any potential activity as carcinogens, mutagens or as toxicants affecting wildlife (i.e.,

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

the potential environmental effects of the flame retardants; see Sections 6.5 and 6.6);
- a life cycle analysis for the generation of toxic combustion products and of environmental damage when comparing flame-retarded products with non-FR products (see Section 6.7).

6.2 Relevant key types of flame retardants

Surveys of the industries making and/or using flame retardants indicate that there are 16 key flame retardants or flame retardant classes for textiles.[5] Those flame retardants are as follows (not necessarily in any specific order, but organized as a function of their chemical composition):

- Halogenated materials:
  - decabromodiphenyl oxide,
  - hexabromocyclododecane and
  - chlorinated paraffins.
- Phosphorus-containing materials:
  - tetrakis (hydroxymethyl) phosphonium salts (chloride salt),
  - tris (monochloropropyl) phosphates,
  - tris (1,3-dichloropropyl-2) phosphate,
  - ammonium polyphosphates,
  - organic phosphonates (e.g., dimethyl hydrogen phosphite),
  - aromatic phosphate plasticizers (e.g., tricresyl phosphate) and
  - phosphonic acid (3-{[hydroxymethyl]amino}-3-oxopropyl)-dimethyl ester.
- Inorganic materials:
  - alumina trihydrate,
  - magnesium hydroxide,
  - zinc borate,
  - antimony trioxide,
  - antimony pentoxide and sodium antimonates and
  - calcium and zinc molybdates.

These 16 chemicals are, of course, not the only ones used and new ones are being developed all the time. However, during a new private survey conducted in early 2012, this group of chemicals still appeared to represent all key textile flame retardants. It also represents the seven heteroelements discussed above. The toxic effects of these flame retardants will be discussed individually in Section 6.5.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

6.3 Toxicity of smoke from combustion products

Flame retardants are used to improve fire performance and thus decrease the amount of combustion products released in a fire. It has long been demonstrated that the smoke toxicity of combustion products is a key factor in fire hazard, together with heat release, flame spread and ignitability. In fact, the cause of death for some two thirds of all fire victims tends to be listed as 'inhalation of combustion products'. However, it has also been shown that it is relatively rare for multiple fire fatalities to occur in fires that have remained small. The reason for these seemingly contradictory facts is that the inherent toxic potency of smoke resulting from burning most combustible materials is very similar and tends to fall within a narrow range. Moreover, it has also been shown that the key fire property that governs the loss of human tenability in a fire atmosphere is the heat release rate of the burning materials, which can vary by orders of magnitude for common combustibles (while, as just stated, the toxic potency of most combustibles is very similar). Thus, toxic hazard is often a direct function of heat release rate rather than of toxic potency, provided the comparison involves materials that have significant differences in fire performance.

It has been shown repeatedly that the most important fire property is the rate of heat release (e.g., Reference 6), since it is the one that both governs the intensity of a fire, and the survivability in a fire scenario. Table 6.1 illustrates this fact, by determining survival time (through computer modeling) in a standard room, with a common chair. When the chair is made of a material with half the time to ignition, the survival time does not change. Similarly if the toxic potency of the chair materials is doubled, it has very little effect on survival time, while doubling the rate of heat release immediately decreases survival time, by a factor greater than 3. This is a very important concept, because it starts putting into perspective the importance (or lack of it) of smoke toxic potency data in terms of fire hazard assessment, or simply of fire safety.

Table 6.1 Effects of different fire properties on survival time

Product	Survival time
Primary chair	>10 min
Double ignitability	>10 min
Double toxic potency	>10 min
Double heat release rate	3 min

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.2 Effects of flame retardant additives on smoke toxicity

Products	Peak temperature (°C)	Smoke toxicity (kg CO)	Tenability time in burn room (s)	Tenability time in target room (s)	CO yield (kg/kg)	Pk HRR (MW)
Non-FR	>600	21	110	200	0.22	1.59
Non-FR	>600	17	112	215	0.18	1.54
Non-FR	>600	16	116	226	0.14	1.79
FR 1B	185	2.6	NA	NA	0.22	0.22
FR	273	5.5	1939	NA	0.23	0.37
FR	285	6.1	2288	NA	0.23	0.35
FR	334	5.6	1140	1013	0.23	0.45

Peak temperature: maximum temperature in burn room;
Smoke toxicity: level of toxicity, calculated in equivalent mass of carbon monoxide;
Tenability time in room: time to reach untenable conditions in burn room or target room (via smoke toxicity or by having reached flashover, i.e. temperatures over 600°C);
CO yield: mass of carbon monoxide (CO) formed per mass of fuel burnt;
Pk HRR: maximum heat release rate in room; non-FR: non fire-retarded;
Non-FR: non-flame retarded products;
FR: flame retarded products; and
FR 1 B: flame retarded products tested without an auxiliary burner.

One of the most important studies that illustrated this concept is the NIST work on the comparison between flame retarded and non-flame retarded versions of the same set of products.[7] During this work, NIST obtained five different categories of products and tested them in full-scale room fires. In one series, all five products were flame retarded, whereas in the other series the same base polymers were used, but without flame retardant agents added. The products tested were: upholstered furniture mock-ups, business machine housings, television housings, electric cables, and electronic circuit board laminates. The most interesting results were those from the full scale fire tests, which also helped to identify the importance of heat release rate as the most important physical variable in these tests which is a predictor of the fire hazard. Table 6.2 shows the most important results of the burns. During the first burn carried out with the flame retarded products under the same conditions as the non-flame retarded products (a single 50 kW burner) very little combustion took place. Thus, all successive tests with the flame retarded products also included an additional 120 kW burner, which allowed all the products to burn.

The results indicated that the use of the flame retarded products was associated with an average four-fold decrease in heat release rate, a three-fold decrease in smoke toxicity in the room, and a ten-fold increase in tenability

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.3 Comparison of heat release rate data for FR and non-FR materials

Material	Heat flux (kW/m²)	Non-FR Pk HRR (kW/m²)	FR Pk HRR (kW/m²)	NFR/FR ratio —	Test method —
ABS (+ FR1)	20	614	224	2.7	Cone
ABS (+ FR1)	40	944	402	2.3	Cone
ABS (+ FR1)	70	1311	409	3.2	Cone
ABS (+ FR2)	20	614	224	2.7	Cone
ABS (+ FR2)	40	944	291	3.2	Cone
ABS (+ FR2)	70	1311	419	3.1	Cone
EVA (Cross-linked)	30	463	110	4.2	Cone
EVA (Thermoplastic)	30	574	83	6.9	Cone
HDPE	30	1803	114	15.8	Cone
HDPE # 2	50	1167	476	2.5	Cone
LDPE	20	913	88	10.3	Cone
LDPE	40	1408	192	7.3	Cone
LDPE	70	2735	268	10.2	Cone
Polypropylene	30	1555	174	8.9	Cone
PVC rigid	20	102	25	4	Cone
PVC rigid	40	183	84	2.2	Cone
PVC rigid	70	190	93	2.1	Cone
PVC rigid # 2	30	98	42	2.3	Cone
PVC rigid # 3	30	118	56	2.1	Cone
PVC wire and cable	20	116	9	12.8	Cone
PVC wire and cable	40	167	64	2.6	Cone
PVC wire and cable	70	232	100	2.3	Cone
PVC wire and cable # 2	20	116	72	1.6	Cone
PVC wire and cable # 2	40	167	92	1.8	Cone
PVC wire and cable # 2	70	232	134	1.7	Cone
Particle board	25	151	66	2.3	OSU
Particle board B (+FR 1)	25	160	70	2.3	Cone
Particle board B (+FR 1)	50	227	141	1.6	Cone
Particle board B (+FR 2)	50	227	52	4.4	Cone
Plywood	25	114	43	2.7	Cone
Plywood	50	150	75	2	Cone
Polyester	30	186	95	2	Cone
Polystyrene	20	723	277	2.6	Cone
Polystyrene	40	1101	334	3.3	Cone
Polystyrene	70	1555	445	3.5	Cone

Cone: ASTM E1354 (Cone calorimeter) and
OSU: ASTM E906 (Ohio State University heat release rate calorimeter).

time (even though the ignition source used was over three times as intense). This is a clear indication that considerable improvements in toxicity are obtained by decreasing the heat release rate of the materials/products considered, almost irrespective of the actual toxic potency of the materials/products involved. Thus, toxic hazard is a direct function of heat release

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

rate, *provided there has been proper flame retardancy*. In contrast, in at least one other study no improvement in toxic hazard was found following the use of materials (upholstered furniture) containing flame retardants.[8] An analysis of the materials involved indicates that the so-called flame retarded materials simply contained very low levels of flame retardants, insufficient to make a substantial difference to the heat release rate of the final product. Thus, no real improvement in fire performance occurred, resulting in no improvement in toxic hazard.

Table 6.3 is a compilation of a few of the data available wherein the same basic material was tested, using the same technique before and after the addition of flame retardants. The data indicate that, in fact, considerable improvements in rate of heat release are commonplace, including increases of over 10-fold. The range of materials presented in the table is also very broad, including thermoplastics, cross-linked materials, thermosets, and cellulosics. This is crucial data, because it indicates that the effect of fire retardants can, indeed, lead to great improvements in smoke toxicity. In order to understand its further implications with respect to smoke toxicity, it is essential to investigate toxic potency ranges for different materials.

The data above shows that fire hazard is primarily a function of the rate of heat release of the materials or products involved and the importance of heat release rate vastly exceeds that of smoke toxic potency. Heat release rate can be decreased by factors of 10 or more by using flame retardants. Therefore, adequate flame retardance considerably decreases fire hazard, by decreasing heat release rates and improving time to escape.

Note that all the analysis in this section has dealt with the immediate aftermath of a fire, namely, the acute effects of smoke, which is always toxic. The key conclusion to be drawn from this analysis is that, in order to minimize smoke toxicity, it is essential to do everything possible to minimize fires and, failing that, to strive to lower heat release rate.

6.4 Effects of individual combustion products on fire victims

There have been a number of studies investigating the effects of combustion products on fire victims and it has been found that the key toxicant that affects fire victims is carbon monoxide (CO), with smaller contributions from other toxicants. The other major individual toxicants are hydrogen cyanide (HCN), acrolein, hydrogen chloride (HCl), and hydrogen fluoride (HF). CO is emitted by all materials when they burn, almost irrespective of their chemical composition. The other individual toxicants are emitted as a function of the chemical composition of the burning material.

Two studies were made in the United States involving more than 5000 fatalities and covering two aspects: (a) a period between 1938 and 1979 in a

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

localized area (Cleveland, Ohio) and (b) a broad, countrywide study in the early 1990s.[9,10]

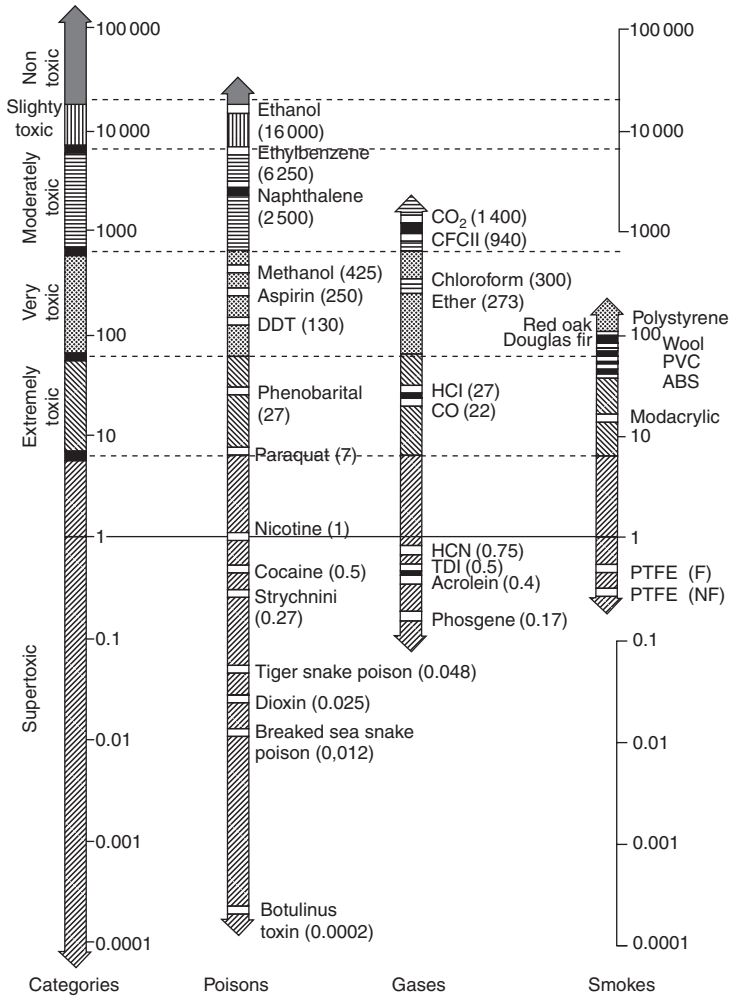
The studies demonstrated that there is an excellent correlation between fire fatalities and levels of CO absorbed in the blood as carboxyhemoglobin (COHb) and that the distribution of COHb concentrations was identical (when comparing populations of the same type, following a statistical multi variant analysis) between fire and non-fire deaths (e.g., defective space heater). The studies also showed that whenever high levels of hydrogen cyanide were found in blood, high levels of COHb were also usually found, indicating that hydrogen cyanide is of minor consequence in the overall study of fire fatalities. However, it is also known that some cases exist, especially when dealing with confined environments (such as small rooms or bedrooms), where hydrogen cyanide is directly associated with the ensuing fire fatalities.

Finally, the studies showed that fatalities can be linked to COHb levels as low as 20% and that it is likely that any COHb level above 30–40% is lethal. The work also reconfirmed that any hydrogen chloride found during fire fatality studies cannot be assigned to fire gases (or smoke) as it cannot be distinguished from the stomach acid naturally present. The overall conclusion of this work, the most extensive ever conducted, is clear: fire fatalities are overwhelmingly associated with the CO generated when fires become big, and other causes of fire deaths are of minor importance. Similar conclusions were obtained earlier by other authors, with smaller databases.[11–13]

Looking at toxic potency of smoke data, Fig. 6.1 indicates that the toxic potency of the smoke of virtually all individual polymers is within such a narrow band (in toxicological terms) as to be almost indistinguishable.[14] In particular, the single polymeric material most widely studied for smoke toxicity has been poly(vinyl chloride) (PVC), and its most common inorganic combustion product, hydrogen chloride (HCl). Extrapolation of most of the conclusions reached for PVC and HCl is probable for many halogenated polymers (such as polymers containing chlorinated and/or brominated flame retardants) and to hydrogen bromide (HBr), a combustion product typically generated from brominated flame retardants and from the polymers containing them.

The smoke toxicity of PVC has been studied extensively and found to be quantitatively similar to that of most other polymers.[15] As discussed earlier, the work on PVC also reviewed HCl toxicity studies, including some of exposure of animals and people, in the late nineteenth century and early twentieth century, to hydrogen chloride alone or exposure to smoke containing hydrogen chloride. The critical issue is that what was studied was the behavior, and whether incapacitation or lethality occurred, rather than looking for hydrogen chloride itself in autopsies (which cannot be detected). The exposures of rats and baboons made in the 1980s at Southwest Research

WPHIRSCHLER
© Woodhead Publishing Limited, 2013



6.1 Scales of toxicity. The figure illustrates the small range of toxic potencies of smoke by showing the degrees of toxic potency of poisons, toxic combustion products and of the smoke emitted by most common polymeric materials in logarithmic scales.

Institute showed that both rats and baboons were not incapacitated at huge concentrations of hydrogen chloride (and in fact sometimes they died a long time after exposure, but they were able to perform the escape functions that they were taught, to escape from exposure).[16,17] This means that the toxic potency of hydrogen chloride is such that exposed primates are not incapacitated at concentrations that may eventually kill them.

Two investigations were conducted in the United States in the late 1970s, wherein fire fighters were sent into buildings equipped with gas

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

monitors.[14,18,19] In the fires investigated, CO was almost inevitably present, with the maximum concentration found being 7450 ppm (approximately 150% of the 30 min LC_{50} for CO). The three other gases most commonly found in fires in those studies were (in descending order of number of times the gas was found and shown in relation to the 30 min LC_{50}):

1. acrolein (with a maximum concentration of 100 ppm, approx. 50–80% LC_{50});
2. hydrogen chloride (with a maximum concentration of 280 ppm, approx. 8% LC_{50}) and
3. hydrogen cyanide (with a maximum concentration of 10 ppm, approx. 6% LC_{50}).

