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Quantitative Assessment of Potential Health Effects From the Use of Fire Retardant (FR) Chemicals in Mattresses

January 9, 2006

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Executive Summary

To address the hazards associated with the flammability of mattresses, the U.S. Consumer Product Safety Commission (CPSC) staff has developed a performance standard to reduce mattress fires without creating additional hazards to consumers. The CPSC's Directorate for Health Sciences (HS) conducted a preliminary qualitative assessment of the potential risk of health effects from exposure to five fire retardant (FR) chemicals/chemical classes (i.e., antimony trioxide, boric acid/zinc borate, decabromodiphenyl oxide, melamine, and vinylidene chloride) that may be incorporated in mattresses to meet the proposed standard (Thomas and Brundage, 2004). At the time, data on potential exposures to FR chemicals were not available. Therefore, based on the finding of the qualitative risk assessment, CPSC staff conducted a quantitative risk assessment to provide a more accurate estimate of the potential risk to consumers associated with exposures to these FR chemicals/chemical classes in commercially-available FR-treated barriers that may be used by mattress manufacturers to meet the proposed flammability standard.

To quantify the amount of FR chemical(s) that may be released from the barriers, the CPSC's Directorate for Laboratory Sciences, Division of Chemistry (LSC) staff conducted migration/exposure assessment studies on FR-treated mattress barriers. These barriers were treated with a variety of FR chemicals including: ammonium polyphosphate, antimony trioxide, boric acid, decabromodiphenyl oxide, melamine, and vinylidene chloride. The exposure studies were conducted in three sequential phases to determine: the total amount of FR chemical present in the barrier; the potential migration of the FR chemical(s) in the barrier to a surrogate material for the skin of the consumer, to estimate dermal absorption, as well the amount that may be ingested; and the airborne particle-bound release of the FR chemical(s) from the barrier during normal use over 10 years to estimate potential inhalation exposures. There were also limited aging studies to assess the effects of environmental factors, such as heat and humidity, on the release of airborne particle-bound FR chemicals.

HS staff quantitatively assessed all applicable routes of exposure (i.e., dermal, oral, and inhalation) for the FR chemicals for which migration/exposure data were available and determined the potential risk associated with exposure to these FR chemicals. The analysis included estimates of average exposure, as well as the reasonable upper bound exposures.

The results of the exposure and risk assessment of the FR chemicals suggests that there are commercially available FR-treated barriers that can be used to meet a staff's draft final mattress flammability standard that are not expected to pose any appreciable risk of health effects to consumers who sleep on treated mattresses.

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UNITED STATES
CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, DC 20207

Memorandum

Date: January 9, 2006

TO : Margaret Neily, Director, Division of Fire Sciences, Directorate for Engineering Sciences

THROUGH: Mary Ann Danello, Ph.D., Associate Executive Director, Directorate for Health Sciences
Lori E. Saltzman, M.S., Director, Division of Health Sciences

FROM : Treye A. Thomas, Ph.D., Toxicologist, Division of Health Sciences
Patricia Brundage, Ph.D.*, Pharmacologist, Division of Health Sciences

SUBJECT : Quantitative Assessment of Potential Health Effects From the Use of Fire Retardant (FR) Chemicals in Mattresses**

I. Introduction

The U.S. Consumer Product Safety Commission (CPSC) initiated a regulatory proceeding in 2001 to address the hazard of flame ignitions of mattresses (Neily, 2001). From 1999-2002, there were approximately 15,300 fires per year in the U.S. in which mattresses or bedding were the first items ignited, resulting in about 1,750 injuries and 350 deaths annually (Smith and Miller, 2005). In 2004, CPSC proposed a performance standard to reduce mattress ignitions (70 FR 2470). To meet the proposed mattress performance standard, manufacturers of mattresses would be able to select from a number of available technologies (e.g., barriers and foam), some of which might contain fire retardant (FR) chemicals.

Previously, the CPSC's Directorate for Health Sciences (HS) staff conducted a qualitative assessment of the potential risk that might result from consumer exposure to FR chemicals applied to mattresses designed to meet the staff's draft mattress flammability standard (Thomas and Brundage, 2004). Toxicity reviews on five chemicals/chemical classes that may be used to meet staff's draft standard were completed by HS staff. The chemicals reviewed were: antimony trioxide, boric acid/zinc borate, decabromodiphenyl oxide, melamine, and vinylidene chloride. At that time, data on potential exposures to FR chemicals from mattresses did not exist. Because of the lack of exposure data, staff conducted a preliminary qualitative assessment of the potential risk of health effects from exposure to FR chemicals that may be incorporated to meet the staff's draft standard based on their assessment of available toxicity data,

* Dr. Brundage was a major contributor but has very recently left CPSC.
**These comment are those of the U.S. Consumer Product Safety Commission (CPSC) staff, have not been reviewed or approved by, and may not necessarily reflect the views of, the Commission.

knowledge of how FR chemicals might be used in mattresses, and staff's professional judgment.

Recently, CPSC's Directorate for Laboratory Sciences, Division of Chemistry (LSC) staff conducted laboratory migration/exposure assessment studies on mattress components containing FR chemicals to obtain the data needed to quantify the amount of FR chemical that may be released from these mattress components. The purpose of the present report is to quantitatively assess the potential health risks that might result from consumer exposure to FR chemicals in mattresses designed to meet the staff's draft final mattress flammability standard.

A. FR Chemicals and Application in Mattresses

The Commission's staff requested information from manufacturers on existing and potential FR chemical use in products (including barriers, foams, or other materials) that may be used to meet the staff's draft final flammability standard for mattresses.

Flame resistant barrier materials containing FR chemicals are generated using various methods. Manufacturers may apply FR chemicals to a finished component product, possibly as a backcoating. FR chemicals may also be used as a topical treatment by coating fabrics and components with FR chemicals. Some of the topical treatments trap the FR chemical(s) in a resin binder. FR chemicals may also be incorporated into manufactured fibers at the time of the fibers are made. These fibers are then used to create a variety of barriers.

B. Risk Assessment of FR Chemicals

1. Risk Assessment under the Federal Hazardous Substances Act

CPSC staff assesses a product's potential health effects to consumers under the Federal Hazardous Substances Act (FHSA). The FHSA is risk-based. To be considered a "hazardous substance" under the FHSA, a household product must satisfy a two-part definition; 15 USC 1261 (f)(1)(A). First, it must be toxic under the FHSA, or present one of the other hazards enumerated in the statute. Second, it must have the potential to cause "substantial illness or injury during or as a result of customary and reasonably foreseeable handling or use." Therefore, exposure and subsequent risk must be considered in addition to toxicity when assessing potential hazards under the FHSA (CPSC, 1992). The FHSA addresses both acute and chronic hazards. While the FHSA does not require manufacturers to perform any specific battery of toxicological tests to assess the potential risk of chronic health hazards, manufacturers are required to label products appropriately according to the requirements of the FHSA.

The first step in the risk assessment process is hazard identification, that is, a review of the available toxicity data for each chemical under consideration and a determination of whether the chemical is considered to be "toxic" under the FHSA. Acute toxicity is

defined by the acute oral median lethal dose[1] (LD_{50}) values in animals in regulations issued under the FHSA; 16 CFR 1500.3 (c)(2)(i). In evaluating the chronic toxicity data, CPSC staff apply the definitions for toxicity in the regulations (16 CFR 1500.3 (c)(2)(ii)) and chronic hazard guidelines (CPSC 1992; summarized at 16 CFR 1500.135) promulgated under the FHSA (15 U.S.C. 1261-1278). A substance or mixture is classified as "known to be toxic" in humans only if there is sufficient evidence in humans, and is regarded as "probably toxic" if there is either limited evidence in humans, or sufficient evidence in animals (Table 1). If a chemical or substance is known to be toxic or probably toxic in humans, it is considered "toxic" under the FHSA[2]. If a chemical or substance is possibly toxic, it would not be considered "toxic" under the FHSA.

Table 1. Classification of Chronic Hazards under the FHSA.

Evidence	Human studies	Animal studies
Sufficient evidence	**Known** [a]	**Probable** [a]
Limited evidence	**Probable** [a]	Possible
Inadequate evidence	Possible	---

[a] Considered "toxic" under the FHSA.

2. Health Effects

Determinations of toxicity for several chemicals/chemical classes of FR chemicals that mattress manufacturers may use were made by CPSC staff (Babich et al., 2004; Bittner, 1999; Bittner, 2001; Ferrante, 1999; Hatlelid, 1999a; Hatlelid, 1999b; Thomas and Brundage, 2004) and the National Research Council (National Research Council, 2000). Some of these assessments were completed for previous staff work on FR chemical use in upholstered furniture. The chemicals/chemical classes of interest for use in mattresses are: ammonium polyphosphates, antimony trioxide, boric acid/zinc borate, decabromodiphenyl oxide, melamine, and vinylidene chloride. The staff reviewed and evaluated all the available data for each chemical/chemical class and determined whether they may be considered "toxic" as defined by the FHSA. Acceptable daily intake[3] (ADI) values were calculated when a given chemical was considered "toxic" due to chronic effects and when sufficient toxicity information was available. The chronic health effects assessed include carcinogenicity, neurotoxicity, reproductive and developmental toxicity, and chronic organ toxicity. The risk assessment described in this memo is limited to the assessment of chronic health effects, although the acute health effects of the FR chemicals/chemical classes are also presented. The toxicity information for the

[1] The median lethal dose (LD_{50}) is the amount of a chemical which kills 50 percent of a sample population; typically expressed as milligrams per kilogram of body weight.

[2] While a product may meet the definition of toxic it might not necessarily be a hazardous substance. A substance must meet both parts of a two-part definition in order to be a hazardous substance.

[3] Acceptable daily intake (ADI) is an estimate of the amount of a compound that one may be exposed to on a daily basis without posing a significant risk of health effects. This is typically derived by dividing an experimentally determined no-observed-adverse-effect-level (NOAEL) by uncertainty factors.

chemicals/chemical classes is summarized in Table 2. For antimony trioxide and boric acid, all doses and exposures are expressed in terms of antimony and boron, respectively.

Ammonium Polyphosphate

Staff reviewed the toxicity information on three ammonium polyphosphates considered likely to be used as flame retardants in upholstered fabrics (Ferrante, 1999; Bittner, 2001). Based on the available data, ammonium polyphosphates are not considered to be acutely toxic under the FHSA. Ammonium polyphosphates are not dermal or ocular irritants and are not mutagenic. There were no available data on subchronic or chronic exposures, pharmacokinetics, carcinogenity, or reproductive, developmental, or neurological effects. Ammonium polyphosphates, in staff's opinion, do not meet the definition of "toxic" under the FHSA, and thus, the calculation of an ADI is unnecessary at this time. Furthermore, the National Academy of Sciences' (NAS) National Research Council (NRC), which completed a toxicological risk assessment of 16 FR chemicals, concluded that despite the limited toxicological data, the acute studies indicate that ammonium polyphosphates are "probably not very potent toxicants" and that no further research was needed to assess the health risks due to ammonium polyphosphate exposure (NRC, 2000).