There have been extensive studies indicating that CO yields of 0.2 g/g for post-flashover fires are widespread (Table 6.4). Moreover, it has been shown that CO yields in small scale tests are very low.[10,11,20] Evidence exists that CO yields can be higher than 0.2 g/g if significant amounts of oxygen-containing material (typically when cellulosic materials, such as wood) pyrolyzes in the upper layer.[21] Such an increased yield does not affect the conservative nature of the approach outlined here, since the importance of the CO produced by the room is higher. There is no evidence to date of cases with CO yields substantially lower than 0.2 g/g in the original fire compartment. This does not invalidate the procedure, since the assumption that the CO present is equivalent to a yield of 0.2 g/g is more conservative than if the yield is actually higher (and post-flashover fires are even more toxic). No information exists to suggest that post-flashover yields are lower, on average, than 0.2 g/g.

In consequence, the following is now widely accepted:[7,9,10,26–29]

- Fire fatalities tend to occur in fires that become very large. In fact, US statistics indicate that such fires account for over six times more fatalities than all other fires.
- CO concentrations in the atmospheres of flashover fires (the fires most likely to produce fatalities), are determined by geometric variables and oxygen availability, but are virtually unaffected by chemical composition of fuels.
- All small scale fire tests underpredict CO yields. They cannot therefore be used to predict toxic fire hazard for ventilation-controlled flashover fires, unless CO yields are calculated by analogy with full scale fire test results. Such tests do not underpredict yields of other toxicants, such as hydrogen chloride or hydrogen cyanide.
- CO yields in full scale flashover fires are approximately 0.2 g/g, which translates to a toxicity of 25 mg/L.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.4 Yields of CO in literature

Material	Yield	Reference	Organization
Plywood walls, wood fiberboard ceiling	0.35	Budnick, 1978[22]	HUD
FR plywood walls, wood fiberboard ceiling	0.42	Budnick, 1978[22]	HUD
Plywood walls, no ceiling	0.10	Budnick, 1978[22]	HUD
Upholstered chair, bed FR plywood walls	0.36	Budnick *et al.*, 1978[23]	HUD
Plywood walls, bed	0.31	Budnick *et al.*, 1978[23]	HUD
Plywood walls, wood cribs, cellulosic ceiling tile	0.29	Levine and Nelson, 1990[24]	NIST
Non-FR chair, TV cabinets, cables, etc.	0.22	Babrauskas *et al.*, 1988[7]	FRCA
FR chair, TV cabinets, cables, etc.	0.23	Babrauskas et al., 1988[7]	FRCA
PMMA walls	0.28	NIST unpublished	NIST
Wood cribs	0.15	Gottuk *et al.*, 1992[25]	VPI
Flexible PU foam	0.25	Gottuk *et al.*, 1992[25]	VPI
PMMA	0.30	Gottuk et al., 1992[25]	VPI
Hexane	0.23	Gottuk *et al.*, 1992[25]	VPI
Propane	0.23	Beyler, 1986[20]	Harvard
Propene	0.20	Beyler, 1986[25]	Harvard
Hexanes	0.20	Beyler, 1986[20]	Harvard
Toluene	0.11	Beyler, 1986[20]	Harvard
Methanol	0.24	Beyler, 1986[20]	Harvard
Ethanol	0.22	Beyler, 1986[20]	Harvard
Isopropanol	0.17	Beyler, 1986[20]	Harvard
Acetone	0.30	Beyler, 1986[20]	Harvard
Polyethylene	0.18	Beyler, 1986[20]	Harvard
PMMA	0.19	Beyler, 1986[20]	Harvard
Pine	0.14	Beyler, 1986[20]	Harvard
Average	0.236	Cases: Total #: 24	Total #: 5

- Toxic potency values from the NIST radiant small scale test (ASTM E1678 or NFPA 269;[30,31] with rats as the animal model, but used only for confirmatory purposes) is well validated with regard to toxicity in full scale fires. However, such validation cannot be done to a better approximation than a factor of 3.
- The consequence of this is that any toxic potency (LC_{50}) higher than 8 mg/L (i.e., any toxicity lower than 8 mg/L) will be subsumed within the toxicity of the atmosphere, and is of no consequence. Thus, values 8 or greater should be converted to 8 mg/L for reporting purposes.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

- In order to correct small scale test data, the CO yield that should have been obtained can be calculated by making a CO correction.
- The rationale for the corrections to the CO yields is the comprehensive study of fire (and non-fire) fatalities (approximately 5000) associated with CO.[9,10] The study found that:
- The toxicity of fire atmospheres is determined almost solely by the amount of CO, since victims of poisoning by pure CO die at virtually identical levels, once other factors have been considered. There is no universal lethal CO threshold level (which was previously thought to be 50% carboxyhemoglobin, COHb). This depends on the age and physical condition of the victim. Any blood COHb value > 20% can produce lethality on its own.
- The populations of victims of fire and of non-fire CO exposures are inherently very different: fire victims are predominantly both much older and much younger than the victims of non-fire CO exposures, and they suffer much more often from some pre-existing disease. Thus fire victims are more sensitive to CO than those in non-fire CO exposures.
- A comparison of fire fatalities before and after the plastics era indicates that the use of man-made materials to make household goods has made no difference to fire atmosphere toxicity.

Thus, instead of exposing animals, the following equation is a predictor of smoke toxicity, where smoke is lethal if FED (fractional effective dose) is near unity:

$$\mathrm{FED} = \frac{m[\mathrm{CO}]}{[\mathrm{CO_2}]-b} + \frac{[\mathrm{HCN}]}{\mathrm{LC_{50}HCN}} + \frac{[\mathrm{HCl}]}{\mathrm{LC_{50}HCl}} + \frac{[\mathrm{HBr}]}{\mathrm{LC_{50}HBr}} + \frac{21-[\mathrm{O_2}]}{21-\mathrm{LC_{50}O_2}}$$

The constants used in the equation above (dealing with a 30 min exposure) are shown below, and the first 2 (m and b) vary with the concentration of CO_2. The other terms in the equation are the concentrations of CO, hydrogen cyanide, hydrogen chloride, hydrogen bromide (all in ppm) and oxygen (%):

m	–18	Units: 1/ppm (if CO_2 is ≤ 5%)
b	122 000	Units: ppm (if CO_2 is ≤ 5%)
m	23	Units: 1/ppm (if CO_2 is > 5%)
b	–38 600	Units: ppm (if CO_2 is > 5%)
LC_{50} HCN	200	Units: ppm
LC_{50} HCl	3700	Units: ppm
LC_{50} HBr	3000	Units: ppm
LC_{50} O_2	5.4	Units: %

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

If CO_2 is 3–5% (normal range to be expected in a large fire) the LC_{50} of CO will be in the range between 5100 and 4000 ppm. If the data are to be used for flashover fires, the CO concentration must be corrected (as discussed above), using the equation below, that gives the added CO concentration:

$$\Delta[\mathrm{CO}] = 0.2 - \frac{[\mathrm{CO}] \times 0.200}{m_{100}} \times \frac{28}{24.5 \times 10^6}$$

Note that all the analysis in this section has also dealt with the immediate aftermath of a fire, namely, the acute effects of smoke, which is always toxic.

Following this analysis, it should be clear that it is rarely important to measure individual toxic gases for hazard assessment purposes, even for flame retardant chemicals. The measurement of toxic gases is mainly helpful in material development and in understanding the fire performance of any material or product, especially before bringing it to market. There are some very rare cases where smoke toxicity can have an important contribution in fires. That is the case when people die in very small fires (often smoldering) that destroy minimal amounts of material. Fortunately, statistics indicate that such fire cases are extremely rare and a more complete investigation may be needed for each individual case.

There is another reason to measure gases emitted during burning, and it is not related to fire hazard or to fire safety, but to the ability to sell materials. Many specifications and requirements demand that materials be certified as lacking in a certain component, typically halogen elements. Thus, measurement of combustion products is often associated with 'passing' certain specifications for material composition.

Fire safety and fire hazard are associated primarily with the control of heat release and with ensuring that a fire remains small. On the other hand, smoke toxicity is generally no more than a small component of fire safety, except in some special circumstances. Smoke toxicity is primarily identified with CO, while several other gases are additional contributors: acrolein, hydrogen cyanide and hydrogen chloride primarily. The key conclusion to be drawn from this analysis is that the chemical composition of the burning materials themselves has a very low impact on the smoke toxicity of a fire atmosphere.

6.5 Potential toxicity issues associated with individual flame retardants

This section will deal with the inherent toxic effects of individual flame retardants or flame retardant classes, both on their immediate effects (acute) and on their long-term effects (chronic), primarily focusing on the

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

latter. The toxicity analyses presented in this section summarize the investigation of the US National Research Council (NRC) (Committee on Toxicology, Subcommittee on Flame-Retardant Chemicals) in 2000.[5] In its 1999 fiscal year appropriations report for the US Consumer Product Safety Commission (CPSC), the US Congress requested an independent study by the National Academy of Science NRC concerning health risks posed by exposure to FR chemicals likely to be used in residential upholstered furniture to meet a flammability standard that CPSC was considering (and still is considering as of 2012). CPSC, with the help of the industry, identified the chemicals (or chemical classes) listed in Section 6.2 that became the focus of the NRC study.

The NRC assigned the project to the Committee on Toxicology of the Board on Environmental Studies and Toxicology and it convened the Subcommittee on Flame-Retardant Chemicals, which prepared a report. Subcommittee members were chosen for their recognized expertise in toxicology, epidemiology, pharmacology, chemistry, exposure assessment, risk assessment, and biostatistics. The subcommittee was charged with reviewing the toxicological and exposure data on the above 16 flame retardant chemicals (FRs) to assess potential health risks to consumers and the general population resulting from exposure to these chemicals in residential furniture. The subcommittee was also asked to identify data gaps and make recommendations for future research. The subcommittee was not required, or able with the available data and resources, to evaluate adverse effects from occupational exposures, or the potential ecological effects that might result from the disposal of household furniture. The subcommittee was also not charged to compare FRs for efficacy, or to evaluate the cost, technology, exposure-standard achievability, or the benefits of using FRs on upholstered furniture to reduce fire risk. The subcommittee did not consider the toxicity of combustion products of FR materials from fires involving treated textiles or other materials but the issue of combustion product toxicity and flame retardants is being addressed in the present chapter.

For some of these materials additional studies were performed after the NRC work and most of that will not be covered in this chapter. Most of the additional work, much of which was done for European Union risk analyses by chemical companies and not directly published, has filled in some of the gaps identified by the NRC study. In the case of tris (monochloropropyl) phosphates (TCPP, the most widely known one being tris (1-chloro-2-isopropyl) phosphate) Section 6.5.5 will present some of the added information, as an example of the continuing work that was performed. This work did not invalidate the conclusions of the NRC report but presented additional information covering many of the gaps identified by NRC.

Another study that looked at toxic and environmental impacts of flame retardants was conducted by the US Environmental Protection Agency

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

(EPA) in a study for the Flame Retardants in Printed Circuit Boards Partnership.[32,33] This report focused heavily on the environmental aspects of flame retardants but studied some different flame retardants and will form the basis of the analysis in Section 6.6.

The NRC subcommittee started by making assessments to determine whether causal relationships existed between the dose of each FR chemical and each adverse health effect. To identify adverse effects associated with an FR chemical, the subcommittee reviewed human (epidemiological studies, clinical observations, and case reports) and laboratory animal data on neurotoxicity, immunotoxicity, reproductive and developmental toxicity, organ toxicity, dermal and pulmonary toxicity, carcinogenicity, and other local and systemic effects. It also reviewed *in vitro* data to determine the potential for genotoxicity as well as other toxic effects and to understand the mechanisms of toxic action. Toxicokinetic studies were reviewed to understand the absorption, distribution, metabolism, and excretion of the FR chemicals.

For some types of toxic effects, notably most cancers, the subcommittee conservatively assumed that no threshold for a dose-response relationship exists or that, if one does exist, it is very low and cannot be reliably identified. Therefore, the subcommittee used a different risk-estimation procedure for carcinogens than that for non-carcinogens. In the risk-estimation procedure for carcinogens, the relationship between the incidence of cancer and the dose of a chemical reported in an epidemiological study or an experimental animal study was extrapolated linearly to doses much lower than those at which humans might be exposed. This procedure overestimated conservatively the excess lifetime risk of cancer resulting from lifetime exposure to a chemical at a particular dose rate. This procedure does not provide a 'safe' dose with an estimated risk of zero (except at zero dose), although at sufficiently low doses, the estimated risk becomes very low and is regarded to have no public-health significance. In fact, even though actual risk cannot be determined, the subcommittee concluded that the actual risk will not exceed the upper bound. The actual risk is also highly likely to be lower than the upper bound, and it might be zero.

In the absence of adequate toxicity data to derive dermal safe doses, the subcommittee characterized potential risks from dermal exposures by using oral safe doses to calculate the hazard indices. In addition, sufficient data to derive safe oral inhalation concentrations were available for only two FRs. The values for inhalation data for other FRs were extrapolated from oral data by using typical body weights and breathing rates.

The subcommittee assumed that human exposure to FR-treated fabrics in homes can occur potentially via skin contact, ingestion (specifically for infants or children who might suck or chew on fabric), inhalation of particles generated during abrasion of surface fibers, and inhalation of vapors off-gassing from treated fabric. In estimating exposures to FRs, the

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

subcommittee evaluated three exposure scenarios that involved different exposure routes: dermal, oral, and inhalation. For dermal exposure, the scenario was that of an adult sitting on FR-treated fabric of a couch for a substantial fraction (25%) of the time, with potential exposure over 25% of the upper torso area; clothing was conservatively assumed to provide no barrier to exposure. For ingestion, the scenario was of an infant or a child repeatedly sucking on FR-treated fabric of a chair or couch. For inhalation, the scenario was of a person spending time in a closed (but ventilated) room containing FR-treated upholstered furniture fabrics that shed FRs as small (respirable) particles, or from which FR chemicals evaporate. In all of those exposure scenarios, the subcommittee intentionally overestimated exposures by using extremely conservative assumptions.

In the final phase of the risk-assessment process, the subcommittee integrated data and analyses from hazard identification, dose-response assessment, and exposure assessment to determine the likelihood that individuals might experience adverse effects from the FR chemical under anticipated conditions of exposure. In order to characterize the health risk from exposure to a non-carcinogenic chemical, a hazard index was calculated to judge whether a particular exposure would be likely to present a non-cancer toxicological risk.

In the absence of adequate human carcinogenicity data for any FR chemical, the subcommittee took an approach for estimating cancer risks from exposure to carcinogenic FRs which involved the extrapolation of observations of cancer at relatively high doses in laboratory animals to much lower doses anticipated for humans in residential settings. The upper limit on the cancer potency factor extrapolated from animal experiments was multiplied by the estimated lifetime average dose rate to estimate an upper limit on lifetime cancer risk. The subcommittee did not recommend an acceptable cancer risk level for carcinogenic FR chemicals, because that is a regulatory policy question, not a scientific matter. Some regulatory agencies consider as acceptable excess lifetime cancer risks ranging from 1×10^{-4} to 1×10^{-6} of exposed people. The toxicological risk assessments made by the subcommittee for each of the 16 FR chemicals are presented in Sections 6.5.1 through 6.5.16 and in Tables 6.5–6.20. Key concepts used are:

- RfD: reference dose, which is an estimate of lifetime daily dose that is believed to have a reasonable certainty of no harm.
- RfC: reference concentration, similar to RfD, and calculated or mathematically scaled from the RfDs.

6.5.1 Decabromobiphenyl oxide (DBDE)

Non-cancer dermal exposure: The assessment of non-cancer risk for the dermal route of exposure is based on a scenario that assumes an adult spends a

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

quarter of the time sitting on furniture upholstery back-coated with DBDE and also assumes that a quarter of the upper torso is in contact with the upholstery and clothing presents no barrier. Exposure to other chemicals present in the back-coating was not included in this assessment. As a first estimate of exposure, it was assumed that the skin and clothing of the person sitting on the couch, and the fabric of the couch, would present no barrier to movement of DBDE. In addition, it was assumed that there would be sufficient water present (e.g., from sweat) to allow dissolution of the DBDE in the water, and transfer to the skin and into the body of the sitting individual. The only limiting factor on the transfer rate using these assumptions results from the limited dissolution rate from the fabric, since all the DBDE that dissolves is assumed to be absorbed immediately by the seated individual. For this calculation, the subcommittee estimated an upholstery application rate for DBDE of 5 mg/m^2. The results indicated that DBDE does not pose a non-cancer risk by the dermal absorption route when used as an upholstery fabric flame retardant. An alternative iteration of the exposure assessment was also performed, using the same exposure assumptions, except that the assumption of immediate absorption of all the DBDE that dissolves was modified. Instead, an estimate of the rate at which DBDE can penetrate the skin was determined, assuming that DBDE dissolves up to its solubility limit in water and this rate of penetration was then factored into the exposure assessment. Again, the hazard index calculated demonstrated that DBDE, used as an upholstery fabric flame retardant, is not likely to pose a non-cancer risk for dermal exposure.

Non-cancer inhalation exposure to particles: Inhalation exposure estimates for DBDE were calculated using an exposure scenario that assumes a person spends a quarter of his or her life in a room with low air-change rates (0.25 per hour) and with a relatively large amount of fabric upholstery (30 m^2 in a 30 m^3 room), with the DBDE treatment gradually being worn away over 25% of its surface to 50% of its initial quantity over the 15 year lifetime of the fabric. A small fraction, 1% of the worn-off DBDE is released into the indoor air as small particles that may be inhaled. Factoring in the fraction of a day a person spends in the room containing upholstery (0.25), the subcommittee chose to estimate the inhalation RfC from the oral RfD. The subcommittee, however, recognized that this is not an ideal approach and also recognizes that the estimated RfC might be different than the actual reference concentration (if inhalation data were available), but the subcommittee believes that its extrapolation of the oral RfD to the inhalation RfC is highly conservative (i.e., the actual hazard may be lower) as it assumes that all of the inhaled compound is deposited in the respiratory tract and is completely absorbed into the blood. The NRC Committee on Toxicology had used this approach before when inhalation exposure data were insufficient to derive inhalation exposure levels. Based on this excessively conservative approach,

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

this indicates that under the worst case exposure assumptions, DBDE, used as an upholstered fabric flame retardant, does not pose any non-cancer risk via inhalation of DBDE in the particulate phase.

Non-cancer inhalation exposure to vapors: In addition to the possibility of release of DBDE in particles from upholstery fabrics, the subcommittee considered the possibility of its release by evaporation. The approach uses the same scenario as that previously described for exposure to DBDE in the particulate phase. The results indicate that, under the worst case scenario, exposure to DBDE used as a flame retardant for upholstery is not likely to pose a non-cancer risk via the inhalation route when exposure occurs in the vapor phase.

Non-cancer oral exposure: The assessment of the non-cancer risk for the oral exposure route is based on a scenario assuming that a child is exposed to DBDE through sucking on 50 cm^2 of fabric back-coated with DBDE daily for 2 years, at 1 h per day. Using these values it was concluded that DBDE used as an upholstery fabric flame retardant does not pose any non-cancer risk via the oral route.

Cancer dermal exposure: The human cancer risk for dermal exposure to DBDE was calculated by multiplying the lifetime oral cancer potency factor for DBDE by the lifetime average dermal dose rate. Using the lifetime average dermal dose rate obtained in the alternative dermal exposure for the non-cancer dermal exposure and multiplying this by the cancer potency estimate a lifetime risk estimate of 1.20×10^{-12} is obtained. This estimate is small enough that the cancer risk through dermal contact with DBDE used as an upholstery fabric flame retardant can be considered negligible.

Cancer inhalation exposure to particles: For DBDE, no inhalation cancer unit risk is available. However, an inhalation cancer unit risk of 2.57×10^{-7} per $\mu g/m^3$ was estimated from the oral carcinogenic potency. The average room-air concentration and average exposure concentration to DBDE were obtained as described in the non-cancer section. From this estimate, DBDE, used as an upholstery fabric flame retardant, poses a negligible cancer risk via inhalation in the particulate phase.

Cancer inhalation exposure to vapors: The equilibrium concentration of vapor-phase DBDE in room-air was estimated as described in the non-cancer section. The long-term time-average vapor exposure concentration was estimated from the equilibrium vapor concentration in room-air and this estimate indicates that DBDE, used as a flame retardant, poses a negligible cancer risk via inhalation in the vapor phase.

Cancer oral exposure: The lifetime average dose rate estimate for DBDE ingestion multiplied by the cancer unit risk gives a lifetime cancer risk estimate small enough that the cancer risk via the oral route can be dismissed as negligible when DBDE is used as an upholstery fabric flame retardant.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.5 Toxicity data for decabromobiphenyl oxide (DBDE) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: liver toxicity
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	4
	Cancer potency factor (oral)	9.0×10^{-4} per mg/kg-d
	Cancer potency factor (inhalation)	2.6×10^{-7} per μg/m^3
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	1.3×10^{-9}
	Inhalation of particles[c] (μg/m^3)	0.48
	Inhalation of vapors[c] (μg/m^3)	0.38
	Oral (mg/kg-d)	2.6×10^{-2}
Hazard index[a] for non-cancer effects	Dermal[d]	3.3×10^{-10}
	Inhalation of particles[e]	3.4×10^{-5}
	Inhalation of vapors[e]	2.7×10^{-5}
	Oral	6.5×10^{-3}
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	1.2×10^{-12}
	Inhalation of particles	1.2×10^{-7}
	Inhalation of vapors	9.7×10^{-8}
	Oral	6.7×10^{-7}

[a] The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.

[b] Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.

[c] Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.

[d] Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.

[e] Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.

N/C, not calculated because of inadequate data.

No data were found indicating effects of DBDE on reproductive and developmental toxicity from oral exposure. Similarly, no data were found for immunological or neurological effects or for genotoxicity. Table 6.5 gives details of all the analyses.

Conclusion: The NRC committee concluded that there are inadequate subchronic and chronic dermal and inhalation toxicity data to establish either a dermal RfD or an inhalation RfC for DBDE. In addition, there

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

are no dermal absorption data. DBDE is nearly insoluble in water and has a relatively low vapor pressure. It is used as a fabric back-coating with antimony trioxide and is encapsulated in a polymer matrix. Based on existing reviews of toxicity and use information, and the its conservative exposure assumptions, the NRC subcommittee concluded that no further research is needed for assessing health risks from DBDE. Note that, in spite of these conclusions, and others, following negotiations with the US EPA, the three companies manufacturing DBDE have agreed to phase out production and sale of the brominated flame retardant by the end of 2013. Albemarle and Chemtura, which produce DBDE in the U.S., and Israeli ICL Industrial Products (IP), the largest US importer of the chemical, say they will end sales for all remaining 'essential uses' by the end of 2013.

6.5.2 Hexabromocyclododecane (HBCD)

Non-cancer dermal exposure: The assessment of non-cancer risk for the dermal route of exposure of HBCD is based on the same scenario as for DBDE. As a first estimate of exposure, it was assumed that the skin and clothing of the person sitting on the couch, and the fabric of the couch, would present no barrier to movement of HBCD. In addition, it was assumed that there would be sufficient water present (e.g., from sweat) to allow dissolution of the HBCD in the water, transfer to the skin and into the body of the sitting individual. The only limiting factor on the transfer rate using these assumptions was assumed to be absorbed immediately by the sitting individual. With the same analysis as before it was estimated that HBCD might pose a non-cancer risk by the dermal absorption route when used as an upholstery fabric flame retardant. Therefore an alternative iteration of the exposure assessment was performed. For the alternative iteration of the dermal assessment, the same exposure assumptions were made as in the first iteration, except that the assumption of immediate absorption of all the HBCD that dissolves was modified. Instead, an estimate of the rate that HBCD could penetrate the skin was determined, assuming that HBCD dissolves up to its solubility limit in water. This rate of penetration was then factored into the exposure assessment. The rate of penetration of a chemical through skin can be estimated and this was done, conservatively, from the octanol-water partition coefficient and the molecular weight. Using this analysis the hazard index indicates that HBCD, used as an upholstery fabric flame retardant, is not likely to pose a non-cancer risk via the dermal exposure route.

Non-cancer inhalation exposure to particles: Inhalation exposure estimates for HBCD in the particulate phase were calculated using the same scenario as for DBDE. For the purpose of estimating a hazard index for the inhalation of HBCD, and in the absence of relevant inhalation exposure date, the subcommittee chose to estimate the inhalation RfC from the oral RfD, again an

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

excessively conservative approach. This RfC should be used as an interim or provisional level until relevant data become available for the derivation of an inhalation RfC for calculating the hazard index. With that provisional RfC, a hazard index was estimated indicating that under the worst-case exposure scenario, HBCD used as an upholstery fabric flame retardant is not likely to pose non-cancer risk via inhalation in the particulate phase.

Non-cancer inhalation exposure to particles: In addition to the possibility of release of HBCD in particles from upholstery fabric, the subcommittee considered the possibility of its release by evaporation and repeated the analyses. The hazard index indicates that, under the worst-case scenario, exposure to HBCD used as an upholstery fabric flame retardant is not likely to pose a non-cancer risk via the inhalation route, when exposures occur in the vapor phase.

Non-cancer oral exposure: The same scenario as before was used with the conclusion that HBCD used as an upholstery fabric flame retardant is not likely to pose a non-cancer risk by the oral route.

Cancer exposure: Based on inadequate carcinogenicity data from any route of exposure, the subcommittee concluded that the potential carcinogenicity of HBCD cannot be determined.

No data were found indicating effects of HBCD on reproductive and developmental toxicity from oral exposure. No studies were found for immunological or neurological effects or for genotoxicity. Table 6.6 gives details of all the analyses.

Conclusion: The NRC committee concluded that there are no subchronic or chronic inhalation or dermal bioassays to evaluate systemic toxicity or carcinogenicity of HBCD. There are no dermal absorption studies. HBCD has a high octanol-water partition coefficient, low vapor pressure, and low solubility in water. It is used as a fabric back-coating and is encapsulated in a polymer matrix. Therefore, direct exposure is likely to be minimal. Based on existing reviews of toxicity and use information, the conservative exposure assumptions, and a hazard index of less than 1 for all exposure routes, the NRC subcommittee concluded that no further research is needed for assessing health risks from HBCD.

6.5.3 Chlorinated paraffins (Chlor Par)

Chlorinated paraffins are complex mixtures that are expected to differ with respect to their chemical content between manufacturers and even between 'batches'. Chlorinated paraffins will differ in the amounts of carbons in their chain and in chlorine content and, thus, in their toxicity. Those most commonly used for back-coating of fabrics are long chained with some 70% chlorine content, often in combination with antimony oxide.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.6 Toxicity data for hexabromocyclododecane (HBCD) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: liver toxicity
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	0.2
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	1.3×10^{-6}
	Inhalation of particles[c] (μg/m^3)	0.48
	Inhalation of vapors[c] (μg/m^3)	3.4
	Oral (mg/kg-d)	2.6×10^{-2}
Hazard index[a] for non-cancer effects	Dermal[d]	6.7×10^{-6}
	Inhalation of particles[e]	$6.8 \times 10–^{6}$
	Inhalation of vapors[e]	$5.0 \times 10–^{3}$
	Oral	0.13
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a] The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.
[b] Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.
[c] Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.
[d] Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.
[e] Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.
N/C not calculated because of inadequate data.
N/A not applicable because the chemical is not carcinogenic by the relevant route.

Non-cancer dermal exposure: The assessment of non-cancer risk for the dermal route of exposure of Chlor Par is based on the same scenario as for DBDE, including all the same first estimates and assumptions. A hazard index of 1.97 indicates that Chlor Par might pose a non-cancer risk by the dermal absorption route when used as an upholstery fabric flame retardant. Therefore an alternative iteration of the exposure assessment was

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

attempted. For the alternative iteration of the dermal assessment, the same exposure assumptions were made as in the first iteration, except that it was assumed that the skin slows the absorption and only a certain amount of Chlor Par is absorbed. This value could not be calculated but it was possible to calculate that dermal exposure would be a concern only if water solubility exceeds 650 g/L, which is not possible. Using this analysis indicates that Chlor Par, used as an upholstery fabric flame retardant, is not likely to pose a non-cancer risk via the dermal exposure route.

Non-cancer inhalation exposure to particles: Inhalation exposure estimates for Chlor Par in the particulate phase were calculated using the same scenario as for DBDE. For the purpose of estimating a hazard index for the inhalation of Chlor Par and in the absence of relevant inhalation exposure date, the subcommittee chose to estimate the inhalation RfC from the oral RfD, again an excessively conservative approach. This RfC should be used as an interim or provisional level until relevant data become available for the derivation of an inhalation RfC for calculating the hazard index. With that provisional RfC, a hazard index of 2.7×10^{-4} was estimated indicating that under the worst-case exposure scenario, Chlor Par, used as an upholstery fabric flame retardant, is not likely to pose a non-cancer risk via inhalation in the particulate phase.

Non-cancer inhalation exposure to particles: Volatility data for Chlor Par were not located and the subcommittee did not calculate a worst-case exposure for this scenario.

Non-cancer oral exposure: The same scenario as before was used and the conclusion, based on a hazard index of 0.053, was that Chlor Par used as an upholstery fabric flame retardant is not likely to pose a non-cancer risk by the oral route.

Cancer exposure: Based on inadequate carcinogenicity data from dermal and inhalation routes of exposure, the subcommittee concluded that the potential carcinogenicity of Chlor Par by these routes cannot be determined. EPA has concluded that long-chain chlorinated paraffins should not be considered as potential carcinogens and it is the opinion of the NRC subcommittee that long-chain chlorinated paraffins are not likely to be human carcinogens and studies of other chlorinated paraffins are not warranted.

No data were found indicating effects of chlorinated paraffins on immunological effects but some neurological effects were found on mice. No reproductive or developmental toxicity effects were found. Table 6.7 gives details of all the analyses.

Conclusion: The NRC committee concluded that dermal toxicity data are lacking. It also felt the need for volatility data, but not for cancer data.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.7 Toxicity data for Chlorinated paraffins (Cl Par) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: liver and kidney toxicity
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	0.3
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	0.59
	Inhalation of particles[c] (μg/m^3)	0.28
	Inhalation of vapors[c] (μg/m^3)	N/C
	Oral (mg/kg-d)	1.6×10^{-2}
Hazard index[a] for non-cancer effects	Dermal[d]	1.9
	Inhalation of particles[e]	2.7×10^{-4}
	Inhalation of vapors[e]	2.7×10^{-4}
	Oral	5.3×10^{-2}
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a] The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.
[b] Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.
[c] Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.
[d] Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.
[e] Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.
N/C not calculated because of inadequate data.
N/A not applicable because the chemical is not carcinogenic by the relevant route.

6.5.4 Tetrakis hydroxymethyl phosphonium salts (chloride salt) (THPC)

This assessment assumes that exposure is to unreacted THPC and does not assess exposure to chemically altered forms of THPC that may form during the application of THPC to upholstery fabrics.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Non-cancer dermal exposure: The subcommittee concluded that THPC is an ionic substance and essentially not absorbed through the skin. In addition, THPC is likely to polymerize after application to the upholstery fabric; therefore, exposure to THPC is not likely to occur and it should not pose a toxic hazard by the dermal route of exposure when used as an FR in furniture upholstery.

Non-cancer inhalation exposure to particles: Inhalation exposure estimates for THPC in the particulate phase were calculated using the same scenario as for DBDE. The hazard index calculated was 4.1×10^{-2}. These findings suggest that under this worst-case exposure scenario, inhalation of THPC particles from furniture upholstery is not likely to pose a non-cancer toxicological risk to humans.

Non-cancer inhalation exposure to vapors: The calculated hazard index is apparently calculated to be 2.2×10^{5}. However, further analysis follows and shows that the real hazard index is minimal if not zero. The original results indicate that if all of the THPC is released from the fabric into the air, THPC could be a toxic risk to persons entering the room. In reality, any FR that evaporated so rapidly would be useless in preventing upholstery flammability. Either THPC is much more strongly bound to the fabric than assumed in this scenario, or the chemical is transformed during the application process. In either case, the emission rate is likely to be controlled by some process other than diffusion through a boundary layer of air, as assumed here. The subcommittee believes that this exposure scenario provides no useful information about the potential toxicity of THPC vapors to humans associated with the emission of THPC vapors from treated furniture upholstery. Therefore, further investigation should be carried out to determine if exposure to THPC by this route poses a toxic risk to humans. These results suggest that the vapor inhalation scenario is unrealistic for THPC-treated furniture in a residential setting because evaporative loss of all THPC over 16 h could not occur under normal conditions. Since THPC is chemically cross-linked within the treated upholstery fabric, the vapor pressure of THPC is assumed to be the vapor pressure for the polymerized form. In the absence of any published data, it is assumed that the polymerized form of THPC will have a vapor pressure approaching zero. Thus, the vapor inhalation non-cancer risk from THPC-treated fabric can be assumed to be minimal if not zero.

Non-cancer oral exposure: The calculated hazard index is 0.313 and indicates that oral exposure to THPC is not likely to be a toxic risk under the given exposure scenario and conditions.

Cancer exposure: The evidence for the dermal carcinogenicity of THPC is equivocal. No evidence was found for dermal carcinogenicity of THPC in mice but there was 'equivocal evidence' for the carcinogenicity of THPC when administered in combination with a known tumor promoter

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

or initiator. The subcommittee concluded that the data are inadequate to determine human carcinogenic potential by the dermal route. No adequate data are available in humans or laboratory animals to assess the carcinogenicity of THPC vapors or particles containing THPC. No evidence of carcinogenicity was found in rats or mice following chronic oral administration of THPC. The subcommittee concluded that THPC, used as an FR in upholstery fabric, is not likely to pose a cancer risk by the oral exposure route.