Antimony Trioxide

Staff concluded that antimony trioxide is not acutely toxic by oral or dermal routes. There is sufficient evidence to conclude that antimony trioxide meets the FHSA regulatory definition for toxic based upon its chronic organ toxicity and carcinogenicity in animals (Hatlelid, 1999a). Inhalation of antimony dust caused non-cancerous effects in both animals and humans, and systemic toxicity in several animal species following oral exposure. The inhalation of antimony trioxide also caused lung tumors in animals. Antimony trioxide is a probable human carcinogen based on sufficient evidence of carcinogenicity in animals exposed by inhalation. Antimony trioxide would be considered "toxic" under the FHSA.

CPSC staff calculated an oral ADI of 2.3 mg/kg-day (Hatlelid, 1999a). This was based on the no-observed-adverse-effect-level[4] (NOAEL) of 230 mg/kg-day from a subchronic feeding study in Wistar rats (Sunagawa, 1981) using an uncertainty factor of 100 (10 for interspecies variability and 10 for sensitive populations).

For the inhalation route of exposure, the "inhalation ADI" (the acceptable exposure level for airborne antimony trioxide particles) was 9 ng/m^3 (Bittner, 2001; Hatlelid, 1999a). This was based on the lowest-observed-adverse-effect-level[5] (LOAEL) of 9,000 ng/m^3 for alveolar/intraaveolar macrophage proliferation as a result of chronic inhalation exposure in rats (Newton et al., 1994) using an uncertainty factor of 1000 (10 for interspecies variability, 10 for sensitive populations, and 10 for use of the LOAEL rather than the NOAEL).

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[4] The no-observed-adverse-effect-level (NOAEL) is the experimentally determined dose at which there is no statistically significant adverse effect.

[5] The lowest-observed-adverse-effect-level (LOAEL) is the lowest dose tested with a statistically significant adverse effect.

Boric Acid and Zinc Borate

The toxicity assessment for boric acid and zinc borate is based on the CPSC staff's zinc borate toxicity review and update (Bittner, 2001; Hatlelid, 1999b). Staff considered toxicological information on zinc oxide, boric anhydride and boric acid due to a lack of information on zinc borate, the zinc salt of boric acid. Zinc borate is composed of 45 percent zinc oxide and 34 percent boric anhydride with 20 percent water. Boric acid is formed by the reaction of boric anhydride and water.

Previous staff analyses indicate that boric acid is considered to be acutely toxic by the oral route of exposure. Boric acid is also a probable reproductive and developmental toxicant in humans, based upon sufficient animal data. There is also sufficient evidence of systemic toxicity in animals. There is no evidence of carcinogenicity and neurotoxicity for boric acid. Boric acid meets the definition of "toxic" under the FHSA.

CPSC staff previously derived an oral ADI of 0.088 mg/kg-day for boric acid based upon testicular effects observed in a 90-day study in dogs (Hatlelid, 1999b; Thomas and Brundage, 2004). However, since the completion of these assessments, the U.S. Environmental Protection Agency (EPA) has revised the oral reference dose[6] (RfD) for boron and boron compounds (EPA, 2004), which replaces the RfD that was also based on the NOAEL of 8.8 mg/kg-day determined from the chronic dog studies (Weir and Fisher, 1972). The EPA noted several limitations of the dog studies including, a small number of test animals per dose group (n=4) and the observation of testicular damage in three of the four controls. The EPA chose to base the new RfD on the results of two developmental studies in rats that demonstrated a statistically significant trend of decreasing fetal body weight (Price et al. 1996a; Heindel et al., 1992). In light of the new RfD developed by EPA, CPSC staff has decided to revise the boric acid ADI for oral exposure from 0.088 mg/kg-day to 0.1 mg/kg-day. The revised ADI is based on the NOAEL of 9.6 mg/kg-day (Price et al., 1996) using an uncertainty factor of 100 (10 for interspecies variability and 10 for sensitive populations).

Staff previously determined that zinc oxide is acutely toxic by the oral route of exposure (Bittner, 2001; Hatlelid, 1999b). Zinc oxide would be considered "toxic" under the FHSA as a result of its acute oral toxicity. Based on the limited evidence of systemic toxicity in subchronic feeding studies in ferrets, zinc oxide may be considered possibly toxic to humans. It also is considered a possible developmental and neurological toxicant in humans. Thus, zinc borate does not satisfy the FHSA definition of chronic toxicity.

Decabromodiphenyl Oxide

Decabromodiphenyl oxide has low acute toxicity by the inhalation, oral, and dermal routes of exposure, and thus, is not acutely toxic (Babich et al, 2004; Bittner, 1999; Bittner, 2001). Decabromodiphenyl oxide is, however, considered toxic based on the

[6] Reference dose (RfD) is an estimate of human daily oral exposure that is not expected to have a considerable risk of deleterious effects throughout a lifetime exposure. In contrast to the ADI, a RfD can be calculated from either the NOAEL or benchmark dose (BMD), which is derived from a dose-response curve.

liver and thyroid effects in subchronic and lifetime feeding studies in rodents. Staff concluded that decabromodiphenyl oxide is possibly carcinogenic in humans according to the CPSC's chronic hazard guidelines based on the minimal evidence of carcinogenicity in animals along with the lack of genotoxicity. The conclusion that decabromodiphenyl oxide is a possible carcinogen does not support the finding that decabromodiphenyl oxide is "toxic" based on carcinogenicity. However, decabromodiphenyl oxide meets the definition of "toxic" under the FHSA by virtue of its chronic organ system toxicity.

In a recent neurobehavioral developmental study (Viberg et al., 2003), the effects of decabromodiphenyl oxide on the developing central nervous system were investigated by evaluating the spontaneous motor behavior of adult mice exposed on postnatal days (PND) 3, 10, or 19. Changes in spontaneous behavior tests (locomotion, rearing, and total activity) were observed in 2-, 4-, and 6-month old mice dosed with 2.2 or 20.1 mg/kg body weight on PND 3, in contrast to mice exposed on PND 10 or 19. Twenty-four hours after dosing, about 5% of the ^{14}C-labeled compound was found in the brain. However, there are a number of limitations of the study (i.e., relatively small number of animals per treatment group; dosing done with a fat emulsion; behavioral tests conducted only once; evaluation of only one neurobehavioral endpoint; and the use of only one species) and the relevance of the results to human health is uncertain. At present, decabromodiphenyl oxide is considered a possible neurotoxicant in humans, based on limited evidence in animal studies. Staff will closely follow future developments relating to the neurobehavioral effects of decabromodiphenyl oxide.

Staff calculated an oral ADI of 3.2 mg/kg-day (Bittner, 2001), based on the liver effects observed in male mice in a 2-year chronic feeding study (NTP, 1986). The LOAEL of 3,200 mg/kg-day was divided by an uncertainty factor of 1000 (10 for interspecies variability, 10 for sensitive populations, and 10 for use of the LOAEL rather than the NOAEL).

Melamine

Under the FHSA, melamine is considered acutely toxic based on the oral LD_{50} of 3.2-3.8 g/kg in rats (Thomas and Brundage, 2004). There is no evidence of neurotoxic, reproductive, or developmental effects for melamine. Melamine is not mutagenic and the evidence for carcinogenicity is not sufficient, in staff's opinion, to satisfy the definition of "toxic" under the FHSA regulations. Melamine, in staff's opinion, does not meet the definition of "toxic" by virtue of its chronic toxicity under the FHSA, and thus, the calculation of an ADI is unnecessary at this time.

Vinylidene Chloride

Vinylidene chloride is acutely toxic as defined by the FHSA (Thomas and Brundage, 2004). Acute oral or inhalation exposure adversely affected the lung, kidney, and liver of experimental animals. There is also sufficient evidence of systemic toxicity caused by oral or inhalation exposure in experimental animals in subchronic and chronic studies. Vinylidene chloride may also be regarded as a possible developmental toxicant in humans, based on limited evidence of developmental toxicity in animals. As a possible developmental toxicant, it is not considered "toxic" as defined by the FHSA (Table 1.). In view of the limited evidence of carcinogenicity in animals, vinylidene chloride may be

regarded as a possible carcinogen in humans and is therefore not considered to be "toxic" under the FHSA based on its carcinogenicity. However, vinylidene chloride is considered "toxic" under the FHSA based upon the systemic toxicity from subchronic and chronic exposure.

For oral exposure, the subchronic NOAEL of 28.6 mg/kg-day in rats administered vinylidene chloride five times a week for 13 weeks (NTP, 1982) was used to estimate the ADI by using an uncertainty factor of 100 (10 for interspecies variability and 10 for sensitive populations). Thus, the subchronic oral ADI is 0.3 mg/kg-day[7]. This ADI is for the monomer of vinylidene chloride. Because vinylidene chloride in products is generally in the form of polyvinylidene chloride polymers, only the residual vinylidene chloride monomer will be considered in the risk analysis.

[7] Other organizations have chosen the Quast et al. (1983) study as the basis for their ADI, whereas CPSC staff based their ADI on the NTP study (1982). However, re-calculation of the ADI using the Quast et al. (1983) study would not affect the risk characterization as no vinylidene chloride monomer was extracted in detectable concentrations from the barriers in the aggressive migration studies (Bhooshan, 2005).

Table 2. Toxicity Summary of FR Chemicals

Chemical/Chemical Class	Acute Toxicity[a]	Chronic Toxicity[b]	Endpoint[c]	NOAEL/LOAEL (mg/kg-day)[d]	Uncertainty Factor	ADI (mg/kg-day)[e]
Ammonium Polyphosphates						ND
Antimony Trioxide						
Oral		T	O	230	100	2.3
Inhalation		T	C, O	0.009 (mg/m^3) L	1,000	$9x10^{-6}$ (mg/m^3)
Boric Acid	T	T	D, R, O	9.6	100	0.1
Zinc Borate	T					ND
Decabromodiphenyl Oxide		T	O	3,200 L	1,000	3.2
Melamine	T					ND
Vinylidene Chloride[f]	T	T	O	28.6	100	0.3

[a] Acute toxicity as defined in the FHSA regulations: T, toxic.
[b] Chronic toxicity as defined under the FHSA and the CPSC chronic hazard guidelines: T, toxic. Based on oral studies, except where indicated.
[c] Chronic toxicity endpoint(s): C, cancer; D, developmental; N, neurotoxicity; R, reproductive; O, other (e.g., liver toxicity).
[d] Doses are by the oral route in mg/kg-day, except where indicated. NOAEL, no-observed-adverse-effect-level; LOAEL, lowest-observed-adverse-effect-level, L.
[e] ADI, acceptable daily intake; ND, not determined.
[f] Monomer

3. Quantitative Risk Assessment

Based on the findings of the qualitative risk assessment previously conducted by CPSC staff which is available on the CPSC website (Thomas and Brundage, 2004), CPSC laboratory staff conducted migration/exposure assessment studies on FR-treated mattress barriers to obtain data to quantify the amount of FR chemical(s) that may be released from the barriers. Staff believes FR chemical-treated barriers are the most likely method that mattress manufacturers will use to meet the staff's draft final mattress flammability standard. The purpose of the laboratory studies was to provide a more accurate estimate of the potential risk associated with exposures to FR chemicals used in commercially-available FR-treated barriers that may be used by mattress manufacturers to meet the staff's draft final standard.