Table 6.8 gives details of all the analyses.

Conclusion: The NRC committee concluded that hazard indices for the inhalation of particles and oral exposure to THPC are less than one and therefore, these routes of exposure are not anticipated to be a concern. Dermal exposure to THPC through contact with treated material is not expected to occur since THPC is chemically bound to the fabric.

6.5.5 Tris (monochloropropyl) phosphates (TCPP)

Non-cancer dermal exposure: The calculations of dermal exposure to TCPP resulted in an estimated absorbed daily dose of 1.5 mg/kg but insufficient data were available to derive a dermal or oral RfD. Therefore, no conclusions can be drawn concerning the non-cancer health risks associated with dermal exposure to TCPP. Since there was no appropriate data for estimating TCPP dermal penetration, an alternate iteration was not performed for this material. At a later date, extensive exposure data were developed for workers in both manufacturing and downstream, where real monitoring was done with polyurethane foam containing the most widely used TCPP, namely tris (1-chloro-2-isopropyl) phosphate.[34–36] Moreover, dermal penetration studies were conducted in the European Union, including one study of direct penetration and one study of migration from foam to skin.[37–43] These studies included *in vitro* percutaneous absorption of [14C] tris (2-chloro-1-methylethyl) phosphate (TCPP) through human skin membranes using flow-through diffusion cells and *in vitro* percutaneous absorption of neat [14C]TCPP through human skin membranes using flow-through diffusion cells.

Non-cancer inhalation exposure to particles: Inhalation exposure estimates for TCPP in the particulate phase were calculated using the same scenario as for DBDE. No inhalation RfC had been derived from TCPP at the time of the NRC study. Therefore, no conclusions could be drawn by the committee from furniture upholstery fabrics containing TCPP.

Non-cancer inhalation exposure to vapors: Inhalation exposure estimates for TCPP in the vapor phase were calculated using the same scenario as for DBDE. The results obtained suggest that the vapor inhalation scenario is unrealistic for TCPP-treated furniture in a residential setting. Nevertheless, no inhalation RfC was available to NRC for calculating a margin of exposure

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.8 Toxicity data for tetrakis (hydroxymethyl) phosphonium salts (chloride salt) (THPC) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: liver toxicity
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	3.0×10^{-3}
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	—[f]
	Inhalation of particles[c] (μg/m^3)	0.43
	Inhalation of vapors[c] (μg/m^3)	UE
	Oral (mg/kg-d)	9.4×10^{-4}
Hazard index[a] for non-cancer effects	Dermal[d]	N/C
	Inhalation of particles[e]	4.1×10^{-2}
	Inhalation of vapors[e]	Virtually zero
	Oral	0.31
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a] The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.

[b] Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.

[c] Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.

[d] Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.

[e] Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.

[f] Not calculated because THPC is likely to polymerize after application to the upholstery fabric.

N/C not calculated because of inadequate data.

N/A not applicable because the chemical is not carcinogenic by the relevant route.

for TCPP. Therefore, no conclusions could be drawn by NRC about the non-cancer health risks posed by the inhalation of TCPP vapors. It was later found that acute inhalation toxicity studies had been conducted with TCPP on rats.[44,45]

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Non-cancer oral exposure: The worst case average oral daily dose for TCPP was estimated to be 0.04 mg/kg-d. However, a margin of exposure could not be calculated for the oral route because there were insufficient data to derive an oral RfD for this compound.

Cancer: There are insufficient data for assessing the cancer risk from exposure to TCPP by any route of exposure.

Table 6.9 gives details of all the analyses.

Conclusion: The NRC committee concluded that there are no data on the subchronic or chronic toxicity of TCPPs for the dermal, inhalation, or oral routes of exposure. No studies had been conducted at the time on the effects of TCPP exposure on reproduction (but a later study was conducted on two generations of rats, including oral reproduction toxicity and a dose range finding study and no effects were found[42]). Data on the effects of TCPP exposure on reproduction are also not available. Data on the dermal absorption of TCPPs as well as information on human exposure to TCPPs from treated upholstery are also not available. The subcommittee recommended that the potential for release of TCPP vapor into air and TCPP released into saline from treated fabric be investigated. Beyond the NRC committee conclusions, it is important to point out that many of the gaps in knowledge identified by the committee have now been filled.[34–45] Note that most studies on TCPP addressed the most common one of them, namely tris (1-chloro 2-isopropyl) phosphate, but the NRC studies and conclusions addressed all the tris (monochloropropyl) phosphates.

6.5.6 Tris (1,3-dichloropropyl-2) phosphate (TDCPP)

Non-cancer dermal exposure: The assessment of non-cancer risk for the dermal route of exposure of TDCPP was estimated using the dermal exposure scenario as in the earlier cases. As a first estimate of exposure, it was assumed that skin, clothing, and the upholstery did not impede dermal exposure to TDCPP present in the back-coating. It was also assumed that there would be sufficient water present from sweat to facilitate dissolution of TDCPP from the back-coating and absorption through the skin. In this scenario, only the dissolution rate of TDCPP from the back-coating is assumed to be the limiting factor in absorption by the body. It is assumed that all of the TDCPP that dissolves is immediately absorbed into the body by the seated person. For this calculation, the subcommittee estimated an upholstery application rate for TDCPP of 5 mg/cm^2. Using these assumptions, an estimated absorbed daily dose of 1.5 mg/kg was calculated for TDCPP, leading to a hazard index of 300. These results suggest that TDCPP could be a toxic hazard if all available TDCPP is absorbed simultaneously. The alternative iteration calculated the estimated dermal daily dose for TDCPP using an estimate of the dermal penetration rate for TDCPP. Instead of assuming that

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.9 Toxicity data for Tris (monochloropropyl) phosphates (TCPP) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Inadequate data for any route
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	N/C
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	1.5
	Inhalation of particles[c] (μg/m^3)	0.48
	Inhalation of vapors[c] (μg/m^3)	UE
	Oral (mg/kg-d)	4.0×10^{-2}
Hazard index[a] for non-cancer effects	Dermal[d]	—[f]
	Inhalation of particles[e]	—[f]
	Inhalation of vapors[e]	—[f]
	Oral	—[f]
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/C
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a] The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.

[b] Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.

[c] Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.

[d] Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.

[e] Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.

[f] There are inadequate toxicity data from any route of exposure to derive RfDs or RfCs for these compounds. However, structurally related compounds were found to be a health concern at the worst-case exposure levels. Therefore, the NRC subcommittee recommended that exposure measurements be made to determine the need for toxicity studies.

N/C not calculated because of inadequate data.

N/A not applicable because the chemical is not carcinogenic by the relevant route.

all dissolved TDCPP immediately penetrates the skin and enters systemic circulation, it was assumed that the skin slows the absorption of TDCPP to a specific amount of chemical absorbed per unit of time. This estimate can be measured experimentally and is referred to as the skin permeability

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

coefficient. However, the dermal penetration constant for TDCPP has not been measured experimentally though it can be estimated from a correlation between the octanol-water partition coefficient and the molecular weight. With this a hazard index of 0.52 was calculated. At that time, the oral RfD was the best estimate of the internal dose associated with dermal exposure to TDCPP. These results suggest that TDCPP should not be anticipated to pose a toxic risk by the dermal route at the stated application concentrations and under the given worst-case exposure conditions. Note that, although no risk assessment studies (including dermal work) had been conducted at the time of the comprehensive NRC study, European Union risk assessment studies were conducted later, parallel to those for TCPP.[46]

Non-cancer inhalation exposure to particles: Inhalation exposure estimates for TDCPP in the particulate phase were calculated using the same scenario as for DBDE. This gives a hazard index of 2.7×10^{-2}. This suggests that, under the subcommittee's worst-case exposure assumptions, TDCPP would not be considered a toxic hazard by the inhalation route of exposure.

Non-cancer inhalation exposure to vapors: Inhalation exposure estimates for TDCPP in the vapor phase were calculated using the same scenario as for DBDE. These results indicate that, if all of the TDCPP is released from the fabric into the air, TDCPP could be a toxic risk to persons entering the room. The NRC committee correctly points out that, in reality, any flame retardant that evaporated so rapidly would be useless in preventing upholstery flammability. Either TDCPP is much more strongly bound to the fabric than assumed in this scenario or the chemical is transformed during the application process. In either case, the emission rate would likely be controlled by some process other than diffusion through a boundary layer of air, as assumed here. It is the opinion of the subcommittee that this exposure scenario provides no useful information about the potential toxicity of TDCPP vapors to humans associated with the emission of TDCPP vapors from treated furniture upholstery. Therefore, further investigation should be carried out to determine if exposure to TDCPP by this route poses a toxic risk to humans.

Non-cancer oral exposure: The same scenario as before was used and the committee concluded that the calculation suggested TDCPP could be a toxic hazard by the oral route of exposure under the subcommittee's worst-case exposure assumptions.

Cancer dermal exposure: The assessment of cancer risk for the dermal route of exposure of TDCPP was estimated using the dermal exposure scenario as in per earlier cases of non-cancer dermal exposure. As a first estimate of exposure, the subcommittee felt that the use of the oral cancer potency factor for TDCPP based on testicular tumors in rats was acceptable for the calculation of cancer risk for dermal exposure since the oral cancer

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

potency factor is based on carcinogenic effects following near-complete systemic absorption and the appearance of tumors other than at the site of TDCPP application. The NRC estimates suggest that the dermal route of exposure may pose a carcinogenic hazard for persons exposed to TDCPP incorporated into residential furniture upholstery at the indicated concentration levels and under the given worst-case exposure scenario.

Cancer inhalation exposure to particles: Inhalation exposure estimates for TDCPP in the particulate phase were calculated using the same scenario as for DBDE. This gives an estimated lifetime cancer risk of 8.2×10^{-6} and suggests that the cancer risk associated with the inhalation of TDCPP particles is negligible at the given upholstery concentrations and the exposure parameters in the worst-case exposure scenario. However, the subcommittee noted that exposure to TDCPP by this route may need further evaluation.

Cancer inhalation exposure to vapors: Inhalation exposure estimates for TDCPP in the vapor phase were calculated using the same scenario as for DBDE. For TDCPP vapors, the equilibrium concentration of vapor phase TDCPP in room-air was estimated as described in the non-cancer inhalation exposure section, with the same conclusions that the results are unrealistic because of the excessively conservative assumptions.

Cancer oral exposure: From literature studies, TDCPP is judged to be a rodent carcinogen. Therefore, the conservative approach for risk assessment purposes is to assume that TDCPP represents a carcinogenic risk to humans. Using the same criteria as in other evaluations, the lifetime cancer risk for this exposure scenario was estimated to be 6.6×10^{-5}. This suggests that under the NRC subcommittee's worst-case exposure assumptions, TDCPP could be a carcinogenic hazard by the oral route of exposure.

Table 6.10 gives details of all the analyses.

Conclusions: The NRC committee concluded that there are no data on the chronic toxicity of TDCPP by the dermal or inhalation routes of exposure. Data on the rate of dermal absorption of TDCPP are needed and there is no information on the metabolism of TDCPP in animal or human systems. No information is available on human exposure to TDCPP from treated furniture upholstery and no studies have been conducted on the leaching of TDCPP from treated materials. Based on an oral hazard index of greater than 1 and on potential cancer risk from all three routes of exposure, the subcommittee recommended that the potential for particle and vapor release and TDCPP release into saline from treated fabric be investigated.

6.5.7 Ammonium polyphosphates (APP)

Non-cancer dermal exposure: The calculated hazard index is 7.3×10^{-3}. Thus it was concluded that APPs used as FRs in upholstery fabric are not likely to pose a non-cancer risk from dermal exposure.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.10 Toxicity data for tris (1,3-dichloropropyl-2) phosphate (TDCPP) from the NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: testicular atrophy and seminal vesicle effects
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	5.0×10^{-3}
	Cancer potency factor (oral)	6.0×10^{-2} per mg/kg-d[f]
	Cancer potency factor (inhalation)	1.7×10^{-5} per μg/m^3 [f]
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	2.6×10^{-3}
	Inhalation of particles[c] (μg/m^3)	0.48
	Inhalation of vapors[c] (μg/m^3)	UE
	Oral (mg/kg-d)	4.0×10^{-2}
Hazard index[a] for non-cancer effects	Dermal[d]	0.52
	Inhalation of particles[e]	2.7×10^{-2}
	Inhalation of vapors[e]	
	Oral	8
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	1.6×10^{-4}
	Inhalation of particles	8.2×10^{-6}
	Inhalation of vapors	UE
	Oral	6.6×10^{-5}

[a] The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.
[b] Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.
[c] Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.
[d] Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.
[e] Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.
[f] The cancer potency factor following inhalation is for exposure to particles and vapors.
N/C not calculated because of inadequate data.
UE modeling produces unrealistic short-term exposure estimates for this material; therefore, maximum exposures are unknown.

Non-cancer inhalation exposure to particles: In the absence of relevant inhalation exposure data, the subcommittee chose to estimate inhalation RfCs from oral RfDs. The calculated hazard index is 6.8×10^{-7}. These findings suggest that under this worst-case exposure scenario, inhalation of

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

APP particles from furniture upholstery is not likely to pose a non-cancer risk to humans.

Non-cancer inhalation exposure to vapors: APPs are ionic with negligible vapor pressure at ambient temperatures. Therefore, inhalation of APP vapor is not likely to pose a non-cancer risk when incorporated into furniture upholstery.

Non-cancer oral exposure: The calculated hazard index is 2.0×10^{-4}. These results indicate that under the given worst-case exposure scenario, oral exposure to APPs is not likely to pose a non-cancer health risk to humans.

Cancer exposure: There are no adequate data available to assess the carcinogenicity of APPs by the dermal, inhalation, or oral routes.

Table 6.11 gives details of all the analyses.

Conclusions: The NRC committee concluded that no further research is needed for assessing health risks from exposure to ammonium polyphosphates.

6.5.8 Organic phosphonates (e.g., dimethyl hydrogen phosphite) (DMHP)

Non-cancer dermal exposure: The assessment of non-cancer risk for the dermal route of exposure of DMHP is based on the same scenario as for DBDE, including all the same first estimates and assumptions. A hazard index of 18.3 was calculated based on the oral RfD for DMHP of 0.12 mg/kg-d. At the time, the oral RfD was used as the best estimate of the internal dose associated with dermal exposure to DMHP. These results suggest that DMHP could pose a toxic risk from dermal exposure. Therefore an alternative iteration of the exposure assessment was attempted. For the alternative iteration of the dermal assessment, the same exposure assumptions were made as in the first iteration, except that it was assumed that the skin slows the absorption and that only a certain amount of DMHP is absorbed. In the absence of a dermal RfD, the subcommittee decided it was appropriate to use the oral RfD for DMHP of 0.12 mg/kg-d as the best estimate of the internal dose from dermal exposure. Using this analysis a hazard index of 95 was calculated indicating that DMHP, used as an upholstery fabric flame retardant, could pose a non-cancer toxic risk to humans and should be investigated further.

Non-cancer inhalation exposure to particles: In the absence of relevant inhalation exposure data, the subcommittee chose to estimate inhalation RfCs from oral RfDs. The calculated hazard index is 1.73×10^{-3}. These findings suggest that under this worst-case exposure scenario, inhalation of DMHP particles from furniture upholstery is not likely to pose a non-cancer risk to humans.

Non-cancer inhalation exposure to vapors: Inhalation exposure estimates for DMHP in the vapor phase were calculated using the same scenario as

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.11 Toxicity data for ammonium polyphosphates (APP) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: calcification of the kidney
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	300
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	2.2
	Inhalation of particles[c] (μg/m^3)	0.71
	Inhalation of vapors[c] (μg/m^3)	N/C
	Oral (mg/kg-d)	5.9×10^{-2}
Hazard index[a] for non-cancer effects	Dermal[d]	7.3×10^{-3}
	Inhalation of particles[e]	6.8×10^{-7}
	Inhalation of vapors[e]	6.8×10^{-7}
	Oral	2.0×10^{-4}
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a]The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.
[b]Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.
[c]Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.
[d]Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.
[e]Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.
N/C not calculated because of inadequate data.
N/A not applicable because the chemical is not carcinogenic by the relevant route.

for DBDE. It was estimated that the concentration could be maintained for approximately 10 h. These results clearly show that the model for this scenario is substantially incorrect for DMHP if it is a useful FR, since any such material would have to be sufficiently well bound to the fabric to stay in place for years. However, the subcommittee has no further information on plausible rates of evaporation of DMHP from treated fabrics, and these calculations suggest that further information is required.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Non-cancer oral exposure: The calculated hazard index is 0.49. These results indicate that under the given worst-case exposure scenario, oral exposure to DMHP is not likely to pose a non-cancer health risk to humans.

Cancer dermal exposure: The assessment of cancer risk for the dermal route of exposure of DMHP is based on the same scenario as for DBDE, including all the same first estimates and assumptions. The lifetime risk estimate is 6.1×10^{-2}. This estimate suggests that the dermal route of exposure may pose a carcinogenic hazard for persons exposed to DMHP incorporated into residential furniture upholstery at the indicated concentration levels rated into residential furniture upholstery at the indicated concentration levels and under the given worst-case exposure scenario. Further evaluation of the cancer risk associated with dermal exposure to DMHP should be conducted.

Cancer inhalation exposure to particles: The average room-air concentration and average exposure concentration to DMHP particles estimated in the previous sections were used for the cancer assessment. The estimated lifetime cancer risk was 1.1×10^{-6}, which suggests that the cancer risk associated with the inhalation of DMHP particulates is negligible at the given upholstery concentrations and the exposure parameters in the worst-case exposure scenario. However, the subcommittee concluded that exposure to DMHP by this route needs further evaluation.

Cancer inhalation exposure to vapors: For DMHP vapors, the equilibrium concentration of vapor-phase DMHP in room-air was estimated as described in the non-cancer inhalation exposure section. The upper bound on lifetime cancer risk for inhalation exposure to DMHP in the vapor phase is 6.6×10^{-4}. This risk estimate indicates that further investigation of cancer risks associated with DMHP vapors should be considered.

Cancer oral exposure: DMHP is judged by the subcommittee to be a rodent carcinogen. Therefore, the conservative approach for risk assessment purposes is to assume that DMHP represents a carcinogenic risk to humans. Lifetime cancer risk for this exposure scenario was then estimated yielding a cancer-risk estimate of 9.1×10^{-6}. This suggests that under the subcommittee's worst-case exposure assumptions, DMHP could be a carcinogenic hazard by the oral route of exposure.

Table 6.12 gives details of all the analyses.

Conclusions: The NRC committee concluded that further research is needed for assessing health risks from exposure to organic phosphonates. There are no data on the subchronic or chronic toxicity of DMHP by the dermal or inhalation routes of exposure. No information is available on human exposure to DMHP from treated furniture upholstery fabrics. No studies have been conducted on the leaching of DMHP from treated materials. Hazard indices greater than 1 were calculated for DMHP for the dermal route of exposure. Cancer risk estimates were greater than 1×10^{-6} for the

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.12 Toxicity data for organic phosphonates (e.g., dimethyl hydrogen phosphite) (DMHP) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: lung hyperplasia and alveolar/ bronchiolar adenomas or carcinomas observed
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	0.12
	Cancer potency factor (oral)	5.4 ×10^{-2} per mg/kg-d[f]
	Cancer potency factor (inhalation)	1.5 ×10–6 per μg/m^3 [f]
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	2.2
	Inhalation of particles[c] (μg/m^3)	0.72
	Inhalation of vapors[c] (μg/m^3)	UE
	Oral (mg/kg-d)	5.9 × 10^{-2}
Hazard index[a] for non-cancer effects	Dermal[d]	18.3
	Inhalation of particles[e]	1.7 × 10^{-3}
	Inhalation of vapors[e]	
	Oral	0.49
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	6.1 × 10^{-2}
	Inhalation of particles	1.1 × 10^{-6}
	Inhalation of vapors	6.6 × 10^{-4}
	Oral	9.1 × 10^{-6}

[a]The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.

[b]Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.

[c]Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.

[d]Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.

[e]Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.

[f]The cancer potency factor following inhalation is for exposure to particles and vapors.

N/C not calculated because of inadequate data.

UE modeling produces unrealistic short-term exposure estimates for this material; therefore, maximum exposures are unknown.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

dermal, inhalation, and oral routes of exposure. Therefore, the subcommittee concluded that future research for DMHP should focus on determining the actual amounts leached from treated furniture and the dermal penetration of these compounds through human skin. Cancer potency slope factors were available for oral and inhalation routes. Because DMHP is soluble in water, there is concern about non-cancer effects after dermal absorption and concern about cancer risk by all three routes of exposure. The subcommittee recommended that the potential for release of vapor and particles into air and DMHP release into saline from treated fabric be investigated. Because of a dermal hazard index of greater than 1, the subcommittee also recommended that the dermal absorptions of DMHP from treated fabric be investigated.

6.5.9 Aromatic phosphate plasticizers (e.g., tricresyl phosphate) (TCP)

Non-cancer dermal exposure: The assessment of non-cancer risk for the dermal route of exposure of TCP is based on the same scenario as DBDE, including all the same first estimates and assumptions. A hazard index of 21.3 was calculated based on using the oral RfD for TCP of 7×10^{-2} mg/kg-d. These results suggest that TCP could be a toxic hazard if all applied TCP is absorbed into the body simultaneously. This is an impossible event. Using the same alternative approach as for other materials a hazard index of 4.3×10^{-2} was calculated. These results suggest that TCP is not anticipated to be a toxic risk by the dermal route at the stated applications concentrations and under the worst-case exposure scenario.

Non-cancer inhalation exposure to particles: In the absence of relevant inhalation exposure data, the subcommittee chose to estimate inhalation RfCs from oral RfDs. The calculated hazard index is 1.9×10^{-3}. These findings suggest that under this worst-case exposure scenario, inhalation of TCP particles from furniture upholstery fabrics is not likely to pose a non-cancer risk to humans.

Non-cancer inhalation exposure to vapors: Inhalation exposure estimates for TCP in the vapor phase were calculated using the same scenario as for DBDE. The results obtained suggest that the equilibrium room-air concentration for TCP could be maintained for approximately 10 years. This yields a hazard index of 1.7, which indicates that inhalation exposure at the worst-case levels might pose a non-cancer risk.

Non-cancer oral exposure: The calculation gives a hazard index of 0.57. This suggests that under the worst-case exposure assumptions applied by the subcommittee, TCP is not likely to pose a health risk by the oral route of exposure.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Cancer dermal exposure: There are no studies available to evaluate the carcinogenicity of TCP in humans or laboratory animals following dermal exposure.

Cancer inhalation exposure: There are inadequate data to assess the carcinogenicity of TCP in humans or animals following inhalation exposure.

Cancer oral exposure: TCP is not likely to be a human carcinogen by the oral route of exposure. Therefore, TCP is not anticipated to cause cancer in humans from oral exposure to treated furniture upholstery.

Table 6.13 gives details of all the analyses.

Conclusions: The NRC committee concluded that there are no chronic toxicity data for TCP for the dermal and inhalation routes of exposure. There is no information on the types and amounts of TCP species from upholstery back-coating, or on the leaching of these species from upholstery back-coating. Information on the dermal penetration of TCP and its possible derivatives would be helpful. Based on an inhalation hazard index greater than 1, the subcommittee recommended that the potential for vapor release from treated fabric should be investigated.

6.5.10 Phosphonic acid (3-{[hydroxymethyl] amino}-3-oxopropyl)-dimethyl ester (PA)

The following exposure and risk assessment assumes that PA is not chemically altered during the application and curing processes and that leachates from treated upholstery fabrics are unreacted PA. The subcommittee notes that this assumption may be inaccurate, but currently there are no data on the chemical forms and amounts of cured PA present in PA-treated upholstery fabrics following the curing process. Some unreacted PA is left on the fabric surface after curing, but in reality much of the free PA present on treated fabrics has evaporated from the fabric or is washed away in the alkaline wash after curing. Therefore, there is very little free PA present on newly treated fabrics that is available for uptake by end-users. It is known that some PA can be released from the treated fabric by ambient hydrolysis over a period of years.

Non-cancer dermal exposure: The assessment of non-cancer risk for the dermal route of exposure of PA is based on the same scenario as for DBDE, including all the same first estimates and assumptions. Using these assumptions, an estimated absorbed daily dose of 0.028 mg/kg was calculated for PA. No adequate data are available to calculate dermal RfD for PA. In addition, PA is likely to polymerize after application to the upholstery fabric; therefore, exposure to PA is not likely to occur and it should not pose a toxic risk by the dermal route of exposure when used as a FR in furniture upholstery.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.13 Toxicity data for aromatic phosphate plasticizers (e.g., tricresyl phosphate) (TCP) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: liver lesions and adrenal gland toxicity
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	7.0×10^{-2}
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	3.0×10^{-3}
	Inhalation of particles[c] (μg/m^3)	0.48
	Inhalation of vapors[c] (μg/m^3)	417
	Oral (mg/kg-d)	4.0×10^{-2}
Hazard index[a] for non-cancer effects	Dermal[d]	4.3×10^{-2}
	Inhalation of particles[e]	1.9×10^{-3}
	Inhalation of vapors[e]	1.7
	Oral	0.57
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a]The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.
[b]Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.
[c]Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.
[d]Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.
[e]Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.
N/C not calculated because of inadequate data.
N/A not applicable because the chemical is not carcinogenic by the relevant route.

Non-cancer inhalation exposure to particles: Using the same scenario a time-averaged exposure concentration for particles was calculated but, since no inhalation RfC is available for calculating a hazard index, no conclusions can be drawn about the non-cancer toxicological risks posed by the inhalation of PA-containing particles.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Non-cancer inhalation exposure to vapors: Using the same scenario, it was estimated that the equilibrium room-air concentration for PA could be maintained for approximately 39 min. These results suggest that the vapor inhalation scenario is unrealistic for PA-treated furniture in a residential setting. This conclusion refers to the worst-case assumption. In practice, and with an acceptable fabric cleaning regime, the flame retardant is most likely to be in a fully polymerized form and so have zero vapor pressure. However, since no inhalation RfC is available for calculating a hazard index, no conclusions can be made about the non-cancer toxicological risks posed by the inhalation of PA vapors.

Non-cancer oral exposure: The worst-case average oral daily dose for PA was estimated as 7.5×10^{-4} mg/kg-d. Therefore, the level of exposure to PA by the oral route is anticipated to be small, given the worst-case parameters and conditions used in the exposure calculation. No oral RfD is available for calculating a hazard index; therefore, no definitive conclusions can be drawn concerning non-cancer toxicological risks associated with oral exposure to PA in upholstered fabric.

Cancer exposure: No data are available on the carcinogenicity of PA by the dermal, oral, or inhalation routes of exposure.

Table 6.14 gives details of all the analyses.

Conclusions: The NRC committee concluded that key information on the types and amounts (and ratios) of PA derivatives present in treated upholstery fabrics are not available. Information on exposure levels to PA from the dermal, inhalation, and oral routes or on the dermal penetration of PA, or on the amounts of PA leached from treated fabric by simulated body fluids, are all unavailable. No toxicity data are available for the dermal, inhalation, or oral routes of exposure or on the effects of PA on reproduction or development. It is important to note that PA polymerizes within the fiber and fabric structure and may also react with other FR formulation components present; therefore, it is likely to undergo other chemical changes that could alter its chemical properties and toxicity. It is also likely that oxidized forms of this FR will be present in or on the aged FR-treated fabric. The subcommittee recommended that research be conducted to determine whether new chemical species are formed and, if so, to identify them.

6.5.11 Alumina trihydrate (ATH)

Non-cancer dermal exposure: The calculated hazard index is 3.9×10^{-2}. Thus it was concluded that ATH used as FRs in upholstery fabric is not likely to pose a non-cancer risk from dermal exposure.

Non-cancer inhalation exposure to particles: Although lack of sufficient data precludes deriving an inhalation RfC for ATH, the oral RfD, which

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.14 Toxicity data for Phosphonic acid (3-{[hydroxymethyl]amino}-3-oxopropyl)-dimethyl ester (PA) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Inadequate data for any route
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	N/C
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	2.8×10^{-2}
	Inhalation of particles[c] (μg/m^3)	0.35
	Inhalation of vapors[c] (μg/m^3)	UE
	Oral (mg/kg-d)	7.5×10^{-4}
Hazard index[a] for non-cancer effects	Dermal[d]	—[f]
	Inhalation of particles[e]	—[f]
	Inhalation of vapors[e]	—[f]
	Oral	—[f]
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a] The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.

[b] Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.

[c] Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.

[d] Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.

[e] Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.

[f] There are inadequate toxicity data for phosphonic acid from any route of exposure to derive RfDs or RfCs; this chemical is likely to crosslink with fabric components, precluding dermal exposure to this FR in furniture upholstery.

N/A not applicable because the chemical is not carcinogenic by the relevant route.

N/C not calculated because of inadequate data.

UE modeling produces unrealistic short-term exposure estimates for this material; therefore, maximum exposures are unknown.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

represents a very conservative estimate, was used to estimate an RfC. This yielded a hazard index of 1.4×10^{-4}, indicating that under the worst-case exposure scenario, exposure to ATH, used as an upholstery fabric flame retardant, is not likely to pose a non-cancer risk from exposure to particles.

Non-cancer inhalation exposure to vapors: In view of ATH having negligible vapor pressure at ambient temperatures, the subcommittee concluded that exposure to ATH vapors from its use as an upholstery fabric flame retardant is not likely to pose a non-cancer risk.

Non-cancer oral exposure: The calculated hazard index is 1.0×10^{-3}. These results indicate that under the given worst-case exposure scenario, oral exposure to ATH is not likely to pose a non-cancer health risk to humans.

Cancer exposure: There are no adequate data available to assess the carcinogenicity of ATH by the dermal, inhalation, or oral routes.

Table 6.15 gives details of all the analyses.

Conclusions: The NRC committee concluded that ATH is used extensively in antacids and cosmetics, and the hazard indices are less than 1 for all routes of exposure using the conservative assumptions of the subcommittee. Therefore, the it concluded that further research is not needed to assess the health risks from ATH when used as a flame-retardant chemical in furniture upholstery fabric.

6.5.12 Magnesium hydroxide (MDH)

Non-cancer dermal exposure: The subcommittee concluded that MDH is an ionic substance and, therefore, is essentially not absorbed through the skin and should not pose a health risk from the dermal route of exposure when used as an FR in furniture upholstery fabrics. However, to be conservative, the subcommittee assumed that ionized MDH permeates the skin at the same rate as water. Using that permeability rate, and the oral RfD for MDH as the best estimate for the worst-case scenario of the internal dose for dermal exposure, yields a hazard index of 1.4×10^{-4}. Thus, it was concluded that MDH used as an FR in upholstery fabrics is not likely to pose any non-cancer risk by the dermal route.

Non-cancer inhalation exposure to particles: Although lack of sufficient data precludes deriving an inhalation RfC for MDH, the oral RfD, which represents a very conservative estimate, was used to estimate an RfC. This yielded a hazard index of 9.1×10^{-6}, indicating that under the worst-case exposure scenario, exposure to MDH, used as an upholstery fabric flame retardant, is not likely to pose a non-cancer risk from exposure to particles.

Non-cancer inhalation exposure to vapors: In view of MDH having negligible vapor pressure at ambient temperatures, the subcommittee concluded

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.15 Toxicity data for alumina trihydrate (ATH) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: developmental toxicity
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	1.5
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	5.9×10^{-2}
	Inhalation of particles[c] (μg/m^3)	0.71
	Inhalation of vapors[c] (μg/m^3)	N/C
	Oral (mg/kg-d)	1.6×10^{-3}
Hazard index[a] for non-cancer effects	Dermal[d]	3.9×10^{-2}[f]
	Inhalation of particles[e]	1.4×10^{-4}
	Inhalation of vapors[e]	1.4×10^{-4}
	Oral	1.0×10^{-3}
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a]The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.
[b]Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.
[c]Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.
[d]Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.
[e]Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.
[f]The cancer potency factor following inhalation is for exposure to particles and vapors.
N/A not applicable because the chemical is not carcinogenic by the relevant route.
N/C not calculated because of inadequate data.

that exposure to MDH vapors from its use as an upholstery fabric flame retardant is not likely to pose a non-cancer risk.

Non-cancer oral exposure: The calculated hazard index is 1.7×10^{-3}. These results indicate that under the given worst-case exposure scenario, oral exposure to MDH is not likely to pose a non-cancer health risk to humans.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.16 Toxicity data for Magnesium hydroxide (MDH) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: derived from tolerable upper limit for Mg-induced diarrhea in humans
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	12
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	1.7×10^{-3}
	Inhalation of particles[c] (μg/m^3)	0.38
	Inhalation of vapors[c] (μg/m^3)	N/C
	Oral (mg/kg-d)	2.1×10^{-2}
Hazard index[a] for non-cancer effects	Dermal[d]	1.4×10^{-4}
	Inhalation of particles[e]	9.1×10^{-6}
	Inhalation of vapors[e]	9.1×10^{-6}
	Oral	1.7×10^{-3}
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a]The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.
[b]Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.
[c]Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.
[d]Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.
[e]Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.
N/A not applicable because the chemical is not carcinogenic by the relevant route.
N/C not calculated because of inadequate data.