The quantitative risk assessment includes a consideration of dose response, bioavailability, and exposure. Quantitative exposure assessments may include estimates of average exposure, as well as the range of exposure or reasonable upper-bound exposures. Only chronic health effects are considered. Chronic health effects generally occur at lower levels than acute effects. Furthermore, most of the FR chemicals considered in this risk assessment had low levels of acute toxicity. The amount of FR chemical(s) released from the barriers is not expected to reach the levels necessary to cause acute effects.

This exposure assessment includes all applicable routes of exposure: dermal, oral, and inhalation. Staff evaluated potential exposure through all three routes combined, as well as individually. The exposure studies were conducted in three sequential phases. Phase 1 involved determining the total amount of FR chemical present in the barrier. Phase 2 consisted of migration tests to determine the potential migration of the FR chemical(s) in the barrier to the skin of the consumer. This provided staff with data to estimate dermal absorption, as well as to estimate the amount that may be ingested from the skin surface or mattress surface as a result of certain mouthing behaviors. Phase 3 measured the airborne particle-bound release of the FR chemical(s) from the barrier during normal use. This was an attempt to quantify the upper-bound concentration of the FR chemical released from the barrier into the indoor air using mechanical force to simulate the release of the FR chemical during normal use over 10 years. There were also limited aging studies to assess the effects of environmental factors, such as heat and humidity, on the release of airborne particle-bound FR chemicals.

For non-cancer effects, staff calculated the average daily dose (ADD) for each FR chemical using data from the exposure studies to determine whether the FR chemicals in the barriers would be expected to present a risk to consumers. The ADD is the estimated dose received due to a particular exposure scenario (i.e., dermal, oral, or inhalation). For antimony trioxide, which acts directly on the respiratory tract, the average daily exposure (ADE) was used to determine inhalation exposure, or average airborne concentration of antimony to which a person would be exposed. The risk associated with exposure to the FR chemicals, the hazard index (HI), was determined by summing the ADDs for all exposure routes and dividing by the oral ADI (Table 2). To

determine the HI for antimony trioxide inhalation exposure, the ADE was divided by the "inhalation ADI" for antimony. If the HI is greater than one, the exposure scenario under consideration is expected to present a risk to consumers.

Of the FR chemicals considered, only antimony trioxide is a probable carcinogen. To determine the cancer risk for the inhalation of antimony trioxide, which is a probable inhalation carcinogen in animals, staff calculated the lifetime average daily exposure (LADE) by the inhalation route. The lifetime individual excess cancer risk was then calculated by multiplying the LADE by the inhalation cancer potency for antimony.

The staff's studies and analysis applied conservative assumptions in areas of scientific uncertainty, that is, assumptions that tend to overestimate exposure and risk.

II. Methods

A. Experimental Protocol

The CPSC's LSC staff conducted a series of experiments using commercially-available barriers as obtained from the manufacturers (Bhooshan, 2005; Cobb, 2005). Appendix 2 provides an overview of the experimental methodology.

1. Phase 1: Barrier Sample Identification and Total FR Load

The total FR load was measured in the FR barriers using different methods, depending on the FR chemical(s) present. The total boric acid analytical load was measured by digesting the barrier in nitric acid and using inductively coupled plasma atomic emission spectroscopy (ICP). To measure the total antimony trioxide chemical load, antimony was extracted from the barrier with hydrochloric acid and analyzed by ICP. For decabromodiphenyl oxide and melamine, the chemicals were extracted with dioxane and hot deionized water, respectively, and analyzed using high pressure liquid chromatography (HPLC). The total vinylidene chloride chemical load was determined using gas chromatography mass spectrometry (GC-MS).

2. Phase 2: Migration

a) Aggressive Agitation Studies

The aggressive agitation studies, also referred to as the head-over-heels (HOH) analysis, determined the maximum amount of FR chemical that could be released due to aggressive mechanical agitation of the barrier in a saline solution. For each of the barriers, a circular piece of barrier measuring 5.5 centimeters (cm) in diameter was weighed and placed in a screw cap bottle with 25 milliliters (ml) of a solution of 0.9 percent sodium chloride. The bottle was rotated in a vertical circular motion at 60 rotations per minute (rpm) for 30 minutes, after which the solution was removed and saved for analysis. The process was repeated using the same barrier sample two additional times for a total of three times. For boron and antimony, each sample was analyzed by ICP. For melamine and decabromodiphenyl oxide, the solutions were analyzed using HPLC. Vinylidene chloride was measured using GC-MS. Four replicates were done for each barrier sample.

b) Aggressive Barrier Migration Studies

In a series of surface migration studies, staff estimated the quantity of FR chemical that might migrate to the skin from the FR barriers under certain use conditions. These tests measure the migration of the FR chemical(s) from the barriers to filter paper. For the purpose of this risk assessment, the amount transferred to the filter paper (i.e., surrogate skin) is considered to be the amount transferred to the skin from the barrier. All tests used a one pound per square inch (psi) stainless steel weight measuring 2 inches in diameter; this weight was used to simulate typical peak interface pressure of an adult lying prone on a mattress (Midgett, 2005; Shelton et al., 1998).

The initial tests of barriers measuring surface migration showed high amounts of FR chemicals released from five barriers. Three other barrier samples had FR chemicals comparable to the five barriers, but were found to have lower measurable releases of the FR chemicals. These three were not tested further. Additionally, three barriers containing only polyvinylidene chloride were not tested further as no vinylidene chloride monomer was extracted in detectable concentrations from the barriers in the two prior experiments (i.e., total FR load and HOH analysis). Two reagent extract solutions were used: simulated sweat and simulated urine. A circular piece of barrier with a diameter of 5.5 cm was placed in a 600 ml beaker. A 5.5 cm diameter piece of filter paper was placed on the barrier sample and 2 to 4 ml (Appendix 4) of one of the reagent extract solutions was poured onto the filter paper to thoroughly wet the barrier sample. After allowing the filter paper and barrier material to dry for 6 to 8 hours, the dry filter paper was analyzed for each FR chemical. The barrier sample in the beaker was covered with another filter paper and the process was repeated with the same reagent extract solution a total of four times. For boron, the filter papers were digested in nitric acid prior to analysis by ICP. To measure antimony, the antimony was extracted with hydrochloric acid and analyzed using ICP. Dioxane was used to extract the decabromodiphenyl oxide prior to analysis by HPLC. Five replicate tests were done for each barrier sample using each of the reagent extracts, with and without the one psi weight on the wetted filter paper and barrier sample.

c) Ticking/Sheet Migration Studies

Additional surface migration tests were conducted on two barrier samples containing both boric acid and antimony trioxide, under conditions which more closely represent the construction of a mattress with the FR barrier covered by ticking or ticking plus a sheet. Three different scenarios were tested. The procedure used for the additional tests was similar to the initial surface migration beaker tests with the following exceptions. In the first of three scenarios, a 5.5 cm diameter piece of ticking material was placed on top of the circular barrier sample in the beaker and a filter paper was placed on top of the ticking. The barrier, ticking and filter were wetted with 2 ml of simulated sweat. The second scenario was the same as scenario 1 except that the filter paper was not wetted with simulated sweat; only the barrier sample and the ticking (under the filter paper) were wetted. In scenario 3, the barrier sample was covered with ticking and standard sheeting[8]. The filter paper was placed on top and all three were wetted with 2 ml of

[8] Standard sheeting is white, 100 percent cotton material not treated with a chemical finish.

simulated sweat. The 1 psi weight was placed on top of the filter paper in all three scenarios. The filter papers were analyzed for boron using ICP as described above in initial surface migration tests. Antimony levels were not analyzed due to the low levels of antimony measured in the previous aggressive barrier migration studies. Five replicates were done for each scenario.

d) Mini-Mattress Migration Studies

Surface migration tests were also done using miniature mockup mattresses (mini-mattresses) representing a more realistic exposure scenario. The mini-mattresses consisted of a 9 inch by 9 inch by 0.5 inch piece of plywood covered with a 9 inch by 9 inch by 3 inch piece of non-FR chemical-treated foam (Appendix 3). The FR barrier, which was placed on the foam, was covered with ticking material and standard sheeting. The mini-mattresses were wetted with 25 ml of simulated sweat (Appendix 4). Two dry filter papers were placed on top of each wetted mini-mattress and covered with the one psi weights until the filter paper was dry (6 hours). Boron amounts were determined using ICP as described above.

e) Commercially-Available Mattress Migration Studies

Commercially-available full-scale twin mattresses containing a boric acid-treated barrier and an ammonium phosphate-treated barrier were subjected to conditions similar to those used in the mini-mattress migration studies. One section of the mattress was wetted with 25 ml of simulated sweat and another section was wetted with 25 ml of simulated urine. Two dry filter papers were placed on top of each wetted area and immediately covered with 1 psi weights. The weights were left in place for 6 hours on one of the two filter paper placed on the area wetted with simulated sweat and one on the area wetted with simulated urine. The weights were immediately removed from the other two filter papers once the filter papers were thoroughly wetted. The filter papers were analyzed for boron using ICP as described above. To measure ammonium polyphosphate, phosphate was extracted from the filter paper with nitric acid and analyzed using ICP.

3. Phase 3: Airborne Particle Release

a) Airborne Particle Studies

The next phase measured the simulated release of airborne particles from FR-treated barriers that might be expected during 10 years of use. Mini-mattresses, as previously described but with no sheet over the ticking, were subject to vertical impaction with a 4-inch diameter plastic convex head impactor for 100,000 cycles with an impact force of 3 psi. This impaction protocol is based in part on the ASTM testing method (ASTM F1566, part 9). Impaction, which occurred at a rate of one second per cycle, simulated the wear that would occur over the useful life of a mattress (i.e., 10 years of use). All tests were performed in an inflatable glove bag placed over a 13.5 inch by 20 inch by 27 inch frame. The glove bag was sealed during testing. Using four calibrated sampling pumps, 2 liters of recirculated air per minute were drawn through 5 micrometer (μm) polyvinyl chloride (PVC) filters, 0.8 μm cellulose filters, or glass fiber filters placed in cyclone samplers during the 100,000 cycles of impaction. The PVC and cellulose filters were digested using the procedures outlined in the U.S. Occupational Safety and Health

Administration (OSHA) method for Metals and Metalloid Particles in Workplace Atmospheres (ICP Analysis) and analyzed for boron and antimony using ICP. The PVC filters were analyzed for the respirable fraction of boron, whereas the cellulose filters were analyzed for the respirable particles of both boron and antimony. The glass fiber filters were analyzed for the respirable particles of decabromodiphenyl oxide, which were extracted with acetonitrile and analyzed using HPLC.