Cancer exposure: The subcommittee concluded that MDH is not likely to be carcinogenic to humans by the oral route. There are no adequate data available to assess the carcinogenicity of MDH by the dermal or inhalation routes.

Table 6.16 gives details of all the analyses.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Conclusions: The NRC committee concluded that, because the hazard indices for non-cancer effects for dermal, inhalation, and oral routes of exposure are less than 1, no further research is needed for assessing health risks from MDH when used as a flame-retardant chemical in furniture upholstery fabric.

6.5.13 Zinc borate (ZB)

Non-cancer dermal exposure: The calculated hazard index is 1.0×10^{-2}. Thus, it was concluded that ZB used as an FR in upholstery fabrics is not likely to pose any non-cancer risk by the dermal route.

Non-cancer inhalation exposure to particles: Although lack of sufficient data precludes deriving an inhalation RfC for ZB, the oral RfD, which represents a very conservative estimate, was used to estimate an RfC. This yielded a hazard index of 9.1×10^{-5}, indicating that under the worst-case exposure scenario, exposure to ZB, used as an upholstery fabric flame retardant, is not likely to pose a non-cancer risk from exposure to particles.

Non-cancer inhalation exposure to vapors: In view of ZB's negligible vapor pressure at ambient temperatures, the subcommittee concluded that exposure to ZB vapors from its use as an upholstery fabric flame retardant is not likely to pose a non-cancer risk.

Non-cancer oral exposure: The calculated hazard index is 2.8×10^{-4}. These results indicate that under the given worst-case exposure scenario, oral exposure to ZB is not likely to pose a non-cancer health risk to humans.

Cancer exposure: There are no adequate data available to assess the carcinogenicity of ZB by the oral, dermal or inhalation routes.

Table 6.17 gives details of all the analyses.

Conclusions: The NRC committee concluded that there are little toxicity data available for ZB. Once in the body, ZB readily breaks down to zinc oxide and boric acid, for which there are no chronic studies of carcinogenicity. There are no studies that have measured exposure levels from the use of ZB as a flame retardant in upholstery furniture fabric. However, there are extensive databases on the toxicity of zinc oxide and boric acid, and the hazard indices for ZB, based on those data, are less than 1 for all three routes of exposure, using the conservative assumptions of the subcommittee. Therefore, the subcommittee concluded that no further research is needed to assess the health risks from the use of ZB as a flame retardant.

6.5.14 Antimony trioxide (ATO)

Non-cancer dermal exposure: The calculated hazard index is –0.1. Thus, it was concluded that ATO used as an FR in upholstery fabrics is not likely to pose any non-cancer risk by the dermal route.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.17 Toxicity data for zinc borate (ZB) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: developmental toxicity
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	0.6
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	6.3×10^{-3}
	Inhalation of particles[c] (μg/m^3)	0.19
	Inhalation of vapors[c] (μg/m^3)	N/C
	Oral (mg/kg-d)	1.7×10^{-4}
Hazard index[a] for non-cancer effects	Dermal[d]	1.0×10^{-2}
	Inhalation of particles[e]	9.1×10^{-5}
	Inhalation of vapors[e]	9.1×10^{-5}
	Oral	2.8×10^{-4}
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a]The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.
[b]Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.
[c]Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.
[d]Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.
[e]Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.
N/A not applicable because the chemical is not carcinogenic by the relevant route.
N/C not calculated because of inadequate data.

Non-cancer inhalation exposure to particles: The normal analysis yielded a hazard index of 1.2, indicating that under the worst-case exposure scenario, antimony trioxide might possibly, when used as an upholstery fabric flame retardant, pose a non-cancer risk from exposure to particles.

Non-cancer inhalation exposure to vapors: In view of ATO having negligible vapor pressure at ambient temperatures, the subcommittee concluded that exposure to ATO vapors from its use as an upholstery fabric flame retardant is not likely to pose a non-cancer risk.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Non-cancer oral exposure: The calculated hazard index is 2.6×10^{-3}. These results indicate that under the given worst-case exposure scenario, oral exposure to ATO is not likely to pose a non-cancer health risk to humans.

Cancer exposure: There are no adequate data available to assess the carcinogenicity of ATO by the oral or dermal routes. The lifetime excess cancer-risk estimate from exposure to antimony trioxide as particles is 1.7×10^{-4}. However, the inhalation unit risk (cancer potency factor) of antimony trioxide is itself suspect. Furthermore, even if the reservations concerning the studies are discounted and the inhalation unit risk is considered to be accurate, better exposure assessment is required before any definitive conclusions can be drawn about the carcinogenic risk from ATO via inhalation in the particulate phase. Antimony trioxide has negligible vapor pressure at ambient temperatures, so ATO used as a flame retardant in upholstery fabric is not likely to pose a cancer risk for exposure to vapors.

Table 6.18 gives details of all the analyses.

Conclusions: The NRC committee concluded that little data are available on the toxicity of ATO following dermal exposure but the hazard index of 0.1 indicates that it is not likely to pose a non-cancer risk from dermal exposure. Therefore, the subcommittee did not recommend further research on the effects of antimony trioxide from dermal exposure. The risk characterization used by the subcommittee indicates that ATO might possibly pose a risk for non-cancer and cancer end points via inhalation in the particulate phase. Therefore, better exposure information is essential to accurately assess the risks of ATO use as a flame retardant in upholstery fabric. If that research shows actual exposures are substantially lower than the subcommittee estimated, there will be a reduced need to perform toxicity studies. One study indicated that there are reproductive effects following inhalation of ATO but the purity of the ATO in that study is suspect and other studies of other antimony compounds show no reproductive effects. Further studies would clarify if antimony indeed poses a cancer risk following inhalation exposure. There are no studies that evaluated the chronic toxicity of ATO from the oral route of exposure but the hazard index of 2.6×10^{-3} indicates that ATO is not likely to pose a non-cancer risk from oral exposure. Therefore, the subcommittee did not recommend further studies. With respect to cancer, the effects following inhalation exposure are portal-of-entry specific (i.e., only occur in the lung), and, therefore, the subcommittee did not recommend carcinogenic studies following other routes of exposure. Based on an inhalation hazard index greater than one and a potential cancer risk following inhalation exposure, the subcommittee recommended that the potential for particle release from treated fabrics be investigated.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.18 Toxicity data for antimony trioxide (ATO) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: liver toxicity; Inhalation: non-cancer pulmonary toxicity; lung tumors
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	2.0×10^{-4}
	Oral RfD (mg/kg-d)	0.2
	Cancer potency factor (oral)	
	Cancer potency factor (inhalation)	7.1×10^{-4} per μg/m^3 (inhalation)[f]
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	2.0×10^{-2}
	Inhalation of particles[c] (μg/m^3)	0.24
	Inhalation of vapors[c] (μg/m^3)	N/C
	Oral (mg/kg-d)	5.2×10^{-4}
Hazard index[a] for non-cancer effects	Dermal[d]	0.1
	Inhalation of particles[e]	1.2
	Inhalation of vapors[e]	1.2
	Oral	2.6×10^{-3}
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	1.7×10^{-4}
	Inhalation of vapors	
	Oral	N/A

[a]The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.
[b]Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.
[c]Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.
[d]Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.
[e]Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.
[f]The cancer potency factor following inhalation is for exposure to particles and vapors.
N/A not applicable because the chemical is not carcinogenic by the relevant route.
N/C not calculated because of inadequate data.

6.5.15 Antimony pentoxide and sodium antimonate (APO)

Non-cancer dermal exposure: The dermal or oral RfDs for antimony pentoxide or sodium antimonite were not derived because of a lack of adequate toxicity data. As a result, the non-cancer risk associated with dermal

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

exposure to antimony pentoxide or sodium antimonite, used as a flame retardant, could not be characterized at the time.

Non-cancer inhalation exposure to particles: An inhalation RfC was not available for pentavalent antimony. As a result, the non-cancer risk associated with the inhalation of particles containing antimony pentoxide could not be characterized. However, a structurally similar compound, ATO (with trivalent antimony), was found to be a possible concern for non-cancer effects. Therefore, the subcommittee recommended that exposure levels for these compounds be measured.

Non-cancer inhalation exposure to vapors: In view of antimony pentoxide having negligible vapor pressure at ambient temperatures, the subcommittee concluded that exposure to antimony pentoxide vapors from its use as an upholstery fabric flame retardant is not likely to pose a non-cancer risk.

Non-cancer oral exposure: An oral RfD was not available for antimony pentoxide therefore the non-cancer risk associated with the estimated worst-case daily dose could not be characterized at the time.

Cancer exposure: There were no adequate data available to assess the carcinogenicity of antimony pentoxide by the inhalation, oral or dermal routes.

Table 6.19 gives details of all the analyses.

Conclusions: The NRC committee concluded that there are no subchronic or chronic toxicity studies in the literature on pentavalent forms of antimony from any route of exposure. Additionally, there are no studies on the toxic effects of pentavalent antimony on reproduction or development. There are no exposure measurements from any route of exposure. Based on the lack of toxicological data and possible concern from exposure to a structurally similar compound, antimony trioxide, the subcommittee recommended that the release rates into saline solution and air from fabrics treated with antimony pentoxide or sodium antimonite be investigated.

6.5.16 Calcium and zinc molybdates (Mo)

Non-cancer dermal exposure: Calcium and zinc molybdates are considered to be ionic, and are essentially not absorbed through the skin. However, to be conservative, the subcommittee assumed that ionized calcium and zinc molybdates permeate the skin at the same rate as water. This led to a calculated hazard index of 10. Thus, it was concluded that calcium and zinc molybdates used as flame retardants in upholstery fabric may pose a non-cancer risk by the dermal route at the specified concentration and under the given worst-case exposure scenario.

Non-cancer inhalation exposure to particles: The calculation yielded a hazard index of 0.095, indicating that under the worst-case exposure

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.19 Toxicity data for antimony pentoxide and sodium antimonate (APO) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Inadequate data for any route
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	N/C
	Oral RfD (mg/kg-d)	N/C
	Cancer potency factor (oral)	N/A
	Cancer potency factor (inhalation)	N/A
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	2.0×10^{-2}
	Inhalation of particles[c] (μg/m^3)	0.24
	Inhalation of vapors[c] (μg/m^3)	N/C
	Oral (mg/kg-d)	5.2×10^{-4}
Hazard index[a] for non-cancer effects	Dermal[d]	—[f]
	Inhalation of particles[e]	—[f]
	Inhalation of vapors[e]	—[f]
	Oral	—[f]
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	N/A
	Inhalation of vapors	N/A
	Oral	N/A

[a] The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.

[b] Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.

[c] Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.

[d] Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.

[e] Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.

[f] There are inadequate toxicity data from any route of exposure to derive RfDs or RfCs for these compounds. However, structurally related compounds were found to be a health concern at the worst-case exposure levels. Therefore, the NRC subcommittee recommended that exposure measurements be made to determine the need for toxicity studies.

N/A not applicable because the chemical is not carcinogenic by the relevant route.

N/C not calculated because of inadequate data.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

scenario, exposure to calcium or zinc molybdates, used as upholstery fabric flame retardants, is not likely to pose a non-cancer risk from exposure to particles.

Non-cancer inhalation exposure to vapors: In view of the negligible vapor pressure of calcium and zinc molybdates at ambient temperatures, the subcommittee concluded that exposure to calcium or zinc molybdate vapors from their use as upholstery fabric flame retardants is not likely to pose a non-cancer risk.

Non-cancer oral exposure: The calculated hazard index is 0.28. These results indicate that under the given worst-case exposure scenario, oral exposure to calcium or zinc molybdates is not likely to pose a non-cancer health risk to humans.

Cancer exposure: There are no adequate data available to assess the carcinogenicity of calcium or zinc molybdates by the oral or dermal routes. With regard to inhalation exposure via particles, the same average room-air concentration and average exposure concentration were used as for the non-cancer section. This led to a lifetime risk estimate from exposure to calcium or zinc molybdate in the particulate phase of 5.0×10^{-6}. Calcium and zinc molybdates have negligible vapor pressures at ambient temperatures. Therefore, calcium or zinc molybdate used as upholstery fabric flame retardants are not likely to pose any cancer risk via inhalation in the vapor phase.

Table 6.20 gives details of all the analyses.

Conclusions: The NRC committee concluded that there is a substantial amount of data available on zinc, calcium, and molybdates, but not everything covers the two molybdates typically used as flame retardants. For instance, the oral RfD, inhalation RfC, and cancer potency factor determined by the subcommittee for calcium and zinc molybdates are based on molybdenum. Because of the calculated inhalation lifetime cancer risk for calcium and zinc molybdates, the subcommittee believed that the potential of these chemicals to be released as particles from fabric needs to be investigated. Because of a non-cancer dermal hazard index greater than 1, the dermal absorption of calcium and zinc molybdates from treated fabric should be investigated.

6.6 Summary of toxicity issues associated with individual flame retardants

Tables 6.5–6.20 show that, for most of the 16 most widely used FRs for textiles, the hazard indices for non-carcinogenic effects are less than 1 for all three routes of exposure. Thus they are not a concern, since FRs with hazard indices of less than 1 are unlikely to pose non-cancer health risks even at the worst-case exposure levels. FR chemicals with hazard indices greater than 1

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.20 Toxicity data for Calcium and zinc molybdates (Mo) from NRC

	Critical toxicity end point for derivation of RfD or RfC	Oral: increased uric acid levels; Inhalation: degeneration of respiratory epithelium
	Dermal RfD (mg/kg-d)	N/C
	Inhalation RfC (mg/m^3)	2.0×10^{-3}
	Oral RfD (mg/kg-d)	6.0×10^{-4}
	Cancer potency factor (oral)	
	Cancer potency factor (inhalation)	2.6×10^{-4} per μg/m^3[f]
Estimated worst-case human exposure levels	Dermal (mg/kg-d)	6.3×10^{-3}
	Inhalation of particles[c] (μg/m^3)	0.19
	Inhalation of vapors[c] (μg/m^3)	N/C
	Oral (mg/kg-d)	1.7×10^{-4}
Hazard index[a] for non-cancer effects	Dermal[d]	10
	Inhalation of particles[e]	9.5×10^{-2}
	Inhalation of vapors[e]	9.5×10^{-2}
	Oral	0.28
Upper limits on lifetime excess cancer-risk estimate[b]	Dermal	N/A
	Inhalation of particles	5.0×10^{-6}
	Inhalation of vapors	5.0×10^{-6}
	Oral	N/A

[a]The hazard index is calculated by dividing exposure levels by RfDs or RfCs. A hazard index below 1 indicates that the exposure at the intended levels is not likely to pose non-cancer health risks; a hazard index above 1 was considered to possibly pose a concern for non-cancer effects.
[b]Lifetime excess cancer risk above the background lifetime cancer incidence was calculated by multiplying the cancer potency factor by the exposure estimate. For all routes of exposure, lifetime risk calculations were estimated from lifetime average exposures.
[c]Vapor exposure levels were calculated based on the vapor pressure measurements for unreacted starting material. In reality, most or all material becomes bound to upholstery fabric following curing. Vapor exposure levels for chemical entities formed during the curing process were not calculated.
[d]Toxicity information was not available to derive a dermal RfD; the subcommittee used oral RfDs as best estimates for internal dose from dermal exposure.
[e]Toxicity information was not available to derive an inhalation RfC; inhalation RfCs were estimated from oral RfD data to estimate risk.
[f]The cancer potency factor following inhalation is for exposure to particles and vapors.
N/A not applicable because the chemical is not carcinogenic by the relevant route.
N/C not calculated because of inadequate data.

might pose non-cancer health risks, but such effects are unlikely for hazard indices slightly greater than 1, given the highly conservative assumptions used. Carcinogenic risk assessments performed on the FRs that were found to be or likely to be carcinogenic indicate that some of the estimated excess cancer risks may be greater than 1×10^{-6}. However, the NRC committee

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

concluded that actual carcinogenic risk is likely to be much lower because of the extremely conservative (high) exposure estimates.

Several of the 16 chemicals were actually chemical classes rather than single compounds. In three cases, one chemical was selected as a surrogate on the basis of representativeness, availability of data, and potency. They are: tetrakis (hydroxymethyl) hydronium chloride for the tetrakis (hydroxymethyl) hydronium salts and their compounds; dimethyl hydrogen phosphite for organic phosphonates; and tricresyl phosphate for aromatic phosphate plasticizers. Conclusions are based on the properties of the surrogate and the risk from other members of the class might be different from the risk from the surrogate.

The committee intentionally overestimated exposure levels reflecting a precautionary approach to the protection of public health. Such an approach is common, but has potential shortcomings, since overestimating risks from FRs might result in a net adverse effect on public health if the uses of FRs that could reduce the risks of death and injury from fires were avoided because of minor toxicological risks estimated through excessively conservative assumptions.