Particle-bound FR chemical release was also measured after the mini-mattress was wetted. One of the mini-mattresses was wetted with 100 ml of deionized water and allowed to dry prior to impaction and sampling. Another mini-mattress was tested without the ticking material covering the FR barrier.

To measure the release of non-respirable particles from the barrier that may settle onto horizontal surfaces, Circular Whatman ® #2 filters and Ghost™ Wipes were placed on the bottom of the glove bag near the mini-mattresses during the testing. For boron and antimony, the filters and wipes were digested and then analyzed using ICP. The decabromodiphenyl oxide was extracted with aceotnitrile and analyzed using HPLC.

b) Aging Studies

To establish the chemical stability and physical durability of the FR barriers, the mini-mattresses were subjected to simulated aging prior to impaction and sampling. Prior airborne sampling was conducted at room temperature (approximately 27 degrees Celsius) and average humidity (approximately 60 percent). Aging was accomplished by subjecting the mini-mattresses to high heat and humidity. Several different sources were consulted in the development of the aging test conditions (AATCC, 2001; ASTM, 2004; Feller, 1994). Mini-mattresses were exposed to 85 percent humidity at a temperature of 90 degrees Celsius for 96 hours. Subsequent to the simulated aging, the mini-mattresses were conditioned to room temperature and average humidity for 24 hours and then subjected to impaction and airborne sampling. The impaction and airborne sampling conditions were the same as described above.

B. Exposure Models

1. Dermal Exposure

For the purpose of estimating dermal exposure, it is assumed that an external liquid phase facilitates the transfer of the FR chemical from the barrier to the skin (Babich and Thomas, 2001; NRC, 2000). The types of liquid facilitating the transfer while sleeping on mattresses for 8 hours a day are (1) sweat and (2) urine. The average daily dose (ADD) from dermal exposure is calculated by:

$$ADD_D = \frac{L_D \cdot A_{S.1} \cdot k_T \cdot T \cdot N}{W} \quad (1.1)$$

where: ADD_D, average daily dose from sweat-mediated dermal exposure, mg/kg-d; L_D, dermal FR load, mg/cm^2; $A_{S.1}$, skin surface area exposed, cm^2; k_T, percutaneous absorption rate, h^{-1}; T, exposure duration, h; N, number of exposures per day, d^{-1}; and W, body weight, kg.

Certain dermal exposure scenarios, such as a child urinating in bed, will occur intermittently. To determine the chronic health effects due to intermittent exposures over longer time periods, for non-cancer effects, the ADD may be adjusted as follows:

$$ADD_{D.TW} = \frac{L_D \cdot A_{S.2} \cdot k_T \cdot T \cdot N \cdot N_A}{W \cdot T_A} \quad (1.2)$$

where: $ADD_{D.TW}$, time-weighted average daily dose from urine-mediated dermal exposure, mg/kg-d; L_D, dermal FR load, mg/cm^2; $A_{S.2}$, skin surface area exposed to the urine, cm^2; k_T, percutaneous absorption rate, h^{-1}; T, exposure duration, h; N, number of exposures per day, d^{-1}; N_A, the number of days that the exposure takes place during the averaging period, d; W, body weight, kg; and T_A, averaging period, d.

2. Oral Exposure

Oral exposure is expected to result from a combination of hand-to-mouth behaviors, licking of the lips during the night, and mouthing of the sheets and/or mattress (children only). The amount of FR chemical measured from the surface migration studies is considered the amount available for ingestion. The ADD from oral exposure is calculated by:

$$ADD_O = \frac{L_D \cdot Am \cdot E \cdot N}{W} \quad (1.3)$$

where: ADD_O, average daily dose from oral exposure, mg/kg-d; L_D, dermal FR load, mg/cm^2, as measured by the surface migration tests (Cobb, 2005); Am, mouthing area, cm^2; E, extraction efficiency, unitless; N, number of exposures per day, d^{-1}; and W, body weight, kg.

3. Inhalation Exposure

It is assumed that inhalation exposure occurs through the inhalation of respirable particle-bound FR chemicals that may be released into the indoor air as the barrier is worn or ages. A simple one-zone mass balance model may be used to predict the concentration of FR chemicals in indoor air (NRC, 1981). The ADD from inhalation exposure to particle-bound FR chemicals is calculated by:

$$ADD_I = \frac{C \cdot I \cdot T \cdot N \cdot B}{W} \tag{1.4}$$

where: ADD_I, average daily dose from inhalation exposure, mg/kg-d; C, airborne particle-bound FR concentration, mg/m^3; I, average inhalation rate, m^3/h; T, exposure duration, h; N, number of exposures per day, d^{-1}; B, bioavailability, unitless; and W, body weight, kg.

The concentration of airborne particle-bound FR chemical is calculated as follows:

$$C = \frac{S_n}{V \cdot (ACH + K)} \tag{1.5}$$

where: C, airborne particle-bound FR concentration, mg/m^3; S_n, source strength of particle-bound FR chemical, mg/h; V, breathing zone, m^3; ACH, air infiltration rate, h^{-1}; and K, particle deposition rate, h^{-1}.

The total mass released from the mini-mattress is assumed to be released at a uniform rate over the life of the mattress (10 years[9]). The source strength for respirable particle-bound FR chemicals is calculated by:

$$S_1 = \frac{M \cdot 35}{8 \cdot 365 \cdot 10} \tag{1.6}$$

where: S_1, source strength, mg/h; M, mass of respirable FR released from the mini-mattress, mg; 35, factor to convert the surface area of the mini-mattress to a commercially-available twin mattress, unitless; 8, the number of hours of use per day, h/d; 365, the number of days per year, d/y; and 10, the number of years of exposure, y.

[9] The useful lifetime of a mattress as reported by industry.

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

$$ADE = \frac{C \cdot T}{24} \tag{1.7}$$

where: ADE, time-weighted average daily exposure, mg/m^3; C, airborne particle-bound FR concentration, mg/m^3; T, exposure duration, h; and 24, number of hours, h.

C. Risk Equations

1. Non-Cancer Endpoints

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

$$HI = \frac{ADD_D + ADD_O + ADD_I + ADD_{D.TW}*}{ADI} \tag{1.8}$$

where: HI, hazard index, unitless; ADD_D, average daily dose from sweat-mediated dermal exposure, mg/kg-d; ADD_O, average daily dose from oral exposure, mg/kg-d; ADD_I, average daily dose from inhalation exposure, mg/kg-d; $ADD_{D.TW}$, time-weighted average daily dose from urine-mediated dermal exposure (*for children only), mg/kg-d; and ADI, acceptable daily intake, mg/kg-d..

For antimony trioxide, which acts directly on the respiratory tract, exposure is expressed as the average airborne concentration (mg/m^3), rather than the average daily dose (mg/kg-d). Therefore, the HI for antimony trioxide is calculated by:

$$HI_{I.AT} = \frac{ADE}{ADI_I} \tag{1.9}$$

where: $HI_{I.AT}$, hazard index for antimony trioxide inhalation exposure, unitless; ADE, time-weighted average daily exposure, mg/m^3; and ADI_I, "inhalation ADI" for airborne antimony trioxide particles, mg/m^3.

2. Cancer Endpoints

In the case of antimony trioxide, in which the cancer risk is based on the airborne concentration, the lifetime average daily exposure (LADE) by the inhalation route is calculated by:

$$LADE_I = \frac{ADE \cdot N_Y \cdot Y}{365.25 \cdot Y_E} \quad (1.10)$$

where: $LADE_I$, lifetime average daily exposure by inhalation, mg/m^3; ADE, average daily exposure, mg/m^3; N_Y, number of days per year that the product is used, d/y; Y, number of years of product exposure, y; 365.25, number of days per year, d/y; Y_E, average life expectancy, y.

Then, the lifetime individual excess cancer risk is:

$$R_I = Q_I \cdot LADE_I \quad (1.11)$$

where: R_I, lifetime individual excess cancer risk; Q_I, unit cancer risk, or cancer potency, by the inhalation route, $(mg/m^3)^{-1}$; and $LADE_I$, lifetime average daily inhalation exposure, mg/m^3.

D. Input Parameters

1. General Parameters

General parameters are those that are applicable to multiple exposure scenarios. The average lifetime of a mattress is estimated to be 10 years[10] (Midgett, 2005). The average life expectancy of a person is 75 years (EPA, 1997a). Staff estimates a person is exposed to a FR-treated mattress for 70 years, which was derived by subtracting five years from the average life expectancy. This assumes children under the age of five sleep on mattresses protected with vinyl or plastic covers (Midgett, 2005), which would be expected to reduce FR chemical exposure to negligible levels during the first five years of life. The body weight for adults (45-54 years old) is 72.25 kg. For 5 year old children, the body weight is 19.2 kg. The body weight is the average of males and females in the 50th percentile for both adults and children (EPA, 1997a).

[10] The ASTM F1566 (part 9) method, on which CPSC staff based their physical impaction protocol, is assumed to approximate the typical use of a mattress during 10 years. Therefore, HS staff chose to use the conservative estimate of 10 years for the expected average lifetime of a mattress.

Table 4. General Parameters

Parameter			Value	Source
Y_F	Mattress lifetime	Y	10	Midgett, 2005
Y_E	Life expectancy	Y	75	EPA, 1997a
Y	Years of product exposure	Y	70	HS staff estimation
W	Body weight (50^{th} percentile)	Kg	72.25 (adult)	EPA, 1997a
			19.2 (child)	EPA, 1997a

2. Dermal Exposure Parameters

Dermal exposure involves direct contact of the skin with the mattress surface. The estimated average skin surface area in contact with the mattress is based on the assumption that an unclothed child or adult lying on a mattress will toss and turn in bed during sleep, potentially exposing almost the entire surface area of the body to the mattress surface. The skin surface area exposed is estimated to be 1.82 m^2 (18,200 cm^2) for adults and 0.79 m^2 (7,900 cm^2) for 5 year old children. For both adults and children, this is an average of males and females in the 50^{th} percentile (EPA, 1997a). Exposure duration is estimated to be 8 hours a day for an adult and 11 hours a day for a 5 year old child (Midgett, 2005).

The dermal FR chemical load is the amount of FR chemical on the surface of the skin. This amount, which is determined from the average concentration of the FR chemical measured in the filter paper (i.e., surrogate skin) from the surface migration studies, is the amount of FR chemical that is available for transfer to the skin per unit area of the mattress surface.

The estimated percutaneous absorption rates for antimony trioxide, boric acid, and decabromodiphenyl oxide are listed in Table 8. In calculating absorption rates, the absorption was assumed to be linear with time from the percentage of the applied dose absorbed at the given time, typically 24 hours. In an *in vivo* study, the percentage of boric acid absorbed following a 24 hour exposure was 0.226 (Wester et al., 1998). The fraction absorbed is 0.00226 per day, or 9.4×10^{-5} per hour. For decabromodiphenyl oxide, absorption is highly dose dependent. Two percent absorption was observed when 60 nanomoles (nmol) (90 $\mu g/cm^2$) was applied to hairless mouse skin, 3 percent at 30 nmol (45 $\mu g/cm^2$), and 20 percent at 6 nmol (9 $\mu g/cm^2$) in an *in vitro* study (Hughes, 2000). Thus, when less than or equal to 9 $\mu g/cm^2$ of decabromodiphenyl oxide is in contact with the skin, a value of 20 percent absorption may be applied. This is equivalent to a percutaneous absorption rate of 0.01 per hour. When greater than 9 $\mu g/cm^2$ is in contact with the skin, a three percent absorption may be applied, which is equivalent to 0.001 per hour. Percutaneous absorption data were unavailable for antimony trioxide. Inorganic compounds, such as antimony trioxide, are generally absorbed at low but detectable rates (reviewed in EPA, 1992; Hughes et al., 1995; Rahman and Hughes, 1994; Rahman et al., 1994). Therefore, as in the previous risk assessment on FR chemicals in upholstered furniture (Babich and Thomas, 2001), it will be assumed that five percent of antimony trioxide is absorbed in 24 hours. The fraction absorbed is 0.05 per day, or 0.002 per hour.

Certain dermal exposure scenarios, such as a child urinating in bed, are expected to occur intermittently. Regular bed wetting would typically lead caregivers to consistently use mattress covers and/or diapers. In the case of unanticipated bed wetting, the estimated frequency of bed wetting is twice per month (30 days) for a 5 year old (Midgett, 2005). The above frequency is considered to be conservative. The estimated skin surface area exposed to the urine is approximately 13 percent of the total skin surface of a 5 year old child, or 1,027 cm^2 (Midgett, 2005).

Table 5. Dermal Exposure Parameters

Parameter			Value	Source
$A_{S.1}$	Skin surface area exposed (sweat)	cm^2	18,200 (adult)	EPA, 1997a
			7,900 (child)	EPA, 1997a
T	Exposure duration	H	8 (adult)	Midgett, 2005
			11 (child)	Midgett, 2005
N	Number of exposures per day	d^{-1}	1	HS staff estimation
N_A	Number of days exposure (urinating) takes place during the averaging period	D	2	Midgett, 2005
T_A	Average period	D	30	Midgett, 2005
$A_{S.2}$	Skin surface area exposed (urine)	cm^2	1027	Midgett, 2005

3. Oral Exposure Parameters

The average concentration of the FR chemical measured in the filter paper (i.e., surrogate skin) from the surface migration studies is used to estimate the amount of FR chemical ingested. The values from the surface migration studies are used as the surface FR load to calculate the ADD from oral exposure. In adults, the amount of FR chemical present on the skin surface which is transferred from the mattress surface (i.e., the dermal FR load) is considered the quantity available for ingestion. CPSC's Human Factors (HF) staff determined that mouthing of the sheets and mattress in adults is not a significant source of exposure (Midgett, 2005). However, in children who are 5 to 15 years old, CPSC staff estimates that oral exposure to FR chemicals occurs through mouthing of the skin, as well as mouthing of the sheets and mattress.

Oral exposure is expected to occur as the result of a combination of activities involving hand-to-mouth behavior and licking of the lips during the night. For example, hand-to-mouth exposures could presumably take place while eating in bed, or eating before washing in the morning. Moistening of the lips during the night is also considered a source of exposure. For an adult, staff estimates a mouthing area of 6 cm^2 which would comprise an area of 2 cm^2 from each hand and two moistening of the lips during the night (approximately 1 cm^2 each) (Midgett, 2005); mouthing of the sheets or mattress is considered a relatively insignificant exposure route. HF staff estimates a 5 year old child

could ingest the quantity of particles adhering to 8 cm^2 of skin, assuming all the FR chemical comes off (Midgett, 2005). This includes an area of 3 cm^2 from each hand and two moistening of the lips during the night (approximately 1 cm^2 each). CPSC staff also recognizes that mouthing of the sheets and mattress may occur in children 5 to 15 years old. For the mouthing of the sheets and mattress, staff estimates a mouthing area of 5 cm^2. The National Academy of Sciences' National Research Council (NRC) risk assessment of FR chemicals in upholstered furniture (NRC, 2000) defined a mouthing area of 50 cm^2 for a 10 kg child. However, due to the different use pattern of a mattress and the assumption by NRC scientists that the actual oral exposure could be "100-fold less" than the defined mouthing parameter of 50 cm^2, CPSC staff modified the estimated mouthing area for the purpose of this risk assessment. Assuming that the 50 cm^2 was 100-fold more than the actual expected mouthing area, the exposure area would be about 0.5 cm^2. Increasing this value by a factor of 10 to be conservative yields a mouthing area of 5 cm^2. Therefore, for children 5 to 15 years old, CPSC staff estimates a total mouthing area of 13 cm^2, which includes mouthing an area of 3 cm^2 from each hand and two moistening of the lips during the night (approximately 1 cm^2 each), as well as the mouthing of 5 cm^2 of the sheets and mattress. The mouthing area for adults and children is presented as the integration of a number of activities expected to occur once per day.

To be conservative, the FR chemical extraction efficiency, or transfer efficiency, of the FR chemical on the skin, and surface of the sheet and mattress into the mouth for ingestion is assumed to be 100 percent for the purpose of this risk assessment. That is, all of the FR chemical present on the surface of the skin, and the sheets and mattress, which is mouthed, is expected to be ingested.

Table 6. Oral Exposure Parameters

Parameter			Value	Source
Am	Mouthing area	cm^2	6 (adult)	Midgett, 2005
			13 (child)	Midgett, 2005
E	Extraction efficiency	unitless	1	HS staff estimation
N	Number of exposures per day	d^{-1}	1	Midgett, 2005

4. Inhalation Exposure Parameters

The mass of the FR chemical, which is used to calculate the source strength for antimony trioxide, boric acid and decabromodiphenyl oxide, is the total amount of FR chemical collected during 100,000 cycles of impaction on four filters. For barriers containing antimony trioxide and/or boric acid, two PVC filters and two cellulose filters were used. The PVC filters were analyzed for the respirable fraction of boric acid, whereas the cellulose filters were analyzed for the respirable particles of both boric acid and antimony trioxide. The cellulose filters were used to measure both FR chemicals because they are readily dissolved in strong acid which aids the specific analysis of the metals. Both the PVC and cellulose filters capture essentially all airborne particles of interest relative to human exposure potential; the capture efficiency is not limited to the pore size.

The mass of the respirable FR chemical released from a commercially-available twin mattress with a surface area of 2,831 square inches is estimated to be 35 times greater than that released from the mini-mattress with a surface area of 81 square inches. Thus, the total mass of the respirable FR particles collected is multiplied by 35 (assumes direct scaling).

For the purpose of this risk assessment, 100 percent of the airborne particles inhaled are assumed to be absorbed by the lung. The mean inhalation rate for adults is 0.6 m^3 per hour (EPA, 1997a). For children, the mean inhalation rate is 0.4 m^3 per hour (EPA, 1997a). The inhalation rate for both children and adults is for sedentary activities. The exposure duration represents the average time spent sleeping on the mattress per day. Exposure duration is estimated to be 8 hours for adults and 11 hours for children (Howard and Wong, 2001; Midgett, 2005). The number of exposures per day is assumed to be one.

The volume of the breathing zone of an individual lying on a mattress is approximately 1.85 m^3. This volume of air comprises one meter above the surface area of full-scale twin mattress. The indoor air change of 0.5 per hour is the median value for all seasons and all regions in the U.S. (Koontz and Rector, 1993). The particle deposition rate of 0.5 per hour is a published value for particles 1 to 5 μm in diameter (EPA, 1997b).

Table 7. Inhalation Parameters

Parameter			Value	Source
I	Average inhalation rate	m^3/h	0.6 (adult)	EPA, 1997a
			0.4 (child)	EPA, 1997a
T	Exposure duration	H	8 (adult)	Midgett, 2005
			11 (child)	Howard and Wong, 2001
N	Number of exposures per day	d^{-1}	1	HS staff estimation
B	Bioavailability	unitless	1	HS staff estimation
V	Breathing zone	m^3	1.85	HS staff estimation
ACH	Air change	h^{-1}	0.5	Kootz and Rector, 1993
K	Particle deposition rate	h^{-1}	0.5	EPA, 1997b

5. Risk Parameters

Acceptable daily intake values were calculated from the NOAEL or LOAEL of available oral toxicity data using an uncertainty factor approach (CPSC, 1992). Similarly, an "inhalation ADI" was calculated for antimony trioxide based on the adverse lung effects in rats after chronic inhalation of antimony trioxide (Newton et al., 1994).

A cancer potency estimate, or unit cancer risk, was calculated using the default methods outlined in the CPSC chronic hazard guidelines (CPSC, 1992). CPSC uses the maximum likelihood estimate of cancer risk, provided that the dose response is linear at low doses,

to calculate the unit cancer risk. Of the FR chemicals considered, only antimony trioxide is considered a probable carcinogen. Cancer estimates were only made for inhalation exposure to airborne antimony trioxide particles, which caused tumors only at the site of exposure (lung) in rats (reviewed in Hatlelid, 1999a). For calculating the cancer risk for antimony trioxide, the cancer risk for adults and children represents the risk from a cumulative exposure to a FR-treated mattress of 70 years (Table 4). Previously staff calculated an inhalation cancer potency for antimony trioxide of 0.51 $(mg/m^3)^{-1}$ (Babich and Thomas, 2001).

Table 8. Risk and Toxicological Parameters

Parameter			Antimony	Boric Acid (Boron)	DBDPO	Vinylidene Chloride
ADI	Acceptable daily intake	mg/kg-d	2.3	0.1	3.2	0.3
ADI_I	Inhalation ADI	mg/m^3	$9x10^{-6}$	NA	NA	NA
Q_I	Inhalation cancer potency	$(mg/m^3)^{-1}$	0.51	NA	NA	NA
k_T	Percutaneous absorption rate	h^{-1}	0.002	$9x10^{-5}$	0.01 or 0.001	NA

DBDPO = Decabromodiphenyl Oxide
NA = not applicable

6. Upper Bound Exposure Parameters

Upper bound, or worst-case, exposure parameters are used to estimate the possible maximum exposure to consumers (Appendix 5). In the 95th percentile, the body weight for adults (45-54 years old) is 100.7 kg and 26 kg for 5 year old children. The body weight is the average of males and females for both adults and children (EPA, 1997a). For estimating maximal dermal exposure, the skin surface is estimated to be 2.19 m^2 (21,900 cm^2) for adults and 0.935 m^2 (9,350 cm^2) for 5 year old children. For both adults and children, this is an average of males and females in the 95th percentile (EPA, 1997a). To estimate the upper bound exposure due to bed wetting (an intermittent exposure), the estimated skin surface area exposed to the urine is approximately 13 percent of the total skin surface of a 5 year old child in the 95th percentile, or 1,215.5 cm^2 (Midgett, 2005).