Despite the lack of a complete database, the NRC committee concluded that the following FRs can be used on fabrics for residential furniture with minimal risk, even under worst-case assumptions:

- decabromodiphenyl oxide,
- hexabromocyclododecane,
- tetrakis (hydroxymethyl) phosphonium salts (chloride salt).
- ammonium polyphosphates,
- phosphonic acid (3-{[hydroxymethyl]amino}-3-oxopropyl)-dimethyl ester,[1]
- alumina trihydrate,
- magnesium hydroxide, and
- zinc borate.

On the basis of the hazard indices for non-cancer effects and/or the potential for cancer, the subcommittee recommended that exposure studies be conducted on the following FRs to determine whether toxicity studies need to be conducted:

- chlorinated paraffins,
- tris (monochloropropyl) phosphates,
- tris (1,3-dichloropropyl-2) phosphate,
- organic phosphonates (dimethyl hydrogen phosphite),
- aromatic phosphate plasticizers (tricresyl phosphate),
- antimony trioxide,

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

- antimony pentoxide and sodium antimonates, and
- calcium and zinc molybdates.

It is possible that an individual could be exposed by all three routes: oral, dermal, and inhalation. In such cases, the hazard indices or cancer-risk estimates may be summed across the various routes of exposure. This approach is extremely conservative, because it is unlikely that an individual would be exposed at the upper limit for one route of exposure and even less likely that the same individual would be exposed at the upper limits for two or more routes. However, because one route of exposure typically dominates the risk assessments, summing the hazard indices or cancer-risk estimates did not materially change the NRC committee conclusions regarding the safety of flame retardants.

6.7 Environmental effects of smoke with and without flame retardants

The US Environmental Agency Design for the Environment (EPA/Dfe) report points out that exposures of consumers and the environment differ from the exposures of workers and should be evaluated separately. Occupational exposures typically result from direct contact with chemicals at relatively high concentrations during periods when the workers are conducting specific tasks. Conversely, consumers have the potential to be exposed over a much longer period, but they will always be exposed to a much smaller level because the chemical is incorporated into a consumer product. Also, the general population and the environment will be exposed via different pathways than workers and consumers. For example, even someone who does not own a product containing a flame-retarded textile may still be exposed if the chemical leaches from the disposed product into the drinking water supply. Once in the water supply, groundwater, or surface water, there is the potential for it to be ingested by people or consumed by animals. Similarly, if the chemical is released to the atmosphere during manufacture, use, or disposal, it may settle out on food crops and be ingested directly by people or animals. If the chemical is bioaccumulative, it may concentrate in the animal and reach people through the food chain. For these reasons, exposure to the environment and the general population should be assessed independently from occupational exposure.

While a fully quantitative exposure assessment is outside the scope of this chapter, the primary exposure pathways for environment, general population, and consumer are discussed in this section. The EPA report identifies a set of positive attributes to be considered when formulating or selecting a flame retardant to meet or exceed fire safety requirements. These attributes are linked to different aspects of what might happen to a chemical during

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

its life cycle. While ensuring that fire safety is met, the following desirable human health and environmental chemical characteristics and attributes, relevant to many flame retardants, should be considered general 'rules of thumb' or goals, and should be applied to both the flame retardants and any combustion by-products:

1. low hazard to human health and low exposure potential,
2. low ecotoxicity,
3. readily degradable, and
4. low bioaccumulation.

The overall potential risk posed to human health by any chemical, including a flame retardant, is a combination of hazard and exposure. Chemical hazards to human health include acute toxicity, skin sensitization, carcinogenicity, immunotoxicity, reproductive effects, developmental effects, neurological effects, systemic effects, and genotoxicity. Chemical exposure to humans can occur through the skin, inhalation, and ingestion, and is affected by various physicochemical factors of the additive.

Ecotoxicity measures adverse effects observed in living organisms especially aquatic organisms (e.g., fish, invertebrates, algae). Both acute and chronic ecotoxicity need to be considered.

Persistence describes the tendency of a chemical to resist degradation and removal from environmental settings, such as air, water, soil, and sediment. Chemical degradation in the environment occurs through chemical reactivity with its surroundings or through biodegradation by microorganisms. Chemical reactivity is most commonly a result of hydrolysis (reactions with water), photolysis (reactions with sunlight) or oxidative gas-phase processes. In the absence of rapid chemical reactivity, biodegradation is the primary process, either in aerobic settings, via oxidative processes, or in anaerobic settings, via reductive processes. The environmental profile of a chemical improves with its rate of biodegradation. The OECD defines a chemical as readily biodegradable if it biodegrades 60% or more within 10 days of the time when degradation first reaches 10%. Hydrophobic components (such as unsaturated linear alkyl chains) undergo biodegradation more rapidly under aerobic conditions, in sewage treatment plants and the environment, than highly branched chains. Also, hydrophobic and hydrophilic components linked by an easily biodegradable group like a carboxylic acid ester will separate the hydrophobic portion from the hydrophilic portion during the first step through aerobic biodegradation, such as ester hydrolysis. While the rate of degradation is important, it is equally important to be aware of the degradation by-products, which might be more toxic and persistent than the parent compound. It should be noted that flame retardants must be sufficiently persistent within the material in which they

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

are contained so that the fire safety effect is still available when needed. It is also important to note that the technical requirements for flame retardants in textiles, such as high hydrolysis stability, make it impossible to use flame retardants of low chemical stability.

Even if a chemical has negative human health and environmental attributes, concerns may be mitigated if the chemical is permanently incorporated into a commercial product. Thus, the potential for direct exposure is greatly decreased or eliminated. Reactive flame retardants are incorporated into the material or product at early stages of manufacturing, resulting in a loss of the chemical identity of the flame retardants. Additives are mixed throughout the formulation but not chemically bound. Therefore, additives have a much higher potential to migrate, or leach, from the product into the environment. For example, tetrabromobisphenol A (TBBPA, typically used for printed circuit boards) is reacted into the epoxy resin of the board to form a brominated epoxy before the laminate production process begins. This brominated epoxy is the actual flame retardant that provides fire safety. Studies have shown that levels of free, unreacted TBBPA in the brominated epoxy are extremely low. It was found that only 4 μg TBBPA were unreacted for each gram of TBBPA used.

All of these health and environmental considerations are presented in the summary table (Table 6.21). The table only contains information regarding the inherent hazards of flame-retardant (FR) chemicals. Evaluation of risk must consider both the hazard and exposure associated with FR chemicals, as well as the hazard and exposure associated with combustion and degradation by-products. The caveats listed in the legend and footnote sections must be taken into account when interpreting the hazard information in the table.

Multiple factors must be considered when selecting an appropriate flame retardant. In addition to fire performance properties and health and environmental considerations, the flame retarded product must meet all required technical specifications. Since the materials used for many products have complex chemical formulations, a drop-in exchange of flame retardant is usually impossible and some formulation adjustment is required. Formulation changes can significantly affect ease of manufacture as well as performance and compatibility with existing production and processing equipment. Of course, the resulting product must be economically competitive, based not just on the flame retardant but the complete formulation of the resulting product. The EPA/Dfe report explains that such practical considerations must be weighed up together with the human health and environmental issues considered. It is important to note that the key recommendations from the EPA/Dfe project (in particular Table 6.21) show that none of the flame retardants studied (including TBBPA) have such serious human health or environmental effects that there is any need for disqualification from use.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.21 EPA screening level toxicology hazard summary

Chemical	Human health effects				
	Acute toxicity	Skin sensitizer	Cancer hazard	Immunotoxicity	Reproductive
Reactive flame-retardant chemicals[2]					
Tetrabromobisphenol A (TBBPA)[3]	L	L	***L***	L	L
DOPO (6H-Dibenz[c,e][1,2] oxaphosphorin, 6-oxide)	***L***	***L***	***L***	***L***	***L***
	L	L	***L***	***L***	***L***
Reaction product of TBBPA, Phenol, 4,4′-(1-methylethylidene) bis[2,6-dibromo-, polymer with (chloromethyl)oxirane and 4,4′-(1-methylethylidene) bis[phenol])	***L***	M	***M***[◊]	***L***	***M***[◊]
Reaction Product of an epoxy phenyl novolak with DOPO	***L***	M	***M***[◊]	***L***	***M***[◊]
Reaction product of Aryl alkyl phosphonate with bisphenol A, polymer with epichlorohydrin	***L***	***L***	***M***[◊]	***L***	***M***[◊]
Additive flame-retardant chemicals[4]					
Aluminum hydroxide	***L***	***L***	***L***	***M***	***L***
Phosphoric acid diethyl-, aluminum salt	L	L	***L***	***M***	***L***
Melamine polyphosphate[5]	L	***L***	***L***	***L***	***L***
Polyphosphoric acid	L	***L***	***L***	***L***	***L***
Melamine	L	L	L	***L***	***L***
Silicon dioxide, amorphous	L	L	L	***L***	L
Silicon dioxide, crystalline	L	L	H[‡]	H[§]	L
Magnesium hydroxide	L	*L*	L	***L***	***L***

Notes:

L = Low hazard
M[1] = Moderate hazard
H = High hazard
Endpoints in grey text (L, M, and H) were assigned based on experimental data.
Endpoints in black italics (L, M, and H) were assigned using estimated values and professional judgment (Structure Activity Relationships)

[◊] Hazard designations, which are based on the presence of epoxy groups, arise from the analysis of low molecular weight oligomers (molecular weight <1000) that may be present in varying amounts. The estimated human health hazards for higher molecular weight (>1000) components, which contain epoxy groups, are low for these endpoints.

[§] Concern linked to direct lung effects associated with the inhalation of poorly soluble particles less than 10 microns in diameter.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Human health effects				Aquatic toxicity		Environmental	
Developmental	Neurological	Systemic	Genotoxicity	Acute	Chronic	Persistence	Bioaccumulation
M	L	L	L	H	*H*	M	L
L	*L*	*L*	L	*M*	*M*	*L*	*L*
L	*L*	*L*	*L*	*L*	*L*	*H*	*L*
M◊	*L*	*L*	M	*L*	*L*	*M*	*L*
M◊	*L*	*L*	*M*◊	*L*	*L*	*H*	*L*
M◊	*L*	*L*	*M*◊	*L*	*L*	*H*	*L*
L	*M*	L	*L*	H	M	*H*[R]	*L*
M	*M*	L	L	M	*M*	*H*[R]	*L*
L	*L*	*M*	*M*	*L*	*L*	*M*	*L*
L	*L*	*L*	*L*	*L*	*L*	L	*L*
L	*L*	M	M	L	L	M	*L*
L	*L*	H[§]	L	*L*	*L*	*H*[R]	*L*
L	*L*	H[§]	H[§]	*L*	*L*	*H*[R]	*L*
L	*L*	L	*L*	*L*	*L*	*H*[R]	*L*

Notes: Continued

‡ Concern based on potential inhalation of small particles less than 10 microns in diameter that may be present in varying amounts.

R Recalcitrant: substance is or contains inorganics, such as metal ions or elemental oxides, that are expected to be found in the environment >60 days after release.

[1] The moderate designation captures a broad range of concerns for hazard

[2] Reactive FR chemicals and resins may not completely react, and small amounts may be available during other parts of the life cycle.

[3] The EU has published a comprehensive risk assessment for TBBPA in reactive applications. This risk assessment is a valuable source of information.

[4] Although additive flame retardants are present throughout the life cycle of the product, they are locked into the polymer matrix.

[5] Melamine polyphosphate dissociates in water to form polyphosphoric acid and melamine ions. For this reason, Table 6.21 includes both dissociation ions.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

6.8 Life-cycle assessment of environmental effects of using products with and without flame retardants

The final issue that needs to be addressed is a life cycle analysis comparison between the environmental effects of the flame retardants on their own and the environmental effects of the increased levels of combustion products resulting from the use of products that exhibit poor fire performance and, thus, will lead to more fires and more fire victims. This must also be compared with the potential for achieving the same degree of fire performance via the use of inherently fire safe materials and of other techniques (such as barriers) or using active fire protection measures (e.g., sprinklers).

An analysis by Simonson *et al.*[47] of a life cycle assessment (LCA) of TV sets compared TV sets that are required to have flame retarded (or improved fire performance) enclosures (UL 94 V0 flammability rating[48]) with those that do not have such a requirement and simply comply with the poor fire performance associated with a UL 94 HB flammability rating. At the time of the study, European TV sets had to meet the safety requirements of IEC 60065,[49] which permitted enclosures of mains operated electronic and related apparatus for household and similar general use to be rated UL 94 HB. On the other hand, US TVs had to meet UL 1410,[50] which required that the enclosure materials be rated UL 94 V0.

A novel LCA model was defined by the Simonson study for the determination of the environmental cost of measures taken to attain a high level of fire safety. In one application of the model the fire safety performance of the product modeled was attained through the inclusion of a flame retardant additive to the polymeric material used to manufacture the product. The case study concentrated on a comparison between TV sets with an enclosure manufactured from High Impact Polystyrene (HIPS) flame retarded to achieve a UL 94 V0 rating (typical of the US market) and one with non-flame retarded HIPS and with a UL 94 HB rating (typical of the European market). The use of international loss statistics seems to indicate that using UL 94 V0 rated enclosure materials brings the fire risk associated with TV fires down to a very small value, while the European fire risk associated with TV fires is approximately 165 TV fires per year per million TVs where the enclosure material is breached.

The study included large-scale and small-scale (cone calorimeter) fire experiments on both free burning TVs and fully furnished lounge rooms, to provide fire emission data as input to the LCA model. The combustion product species measured included acute toxicants (such as CO, CO_2, HCl, HBr, antimony species, volatile organic compounds (VOCs), and chronic toxicants such as PAH (polycyclic aromatic compounds), PCB (polychlorinated biphenyls), brominated and chlorinated dibenzodioxins and furans,

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

and the typical flame retardant used to render HIPS into an FR HIPS with a UL 94 V0 flammability rating, namely decabromobiphenyl oxide (DBDE) (see also Section 6.5.1). The results obtained are the most detailed measurements of their kind ever made and provide a realistic measure of the environmental cost of a high level of fire safety.

The study included an investigation into the recyclability of commercial quality flame retarded HIPS (FR HIPS) relative to that of non-flame retarded HIPS (non-FR HIPS). The results from the LCA work showed that the presence of the flame retardant does not impede plastic recycling. In fact, the study seemed to show that the grades of FR HIPS used performed better than the non-FR HIPS after a thermo-oxidative aging and recycling cycle. The results also indicated that the flame retardant does not migrate out of the plastic or degrade in the plastic during the aging and recycling process, as shown by the retention of the UL 94 V0 flammability rating of the FR-HIPS even after ageing and recycling.

The LCA study included an investigation of the effect of the presence of flame retarded plastic in the fuel stream of a municipal waste incinerator on the product distribution from the incinerator and an input for the LCA model for energy recovery of the FR HIPS. It also looked at landfills. The full study indicated that the emissions of some key species (such as dibenzodioxins and PAH) are actually lower for the TV with the FR enclosure than for the TV with the non-FR enclosure. This result is critical for the assessment of the environmental impact of the FR TV relative to that of the non-FR TV.

Finally, the study considered the fire risk associated with the use of flame retardants. The analysis of available fire statistics estimated that there are as many as 160 fire fatalities and 2000 fire injuries per year in Europe as a direct result of TV fires, compared to negligible numbers in the US. The results indicate that the hypothesis 'it would be better to allow things to burn more often rather than use flame retardants' is questionable at best. In the case of a number of key toxic species emitted there is a markedly higher total emission over the whole life cycle from the non-FR TV sets than from the FR TV sets for the survey period.

The results of the LCA study show clearly that the energy use within the life cycle is entirely dominated by the period when the TV is in use. In terms of species emissions, however, the period of TV use is the dominant source of CO_2, but not of many other important (and more toxic) species including CO, NO_x, SO_2, hydrocarbons, PAH and dibenzodioxins and furans. In terms of the generation of CO, NO_x and SO_2, the period of TV manufacture or production (including the production of the flame retardant itself) is dominant. The key reason for this is that the industrial use of energy needed for production is generally slightly less efficient than that used to produce the electricity needed for TV use. In the case of these species fires is a relatively

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

Table 6.22 Cancer risk of PAH (as BaP) relative to dioxins and furans (as TCDD)

	Units	Unit risk factor	Non-FR today	FR today	Non-FR future	FR future
BaP equivalents	kg	7×10^{-2} μg/m^3	1.46	3.05×10^{-2}	1.44	7.60×10^{-3}
TCDD equivalents	kg	1.4 μg/m^3	1.36×10^{-5}	1.01×10^{-6}	1.82×10^{-5}	5.71×10^{-6}
Cancer risk factor*	—	—	108 006	30 208	79 154	1331

* (BaP equiv./Unit Risk Factor for BaP)/(TCDD equiv./Unit Risk Factor for BaP).
Today: landfill disposal – Future: incineration disposal.
BaP: Benzo(a)pyrene –TCDD: 2,3,7,8 tetrachlorodibenzo-p-dioxin.

minor source in the whole LCA. In the case of PAH and dibenzodioxins and furans, however, fires are a significant source of the total LCA emissions. If much of the waste is sent to a landfill, there are significant savings in emissions of these species through the inclusion of flame retardants in the TV enclosure. This is entirely due to the major reduction in the number of TV fires. If there is more incineration, PAH are still primarily produced in fires (they are always destroyed in incinerators), but the total emission of brominated dibenzodioxins and furans increases somewhat due to the potential importance of the emissions of these species from inefficient waste incineration (based on an unconfirmed estimation).