To estimate upper bound oral exposure to FR chemicals, staff applied an additional 5-fold factor to the 13 cm^2 mouthing area estimated for children increasing the mouthing area to 65 cm^2. For adults, the mouthing area was also increased by a 5-fold factor giving a total mouthing area of 30 cm^2 to estimate possible maximal oral exposure.

Table 9. Upper Bound Exposure Parameters

Parameter			Value	Source
W	Body weight (95th percentile)	Kg	100.7 (adult)	EPA, 1997a
			26 (child)	EPA, 1997a
$A_{S.1}$	Skin surface area exposed (sweat, 95th percentile)	cm^2	21,900 (adult)	EPA, 1997a
			9,350 (child)	EPA, 1997a
$A_{S.2}$	Skin surface area exposed (urine, 95th percentile)	cm^2	1,215.5	Midgett, 2005
Am	Mouthing area	cm^2	30 (adult)	HS staff estimation
			65 (child)	HS staff estimation

III. Exposure and Risk Assessment

A. Exposure Assessment

1. FR Chemical Migration Studies

A series of migration studies conducted by the CPSC laboratory, were designed to estimate the amount of FR chemical that may be released from the mattress barrier under consumer use conditions. The first stage of this testing series used the HOH apparatus, which was used as a worst-case test, to estimate the relative durability of the FR chemical in the barrier (Bhooshan, 2005; Cobb, 2005). Of the FR barriers tested, those incorporating melamine resin or VC as a polymer did not release detectable amounts of FR. As a result, additional migration tests were not carried out with these samples. By contrast, the releases of boric acid and non-resin (e.g., barrier was surface coated) melamine were high, with the total amount of FR chemical migrating out of the barriers during the test.

Dermal and Oral Exposure Tests

A series of tests was conducted that estimate the amount of FR chemical that is released from the mattress barrier and is available for dermal absorption and oral ingestion. These experiments were conducted in phases starting with the more aggressive HOH test, followed by a series of dermal exposure tests that at each stage were considered more indicative of actual consumer exposure/use scenarios. The first dermal exposure test involved placing a surrogate skin (filter paper) directly on the barrier sample. Subsequent dermal tests involved covering the barrier sample with ticking and sheet samples.

A detailed description of the testing methodology can be found in another CPSC staff report (Cobb, 2005). In these tests, the CPSC laboratory staff cut round portions of barrier, ticking, and sheeting materials and placed them in a beaker. Approximately 2 ml of either simulated urine or sweat was poured on top of the surrogate skin. A weight representing 1 psi was placed on top of some surrogate skin samples whiles others were unweighted (Cobb et al., 2005).

The first and most conservative series of tests involved placing the surrogate skin directly on the FR-treated barrier. The average concentration in the surrogate skin was determined without pressure and when 1 psi was applied to the surrogate skin. For all of the dermal

migration tests conducted in the beaker, a series of four extractions was completed on the same barrier sample. Each extraction in the series was completed over approximately 8 hours. Since the estimated exposure time is 8 hours for an adult and 11 hours for a child in a bed (Midgett, 2005), the amount in each extraction was used in the risk assessment to estimate the amount of FR chemical that would migrate out of a barrier to the surface in one sleeping episode. The amounts of antimony and decabromodiphenyl oxide (DBDPO) released from these tests were low, so no additional dermal migration testing was performed for these compounds (Tables 10 and 11). Because of the relatively high releases of boric acid, a second series of dermal migration tests were completed for samples containing this FR chemical.

The second series of tests conducted in a beaker for boric acid treated barriers involved placing the surrogate skin on top of a barrier sample that was covered with a 1) standard polyethylene ticking, or 2) with the standard ticking and a standard sheet. The test setup was wetted with simulated sweat as in the previous tests. The results of this test suggested that when the standard ticking was placed over the barrier, the amount of boric acid absorbed by the surrogate skin was reduced. When a standard sheet and ticking were placed between the barrier and the surrogate skin, the amount of boric acid absorbed by the surrogate skin was reduced. These results suggest that the ticking and sheets may act as barriers to reduce FR chemical migration to the skin surface.

A series of dermal boric acid migration tests were performed on 9 x 9 inch mini-mattress constructed by CPSC Laboratory Sciences staff. A detailed description of this mattress can be found in the accompanying laboratory report (Cobb, 2005). The mini-mattresses were covered with the same ticking and sheet used in the previously described dermal tests. The results of the dermal migration tests performed on the mini-mattress with boric acid-treated barriers showed that lower amounts of boric acid migrated into the surrogate skin compared to migration results from sampling that occurred in the previous tests.

An explanation for these results could be that the moisture applied to the mini-mattress was absorbed within and across the surface of the mini-mattress, resulting in the amount of moisture per unit area of surface being less than that found in the previous experiments where the barrier, ticking and sheet were placed in a beaker and the moisture in the beaker was contained in a smaller area. The containment of the moisture is believed to have resulted in more moisture per unit area, which is a critical factor in the migration of boric acid out of the treated barrier. Also, any FR chemicals extracted by the moisture could migrate into the internal (foam) portion of the mini-mattress, whereas the migration in the beaker experiments was constrained to go to the filter paper.

A final series of tests were conducted on a commercially available twin mattress containing a boric acid-treated barrier. Sections of the mattress were wetted with 25 ml of simulated sweat and another section was wetted with 25 ml of simulated urine. Two dry filter papers were placed on top of each wetted area and immediately covered with 1 psi weights. The weights were left for 6 hours on one of the two filter paper placed on the area wetted with simulated perspiration and one on the area wetted with simulated urine. The amount of boric acid migration from the full-scale mattress was higher than

the mini-mattress, but considerably lower than migration amounts observed in the beaker experiments.

Staff recently became aware of the use of ammonium polyphosphate barriers in mattresses. Therefore, CPSC laboratory staff also measured the migration of ammonium polyphosphate from a commercially available twin mattress containing an ammoinium polyphosphate barrier, as described above. Although a substanial amount of ammonium polyphosphate was released from the barrier, ammonium polyphosphate is not expected to result in any health effects in consumers because it is not considered "toxic" under the FSHA.

In migration tests where samples are placed in beakers and wetted, the amount of FR chemical migration was higher compared to the full-scale and mini-mattresses where there was believed to be less moisture per unit area. The amount of moisture applied to the mini- and full-scale twin mattresses is believed to be slightly excessive compared to what may be expected in a typical consumer sleep scenario (Appendix 4). However, the excess moisture applied to the barrier samples does account for situations where individuals will typically experience elevated sweat production, such as during febrile illness, sexual activity, perimenopause, and in high temperature, high humidity climates where cooling devices are not available.

When there was minimal migration of certain FR chemicals (antimony and DBDPO) in the aggressive tests, additional testing was not performed (Appendix 2). If more than minimal migration of an FR chemical was observed in the early tests, additional testing representing more realistic dermal exposure scenarios in mattresses was conducted. These results were then used in the risk models to estimate the potential health risk that may result from these dermal and oral FR chemical exposures.

Inhalation Tests

The inhalation of FR chemicals that are released to the surface of the mattresses could be a route of exposure in some scenarios. Consumer use scenarios including forceful play by children on the bed and other activities that occur prior to, or during actual sleep, may agitate the mattress, resulting in releases of FR chemical to the surface. In order to estimate the amount of FR chemicals released into the air, CPSC Directorate of Laboratory Sciences, Division of Mechanical Engineering staff developed a device that subjected mini-mattresses to physical abuse. The impaction device design was based, in part, on the impactor described in the ASTM F1566 (part 9) and is described in the laboratory memorandum by Cobb, 2005 and in an earlier section of this memo. The impaction device subjects the mini-mattress to approximately 3 psi of vertical pressure for 100,000 cycles. The ASTM F1566 method was interpreted by CPSC staff to suggest that this amount of physical impaction serves as a rough approximation of the amount of stress that would occur during 10 years of mattress use.

LSC staff used the impaction device to physically stress artificially aged and unaged mini-mattresses in an enclosed chamber. The 100,000 cycle impaction was completed in 28 hours. The total amount of respirable FR chemical released during the impaction

period was extrapolated over 10 years of mattress use to estimate the average daily exposure to respirable airborne FR chemical particulates.

2. FR Chemicals with Limited Migration Data

Melamine

The toxicity potential of melamine was reviewed in a previous CPSC staff memo (Thomas and Brundage, 2004). Melamine was not considered to meet the definition of toxic under the FHSA. Under the FHSA, a product must be considered toxic and consumers must be exposed to the toxic substance in sufficient quantities for there to be adverse health effects. Because of the lack of toxicity, extensive evaluation of the potential exposure and health risks of melamine used in barriers was not needed. However, CPSC LSC staff did conduct initial migration experiments on two types of melamine barriers. The barrier described in the previous CPSC staff qualitative exposure and risk memorandum, incorporated melamine into the barrier as a resin. The melamine resin in the barrier tested appears to be durable, and did not release detectable quantities of melamine during testing (Cobb, 2005). Barriers that did not contain melamine in resin form released significant amounts of melamine during the rigorous head-over-heels (HOH) test. However, the HOH test is considered an extreme extraction, and estimates migration from materials as a result of direct mouthing (e.g., chewing) of objects which is not expected to occur with barriers in mattresses. Since melamine is not considered chronically toxic, significant releases from mattresses would not be expected to result in unacceptable risks of chronic health effects in humans.

Vinylidene Chloride

Vinylidene chloride is the name of the monomer that is incorporated into mattress barriers as a polymer (polyvinylidene chloride (PVDC)). In general, polymeric materials are not expected to be absorbed into the human body, and are not considered to pose significant health hazards to humans (polymers are generally not expected to release significant quantities of monomer that can be absorbed into the human body). The LSC staff subjected barriers containing vinylidene chloride polymer to an aggressive extraction procedure (HOH test). This experimental procedure was used to determine FR chemical migration from upholstered furniture fabrics (Babich and Thomas, 2001). Staff determined that the HOH test may be used as an extreme extraction to compare the relative losses of FR chemicals between various barriers and FR treatments.

The HOH experimental procedures used on mattress barrier samples are described in another CPSC staff memorandum (Bhooshan, 2005). The results of the testing show that detectable quantities of VC were not released from the barriers containing PVDC. These data confirm the supposition that minimal amounts of VC would be released from the polymeric form of the chemical as it exists in the barrier fabric. Since detectable quantities of VC were not released during the more rigorous HOH test, it was considered unlikely that measurable quantities of VC would be released during any of the other, less intense CPSC Laboratory Sciences FR chemical migration tests.

3. Extensive Migration Testing of FR Chemicals

Barriers containing antimony trioxide (antimony), boric acid, and decabromodiphenyl oxide (DBDPO) were tested to estimate the potential release of FR chemicals during consumer use of mattresses containing barriers treated with these chemicals. Inhalation and dermal tests were conducted by LSC on barriers containing antimony, boric acid, and DBDPO including impaction of mini-mattresses that were subjected to the aging procedure (Cobb, 2005).

a) Antimony Trioxide

Dermal Absorption and Oral Ingestion

Migration tests were conducted on samples from two separate barriers (9 and 11) that contained antimony (Table 10; Cobb, 2005). These tests were conducted using both simulated urine and simulated sweat to wet the barrier and surrogate skin (i.e., filter paper) which was in direct contact with the barrier. This test is the most conservative because there is no material (e.g., sheet, ticking) between the barrier and the surrogate skin. Subsequent tests with boric acid treated barriers suggest that the ticking and sheet will reduce the amount of FR chemical that will migrate into the surrogate skin. The results from barrier 11 were used in the risk calculation for antimony because they presented the highest migration rates. The average concentration of antimony released from the barrier when subjected to wetting with simulated sweat was 2.7 $\mu g/cm^2$ (Table 10). This result represents the amount of antimony that is expected to migrate from a barrier to the surface and adsorb to the surface of the skin. Thus, the concentration of antimony on the skin is conservatively estimated to be 2.7 $\mu g/cm^2$.

Urine-Mediated Exposure

The tests for urine-mediated dermal exposure were conducted along with the simulated sweat for barrier samples 11 and 9. The amount of antimony migrating from barrier 11 when urine was applied was also higher than barrier 9, thus these data from barrier 11 were used to estimate antimony concentrations on skin after bed wetting. The average amount of antimony migrating out of barrier 11 wetted with simulated urine over an 8 hour time period was 2.6 $\mu g/cm^2$.

Inhalation

Aged and unaged mini-mattresses containing barrier 9 were subjected to the impaction tests to estimate the amount of antimony that would be released during the estimated 10 year lifetime of the mattress. Samples were collected from the impaction chamber on two cyclone samplers with cellulose acetate filters collecting respirable particles. The amount of antimony on all of the filters from aged mini-mattresses 4 and 5 was below the method detection limit (0.3 μg). One-half of the method detection limit (MDL) was used to estimate the amount of antimony on each filter. Since two filters were used to sample the total amount of antimony released from each aged mockup, it was assumed that the total amount of antimony release was 0.3 μg (aged only). This value was extrapolated to a full-sized twin mattress (35x) and corrected for non-respirable particles (20x) resulting in a total of 210 μg release from the mattress during the 10 year lifetime of the mattress.

b) Boric Acid

Dermal Absorption and Oral Ingestion

The amount of boric acid that migrated into the surrogate skin declined from the first and most conservative series of tests involving placing the surrogate skin directly on the FR-treated barrier, to the final test where a standard ticking and sheet were placed over the barrier. The results of the dermal absorption tests using a full scale twin mattress for boric acid are summarized in Table 12b. The results of these tests were slightly higher but comparable to the migration from mini-mattresses (Table 12; Cobb 2005). Another important result is the impact of weight on the amount of boric acid migration from barriers. When tests were conducted comparing the migration from barriers with no weight applied to those with 1 psi applied to the surface, migration was higher when weight was applied than when it was not (Cobb, 2005). These results suggest that the pressure applied to the barrier/ticking/sheet matrix will significantly impact the amount of boric acid migration to the surface. It is likely that the pressure increases the amount of liquid picked up by the surrogate skin and subsequently the boric acid contained in the liquid medium.

A third set of tests compared FR migration from the ticking covered barrier when surrogate skin was either wet or dry. The dry surrogate skin appeared to absorb more FR chemical compared to a wetted surrogate skin on top of a barrier ticking. The amount of boric acid leaching out of the barriers was lower on the mini- and twin mattresses compared to the beaker experiments. This is believed to be due, in part, to a lower amount of available moisture per square centimeter, and in part to the presence of other materials to absorb any migrating chemicals. Consequently, it is considered more representative of the amount of moisture that would be seen during consumer use (Appendix 4). The filter paper is expected to absorb more FR chemical than actual human skin. The amount of available boric acid during the sleep period is 23.4 $\mu g/cm^2$ (equivalent to 4.1 $\mu g/cm^2$ of boron) on the bedding and skin surface. A conservative assumption of this assessment is that the entire amount of boric acid released from the barrier is subsequently transferred to the surface of the skin. These results were used in the "typical" exposure scenario.

A final series of tests were conducted on full-scale twin mattresses. The average concentration of boric acid for the boric acid treated barriers with the highest (urine) observed migrations over the four successive extractions on the twin-sized mattress was 47.45 $\mu g/cm^2$ or 8.3 $\mu g/cm^2$ for boron (Table 12b). The migration of boric acid from the mattresses treated with simulated perspiration was slightly lower 38.25 $\mu g/cm^2$ (equivalent to 6.7 $\mu g/cm^2$ of boron). These data are extrapolated to typical consumer uses of mattresses and used as estimates of upper bound exposures (Midgett, 2005; Appendix 5).

Oral Exposure

The boric acid migration data used to estimate dermal exposure have been used to estimate oral exposure for children and adults. The HOH data collected in Phase 2 of this study were not used to estimate oral ingestion because children and adults are not expected to directly mouth FR-treated barriers. The amount of boric acid covering the body is assumed to be available for oral intake in addition to dermal absorption if these exposed areas are mouthed. The amount of area specified is an estimated integration of the total surface area contacted by the mouth during a number of discreet hand-to-mouth activities during the course of a night and early morning (Midgett, 2005). CPSC staff estimated the total amount of surface area to be mouthed through this series of activities to be 6 cm^2 for adults and 8 cm^2 for children (Midgett, 2005). In addition, a child is expected to directly mouth 5 cm^2 of the mattress surface resulting in 13 cm^2 of total mouthed surface. The amount of boric acid that will be transferred to the hand is equivalent to the amount that migrated to the surrogate skin. The amount of FR chemical that is expected to migrate to the hand and mattress surface is 4.1. ug/cm^2 resulting in an intake of 25 $\mu g/d$ for adults and 53 $\mu g/d$ for children. It is unlikely that the transfer efficiency is 100%, therefore this estimate is considered to be a conservative estimate of oral exposure.

Sexual activity may increase the amount of FR chemical that may enter the body through ingestion. The frequency of this activity is highly variable and expected to be intermittent (Midgett, 2005; Weis, 1997-2001). Quantifying representative exposure and extrapolating to a daily intake is difficult. However, the exposures may not add significantly to the overall daily intake of FR chemical if the intermittent ingestions are extrapolated over a monthly time period as in the case of urine-mediated dermal exposure in children.

Inhalation Exposure

Boric acid and antimony containing barriers 1 and 9 were incorporated into mini-mattresses and subjected to the impaction testing. These aged and unaged mini-mattresses were subjected to the impaction tests in an enclosed chamber to estimate the amount of boric acid that may be released during the estimated 10 year lifetime of the mattress. The chamber was a closed system with air flow through pumps re-circulated back into the chamber. Samples were collected from the enclosed impaction chamber on four cyclone impactors. Two of the cyclone samplers contained cellulose acetate filters, and two cyclone samplers contained PVC filters. The amount of boric acid collected on all four filters was combined for mini-mattresses 4 and 5, which contained barrier 9

(Cobb, 2005). An average of 10.0 μg boron was released from mini-mattresses 4 and 5 during the 100,000-cycle impaction. This value was extrapolated to a full-sized twin mattress, and a correction factor of 20 was also applied to the result to account for the non-respirable fraction, for an estimated total of 7 mg released from the mattress during the estimated 10 year lifetime of the mattress.

The value used for the inhalation exposure is based on the concentrations found in Table 7 of the Cobb, 2005 memorandum. The sampling was completed on an aged mini-mattress that contained barrier 9. The aging process is described in the LSC staff report (Cobb, 2005). The values for the PVC and CE filters were used to determine airborne concentrations of boric acid. Since four filters were used to collect boric acid, the amount collected on the 2 CE filters and the 2 PVC filters were combined to estimate the total amount of boric acid released during the entire 100,000 cycle impaction. The filters collect particles that are considered respirable. Since non-respirable particles may also enter the body, a 20-fold correction factor was applied to the results to account for the amount non-respirable particles entering the body.

Based on the correction for the two PVC filters, the overall mean concentration of boric acid released was 10.0 μg over 100,000 cycles. The correction factors of 35, which extrapolates values for the mini-mattress to a full-scale mattress, and 20 which accounts for non-respirable particles, were applied to the 10.0 μg resulting in 7.0 mg as an input value for the risk calculation. The 100,000 cycle was based in-part on ASTM standard F1566 (Part 9) that used a vertical impactor for 100,000 cycles to simulate the wear that may occur during the 10 year useful lifetime of the mattress. The amount of boric acid released during the test is extrapolated over 8 and 11 hours per day for adults and children respectively, for 10 years to estimate the amount of boric acid that may be released during daily consumer use.

c) Decabromodiphenyl Oxide (DBDPO)

Dermal and Oral Exposures

The amount of FR chemical released from barriers treated with DBDPO during consumer use and transferred to the skin was estimated from the more conservative tests simulating direct skin contact with the FR-treated barrier. These tests involved placing the surrogate skin (filter paper) directly on the barriers and applying simulated sweat or urine on top of the surrogate skin. A 1 psi weight was then placed on top of the filter paper. The average concentration of DBDPO released from barrier 7 when simulated sweat was applied was 0.05 $\mu g/cm^2$ and 0.04 $\mu g/cm^2$ for simulated urine. Because the amounts of DBDPO released from the treated barriers were low for this relatively aggressive test, no additional dermal migration experiments were conducted for this barrier/FR chemical.

Inhalation exposure

A mini-mattress was constructed incorporating the DBDPO-treated barrier 7 (Cobb, 2005). The unaged mini-mattress was subjected to the 100,000 cycle 3 psi impaction described earlier. The amount of DBDPO released during the entire impaction process was 0.7 μg (Table 15; Cobb, 2005). This amount was extrapolated to the amount that would be released from a full-sized mattress by applying a correction factor of 35. A

correction factor of 20 was also applied to the result to account for the non-respirable fraction.

B. Risk Assessment

1. Review of Models and Input Parameters

A previous section of this report summarizes the input parameters used to calculate the potential risk of health effects from the FR chemicals reviewed in this report. The models estimate the risks for a 72.25 kg adult and 19.2 kg child. Sleeping in a room with a breathing zone of 1.85 m^3 for 8 and 11 hours per day, respectively, it is assumed that the adult and child sweat heavily and that this moisture penetrates through the sheets and ticking into the barrier. The dermal migration test results estimate the amount of FR chemical that migrates to the surface and comes in contact with the skin. The results have been conservatively extrapolated with the assumption that the entire surface area of the adult (18,200 cm^2) and child (7,900 cm^2) will be covered with the FR chemical in the amounts observed in the surrogate skin in the dermal migration tests.