The emission of PAH is, however, clearly the one with the greatest potential health and environmental threat. This is illustrated by a comparison between the health and environmental effects of PAH and those of dibenzodioxins and furans (Table 6.22 [47]). In the table, the toxicity (as cancer risk) of all PAHs is assessed as a comparison with the most toxic PAH, namely benzo(a)pyrene (BaP), while the dibenzodioxins and furans are compared with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD).[51,52] TCDD is one of the polychlorinated dibenzo-*p*-dioxins (in short, but inaccurately, also called dioxin) and it is the most potent compound (congener) of the series. From these results it is clear that PAH emissions from all sources represent as much as 100 000 times higher risk than do the emissions of dibenzodioxins and furans. This is mainly due to the massive influence of large emissions of these species from fires and is most obvious when fires are associated with non-FR enclosure TVs. The amount of PAHs released from non-FR TVs, both with landfill and with incineration disposal, is approximately 700 kg per year, while the amount released from FR TVs is approximately 3% of that value.[47]

Previous studies have indicated that low ignitability and low heat release are very important for the life safety impact of fires.[6,7,29,54] Long times to

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

ignition and slow fire development are essential to provide ample time for the discovery of a fire and the subsequent rescue and mitigation. This, as discussed earlier, is much more important than smoke toxicity in determining the survivability of victims exposed to fires. The application of the Fire-LCA model to a TV with a flame retarded enclosure and one with a non-flame retarded enclosure can quantify this effect. Similar Fire-LCA studies have also been performed on cables[54] and upholstered furniture,[55] with comparable results.

The key conclusion from such LCA studies is that flame retardants do not pose environmental damage by virtue of effectively improving fire performance. In fact, in the case of the emission of many key species (including PAH, hydrocarbons, dibenzodioxins and furans) it is now clear that a reduction in the number of fires because of the use of products containing materials with improved fire performance is associated with significant benefit to the environment as well as saving lives from fires.

6.9 Conclusions

This chapter has analyzed the potential hazards of flame retardants from the point of view of smoke toxicity, inherent toxicity, carcinogenicity, and environmental damage. It is apparent that the use of flame retardants presents a benefit to society and the environment. Undoubtedly not all flame retardants ever developed or about to be developed are safe from all points of view, but the use of appropriate scientific knowledge and the regulatory environment can effectively ensure that unsafe materials are kept away from consumers. However, it is essential to understand that flame retardants, as a class, effectively provide improved fire safety via lowering the probability of ignition, the heat released and the amounts of smoke, combustion products, and dangerous environmental toxicants.

6.10 References

1. Cullis, C.F. and Hirschler, M.M., *The Combustion of Organic Polymers*. Oxford University Press, Oxford, UK (1981).
2. Hirschler, M.M., 'Recent developments in flame-retardant mechanisms', in *Developments in Polymer Stabilisation, Vol. 5*, Ed. G. Scott, pp. 107–152, Applied Science Publishers, London (1982).
3. Gay-Lussac, J.L., *Ann. Chim. Phys.*, **18**(2), 211 (1821).
4. Wyld, J. (or O.), British Patent # 551 (1735).
5. US National Research Council, Committee on Toxicology, Subcommittee on Flame-Retardant Chemicals: D.E. Gardiner (chair), J.F. Borzelleca, D.W. Gaylor, S. Green, R. Horrocks, M.A. Jayjock, S. Kacew, J.N. McDougal, R.K. Miller, R. Snyder, G.C. Stevens, R.G. Tardiff and M.E. Vore, *Toxicological Risks of Selected Flame-Retardant Chemicals*, National Academy Press, Washington, DC (2000).

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

6. Babrauskas, V . and Peacock, R.D., 'Heat release rate: the single most important variable in fire hazard', *Fire Safety J.*, **18**, 255–72 (1992).
7. Babrauskas, V., Harris, R.H., Gann, R.G., Levin, B.C., Lee, B.T., Peacock, R.D., Paabo, M., Twilley, W., Yoklavich, M.F . and Clark, H.M., *Fire Hazard Comparison of Fire–Retarded and Non–Fire–Retarded Products*, NBS Special Publication 749, National Bureau of Standards, Gaithersburg, MD (1988).
8. Purser, D.A., 'The harmonization of toxic potency data for materials obtained from small and large scale fire tests and their use in calculations for the prediction of toxic hazard in fire', *Fire and Materials*, First International Conference and Exhibition, Crystal City, VA, 24, 25 September 1992 (data incorporated into oral presentation but not included in written paper).
9. Debanne, S.M., Hirschler, M.M. and Nelson, G.L., 'The importance of carbon monoxide in the toxicity of fire atmospheres', in *Fire Hazard and Fire Risk Assessment*, Ed. M.M. Hirschler, pp. 9–23, ASTM STP 1150, American Society for Testing and Materials, Philadelphia, PA (1992).
10. Hirschler, M.M. (Editor-in-chief), Debanne, S.M., Larsen, J.B. and Nelson, G.L., *Carbon Monoxide and Human Lethality – Fire and Non-fire Studies*, Elsevier, London, UK (1993).
11. Anderson, R.A., Thomson, I. and Harland, W.A., 'The importance of cyanide and organic nitriles in fire fatalities', *Fire Mater.*, **3**, 91–99 (1979).
12. Anderson, R.A., Willetts, P. and Harland, W.A., 'Fire deaths in the United Kingdom 1976–82', *Fire Mater.*, **7**, 67–72 (1983).
13. Pounder, D.J., *Bodies Recovered from Fires (Forensic Medicine)*, University of Dundee, Scotland, UK, (1992) at http://www.dundee.ac.uk/forensicmedicine/llb/fires.htm.
14. Hirschler, M.M., 'Fire hazard and toxic potency of the smoke from burning materials', *J. Fire Sci.*, **5**, 289–307 (1987).
15. Hinderer, R.K . and Hirschler, M.M., 'The toxicity of hydrogen chloride and of the smoke generated by poly (vinyl chloride), including effects on various animal species, and the implications for fire safety', in ASTM E-5 Symposium on Smoke, Dec. 3, 1988, Phoenix (AZ), *Characterization and toxicity of smoke*, Ed. H.J. Hasegawa, pp. 1–22, ASTM STP 1082, American Society for Testing and Materials, Philadelphia, PA (1990).
16. Kaplan, H.L., Grand, A.F., Switzer, W.G., Mitchell, D.S., Rogers, W.R. and Hartzell, G.E., 'Effects of combustion gases on escape performance of the baboon and the rat', *J. Fire Sci.*, **3**, 228–244 (1985).
17. Burgess, W.A., Treitman, R.D. and Gold, A., *Air contaminants in structural fire-fighting*, NFPCA Project 7X008, Harvard School of Public Health, Cambridge, MA (1979).
18. Grand, A.F., Kaplan, H.L. and Lee, G.H., *Investigation of combustion atmospheres in real fires*, USFA Project 80027, Southwest Research Institute, San Antonio, TX (1981).
19. Hirschler, M.M., 'Smoke toxicity. How important is it for fire safety?', in *Business Communications Company Sixth Ann. Conference on Recent Advances in Flame Retardancy of Polymeric Materials, May 23–25, 1995, Stamford, CT*, Ed. M. Lewin, pp. 297–311, Business Communications Company, Norwalk, CT (1995).
20. Beyler, C.L., 'Major species production by diffusion flames in a two-layer compartment fire environment', *Fire Safety J.*, **10**, 47–56 (1986).

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

21. Pitts, W.M., Johnsson, E.L. and Bryner, N.F., 'Carbon monoxide formation in fires by high-temperature anaerobic wood pyrolysis', in *Proceedings of the Twenty-Fifth Symposium (International) on Combustion*, pp. 1455–1462, The Combustion Institute, Pittsburgh, PA (1994).
22. Budnick, E.K., *Mobile Home Living Room Fire Studies*, NBSIR 78–1530, National Bureau of Standards, Gaithersburg, MD (1978).
23. Budnick, E.K., Klein, D.P., and O ' Laughlin, R.J., *Mobile Home Bedroom Fire Studies: The Role of Interior Finish*, NBSIR 78–1531, National Bureau of Standards, Gaithersburg, MD (1978).
24. Levine, R.S . and Nelson, H.E., *Full Scale Simulation of a Fatal Fire and Comparison of Results with Two Multiroom Models*, NISTIR 90–4268, National Institute of Standards and Technology, Gaithersburg, MD (1990).
25. Gottuk, D.T., Roby, R.J., Peatross, M.J. and Beyler, C.L., 'CO production in compartment fires', *J. Fire Prot. Eng.*, **4**, 133–150 (1992).
26. Mulholland, G.W., in W.M. Pitts, p. 25, *Executive Summary for the Workshop on Developing a Predictive Capability for CO Formation in Fires*, NISTIR 89–4093, National Institute of Standards and Technology, Gaithersburg, MD, p. 25 (1989).
27. Babrauskas, V., Harris, R.H., Braun, E., Levin, B.C., Paabo, M . and Gann, R.G., *The Role of Bench–Scale Data in Assessing Real–Scale Fire Toxicity*, NIST Tech. Note # 1284, National Institute for Standards Technology, Gaithersburg, MD (1991).
28. Babrauskas, V., Levin, B.C., Gann, R.G., Paabo . M., Harris, R.H., Peacock, R.D. and Yusa, S., *Toxic Potency Measurement for Fire Hazard Analysis*, NIST Special Publication # 827, National Institute for Standards Technology, Gaithersburg, MD (1991).
29. Hirschler, M.M., 'Fire retardance, smoke toxicity and fire hazard', in *Proceedings of the Flame Retardants '94, British Plastics Federation Editor*, Interscience Communications, London, UK, Jan. 26–27, 1994, pp. 225–37 (1994).
30. ASTM E1678, 'Standard test method for measuring smoke toxicity for use in fire hazard analysis', in *Annual Book of ASTM Standards*, Vol. 4.07, American Society for Testing and Materials, West Conshohocken, PA.
31. NFPA 269, *Standard Test Method for Developing Toxic Potency Data for Use in Fire Hazard Modeling*, National Fire Protection Association, Quincy, MA.
32. Hirschler, M.M., 'Environmental protection agency project on flame retardants in printed circuit boards', *Fire and Materials Conference*, San Francisco, CA, Jan. 26–28, 2009, Carole Franks and Stephen Grayson (eds), Interscience Communications, London, UK , pp. 13–24
33. EPA Design for the Environment, *Partnership to Evaluate Flame Retardants in Printed Circuit Boards*, (2009) draft final report, to be found at: www.epa.gov/dfe/pubs/full_report_pcb_flame_retardants_report_draft_11_10_08_to_e.pdf.
34. European Union Risk Assessment Report on Tris (2-Chloro-1-Methylethyl) Phosphate (TCPP), CAS No.: 13674–84–5, EINECS No.: 237–158–7 Part 1-April 2008, Rapporteur: Ireland and United Kingdom – Contact: Dr Majella Cosgrave, Chemicals Policy and Services, Health and Safety Authority, Dublin, Ireland, 294 pp.
35. European Union Risk Assessment Report on Tris (2-Chloro-1-Methylethyl) Phosphate (TCPP), CAS No.: 13674–84–5, EINECS No.: 237–158–7 Part

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

2 – May 2008, Rapporteur: Ireland and United Kingdom – Contact: Dr Majella Cosgrave, Chemicals Policy and Services, Health and Safety Authority, Dublin, Ireland, 411 pp.
36. Thomas, J., Ghanem, R., Stevens, G., Kandola, B . and Horrocks, D., *Final Report on Flame Retardant Release from Foams: Phase 1*, University of Surrey, Polymer Research Centre, University of Surrey, Guildford, UK (2005).
37. Schupp, T., *Estimation of exposure of consumers against TCPP emitted from flame retarded flexible polyurethane foam*, Europur 2001, Elastogran GmbH, Germany (2001).
38. British Rubber Manufacturers Association (BRMA), 'Determination of Flame Retardant Retention in a Foam Sample, Albright and Wilson and Rhodia Phosphorus and Performance Derivatives' (1998–2005).
39. Freudenthal, R.I. and Henrich, R.T., 'Chronic toxicity and carcinogenic potential of tris-(1,3-dichloro-2-propyl) phosphate in Sprague-Dawley rat', *Int. J. Toxicol.*, **19**, 119–125 (2000).
40. TNO Quality of Life Studies, *In vitro percutaneous absorption of [14C] tris (2-chloro-1-methylethyl) phosphate (TCPP) through human skin membranes using flow-through diffusion cells*, TNO (*Netherlands Organization for Applied Scientific Research*), Delft, Netherlands (2005).
41. TNO Quality of Life Studies, *In vitro percutaneous absorption of neat [14C] TCPP (Tris (2-chloro-1-methylethyl) phosphate) through human skin membranes using flow-through diffusion cells*, TNO (Netherlands Organization for Applied Scientific Research), Delft, Netherlands (2006).
42. TNO Quality of Life Studies, *Oral two-generation reproduction toxicity study (including a dose range finding study) with Tris (2-chloro-1-methylethyl)-phosphate in rats*, TNO (Netherlands Organization for Applied Scientific Research), Delft, Netherlands (2007).
43. Caterall, D., *Investigation of TCPP loss by volatility at ambient temperature and pressure from small particles of a flexible polyurethane foam by GC-MS*, Hall Analytical Laboratories Ltd (2005).
44. Inveresk Research International, *Acute Inhalation Toxicity Study on TMCP in Rats* (1990).
45. Inveresk Research International, *Acute Inhalation Toxicity Study on TMCP in Rats* (1990–1).
46. European Union Risk Assessment Report on Tris [2-Chloro-1-(Chloro Methyl) Ethyl] Phosphate (TDCPP), CAS No.: 13674–87–8, EINECS No.: 237–159–2 – May 2008, Rapporteur: Ireland and United Kingdom – Contact: Dr Majella Cosgrave, Chemicals Policy and Services, Health and Safety Authority, Dublin, Ireland, p. 294.
47. Simonson, M., Blomqvist, P., Boldizar, A., Möller, K., Rosell, L., Tullin, C., Stripple, H. and Sundqvist, J.O., *Fire-LCA Model: TV Case Study*, SP Report 2000:13, SP, Swedish National Testing and Research Institute, Fire Technology Department, Borås, Sweden (2000).
48. UL 94, *Standard for Test for Flammability of Plastic Materials for Parts in Devices and Appliances*, Underwriters Laboratories, Northbrook, IL.
49. IEC 60065, 'Audio, video and similar electronic apparatus – Safety requirements', International Electrotechnical Commission, 3, rue de Varembé, CH 1211, Geneva 20, Switzerland.

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

50. UL 1410, *Television Receivers and High-Voltage Video Products*, Underwriters Laboratories, Northbrook, IL.
51. Spindler, E.J., 'Soot from fires – a risk assessment', *Chemische Technik*, **49**(4), 193–196 (1997).
52. Troitzsch, J.H., 'Fire gas toxicity and pollutants in fires – the role of flame retardants', in *Proceedings of the Flame Retardants 2000, British Plastics Federation Editor*, Interscience Communications, London, UK, Feb. 8–9, 2000, pp. 177–84 (2000).
53. Stevens, G.C . and Mann, A.H., *Risks and Benefits in the Use of Flame Retardants in Consumer Products*, A report for the UK Department of Trade and Industry, DTI Ref: URN 98/1026 (1999).
54. Simonson, M., Andersson, P., Rosell L., Emanuelsson, V. and Stripple H., *Fire-LCA Model: Cables Case Study* SP Report 2001:22, SP, Swedish National Testing and Research Institute, Fire Technology Department, Borås, Sweden (2001).
55. Andersson, P., Simonson, M. and Stripple H., *Fire Safety of Upholstered Furniture – LCA Analysis* SP Report 2003:22, SP, Swedish National Testing and Research Institute, Fire Technology Department, Borås, Sweden (2003).

WPHIRSCHLER
© Woodhead Publishing Limited, 2013

WPHIRSCHLER