For children about 5 years old, it is also assumed that additional FR chemical will migrate from the barrier as a result of urination, which is expected to occur for 2 days each month. If urination is more frequent, it was assumed that caretakers would use some type of barrier such as a plastic cover to prevent mattress soiling. This would also minimize FR chemical migration and contact with the skin. FR migration from urine is estimated to cover approximately 1,092 cm^2 (~13%) of a child's skin surface area.

The amount of FR chemical that is deposited on the skin may also be ingested orally. It is assumed that adults and children will mouth 6 cm^2 and 13 cm^2, respectively, of body and mattress (children only) surface, which includes the face and the hands, during the course of the night and during the early morning after the sleep episode before being washed (Midgett et al., 2005).

FR chemicals may also be inhaled. It is assumed that an adult and child will inhale 0.6 and 0.4 m^3/h, respectively, while sleeping. For antimony and boric acid the amount of FR chemical released into the air and available for inhalation was estimated from the impaction of aged mini-mattresses and DBDPO of a new mini-mattress in an enclosed chamber. A certain portion of the airborne particles is assumed to be of respirable size. A correction factor (20) is applied to the final result to account for non-respirable particles entering the body. The particles are assumed to be released at a constant rate and they are expected to be uniform with respect to FR content. The particles are assumed to remain airborne in a confined breathing zone of 1.85 m^3.

2. Estimation of Average Daily Dose

The models and assumptions used to estimate the average daily dose from each route of exposure, dermal absorption, inhalation, and ingestion are described in a previous section of this report. The average daily doses of these compounds are presented in Tables 16 and 17. The average daily dose from each route of exposure was summed to estimate the

total amount of each FR chemical that is expected to enter the body as a result of sleeping on a mattress containing the FR-treated barrier.

The average daily dose is then compared to the ADI. The acceptable daily dose is based on doses that enter through the oral route. However, the entire amount of FR chemical entering the body from all routes of exposure, is compared to the ADI due to the lack of exposure-specific ADIs for these compounds (Tables 16 and 17). If the quotient of the ADD/ADI (referred to as the hazard index (HI)) is greater than one, the product or exposure scenario under consideration is considered to present a hazard to consumers.

3. Inhalation Effects of Antimony

a) Chronic Inhalation Effects

An inhalation-specific ADI does exist for antimony and it was also the only compound that is believed to have any carcinogenic effects. These effects are observed only through inhalation of antimony. The effects are seen in the deep lung and are not cumulative, thus an exposure duration of 10 years was assumed for children and adults. The amount of antimony released during the 100,000 cycle chamber test was extrapolated over the 10 year mattress lifetime to estimate that average daily dose (ADD).

b) Carcinogenic Effects

In calculating cancer risks, which depend on cumulative exposure, the cancer risk in adults represents the risk from a lifetime of exposure, 75 years. The cancer risk in children represents the contribution to the lifetime risk from exposure during 70 years of product use. It was conservatively assumed that after the ten year lifespan of a mattress, the consumer would purchase another mattress containing an antimony-treated barrier, and this purchasing trend would continue for the duration of their lifetime. This conservative assumption of continuous use of a treated mattress throughout the 75 year consumer lifetime (70 years of product use; 75 - 5 years that a child sleeps on a mattress protected with fluid-resistant ticking or mattress covers due to bed wetting) is applied only to antimony since exposures are cumulative with regards to the increased risk of developing cancer later in life.

4. Results

a) Ammonium Polyphosphate

Ammonium polyphosphate is not considered to be "toxic" under the FHSA and, therefore, it is not considered "hazardous." The National Academy of Sciences' (NAS) National Research Council (NRC) also concluded that ammonium polyphosphates are probably not potent toxicants. Because ammonium polyphosphate is not classified as "toxic," an exposure assessment was not needed to determine whether it may be hazardous. However, limited migration data were developed for this compound, where significant quantities were released from treated barriers. Regardless of the amount of exposure, ammonium polyphosphate is not expected to result in any health effects in consumers because it is not considered "toxic".

b) Antimony

The risk of health effects from antimony are represented in three ways. The first case represents the aggregate exposure and risk from all three routes of exposure including urine-mediated dermal exposure in children. The HI for the basic case (systemic effects) is less than one for adults (0.005) and for children (0.01) (Tables 16 and 17), and thus is not expected to present an unacceptable risk of health effects to consumers. In adults, dermal exposure was the primary route of exposure; the contribution to systemic exposure from inhalation of particles was negligible. In children, dermal exposure presented the primary route of exposure.

Antimony trioxide is also considered toxic by inhalation. The inhalation hazard index for non-cancer effects was 0.006 in adults and 0.009 in children, thus antimony is not expected to cause adverse pulmonary effects. The lifetime individual excess cancer risk was estimated to be 0.027 per million (2.7×10^{-8}) in adults and 0.037 per million (3.7×10^{-8}) in children. Generally, cancer risks greater than 1 in a million (1×10^{-6}) are of concern. The calculated estimates for inhalation exposure to antimony are below this level, suggesting that the risk of cancer through inhalation of antimony in treated mattresses is minimal.

c) Boric Acid

Boric acid is typically applied to cotton batting through an immersion process. Boric acid is applied to the cotton fibers along with a small amount of oil and chemical surfactant to facilitate the bonding of the boric acid to the cotton fibers.

Boric acid met the definition of "toxic" under the FSHA as a probable reproductive and developmental toxicant in humans, based upon sufficient evidence of chronic toxicity in animals. Studies of human exposure to boric acid have demonstrated that this compound is poorly absorbed through the skin (Wester et al., 1998).

Laboratory tests demonstrated that boric acid is soluble in aqueous (water based) liquids and will migrate from barriers that have been wetted. Subsequent laboratory tests that added ticking and sheets over barriers show that these materials may reduce exposure to boric acid from mattresses.

The primary route of exposure was oral ingestion for young children and dermal absorption for adults. The HI for all routes of exposure was 0.05 for children and 0.01 for adults. Based on the results of the laboratory testing, boric acid is not expected to present an unacceptable risk of health effects to children or adults.

d) Decabromodiphenyl Oxide (DBDPO)

DBDPO is applied to another material as a back-coating, where it is used in combination with AT. DBDPO is not acutely toxic. However, DBDPO meets the definition of "toxic" under the FHSA, based on sufficient evidence of chronic toxicity in animals (reviewed in Bittner, 1999). DBDPO caused effects in the liver following subchronic or chronic oral exposure in rats and mice. The CPSC staff derived an ADI of 3.2 mg/kg-d. The percutaneous absorption rate was measured *in vitro* (Hughes, 2000). Data used to

estimate the amount of DBDPO that may migrate from barriers were derived from the more aggressive test where the surrogate skin (filter paper) was placed directly on the barrier with a 1 psi weight. Relatively low levels of this compound were released from the barriers. Migration was also very low with the head-over-heels method, which was an aggressive extraction procedure, suggesting that DBDPO is a durable FR treatment.

The HI is 0.0003 in adults and 0.001 in children (Tables 16 and 17). The HI was less than 1.0 in all cases. In adults and children, dermal exposure was the primary route of exposure; the contribution from inhalation of particles was estimated to be negligible. In both adults and children, oral exposure from mouthing contaminated areas of the body represented a relatively small fraction of the total daily dose. Based on this information, DBDPO is not expected to present an unacceptable risk of health effects to consumers.

e) Melamine

Melamine does not satisfy the FHSA definition of "toxic" and, therefore, it is not considered "hazardous." Because melamine is not classified as "toxic," an exposure assessment was not needed to determine whether it may be hazardous. However, limited migration data were developed for this compound in two forms, as a resin and as a surface coating. The exposure to melamine is expected to be minimal when it is incorporated into barriers in the resin form, but may be higher when it is applied as a surface coating. Regardless of the amount of exposure, melamine is not expected to result in any health effects in consumers because it is not considered "toxic".

f) Vinylidene Chloride

Vinylidene chloride is polymerized along with other compounds such as antimony and spun into fibers as polyvinylidene chloride. The vinylidene chloride monomer is rapidly absorbed through inhalation, and toxicity resulting from these inhalation exposures has been observed in laboratory animals. These effects are not expected to occur from the polymerized vinylidene chloride (polyvinylidene chloride), and only minimal amounts of residual vinylidene chloride monomer are expected to be released from the polyvinylidene chloride polymer.

Barriers containing polyvinylidene chloride were subjected to rigorous extraction tests (Bhooshan, 2005) to quantify the release of vinylidene chloride monomer. Detectable concentrations of this compound were not found; thus additional exposure tests were not conducted for this compound. It is believed that exposure to unpolymerized VC monomer would be minimal because of the extreme volatility of this compound. Without any significant exposure, the risk of any health effects resulting from the use of barriers containing polymerized vinylidene chloride is expected to be minimal.

5. Discussion

a) Assumptions and Limitations

The purpose of the present risk assessment is to predict consumer exposure to FR chemicals incorporated into barriers used in mattresses and the potential risk of chronic health effects associated with that exposure. The hazard identification and dose response

assessment were based primarily on animal studies. Only chronic health effects were considered. The exposure assessment was accomplished by evaluating a series of dermal, oral, and inhalation exposure scenarios. Input data for the exposure assessment included migration (leaching) data, *in vivo* or *in vitro* percutaneous absorption data, and assumptions regarding consumer behavior. Due to the complexity of the exposure assessment, only point estimates of exposure were calculated. However, a variety of exposure scenarios were included. As with any risk assessment, there are assumptions, limitations, and sources of uncertainty. These are discussed below.

Risk assessment is an iterative process. Data on carcinogenicity, developmental and reproductive toxicity, or neurotoxicity were not available for all chemicals. Furthermore, it should be noted that percutaneous absorption data were not available for antimony. In these cases, percutaneous absorption rates were assumed based on data obtained with surrogate compounds with similar physico-chemical properties.

The present risk assessment incorporates new data on liquid-mediated migration and inhalation exposure resulting from physical impaction of mini-mattresses. These data were used to estimate dermal, oral, and inhalation exposure and internal dose. However, data gaps remain that can be addressed with additional laboratory studies. Mini-mattress liquid-mediated migration data are available only for antimony and boric acid. Limited testing of full scale mattresses was completed for boric acid. Testing of full-scale mattresses for all chemicals may present an even more realistic estimation of possible consumer exposures.

6. Conclusions and Recommendations

Extensive migration data were available for antimony trioxide (AT), boric acid, and DBDPO. Based on this risk assessment, the CPSC staff concludes that AT, boric acid, and DBDPO are not expected to pose any appreciable risk to consumers who sleep on treated mattresses. Detectable concentrations of vinylidene chloride were not found in initial rigorous extraction studies, thus it is considered highly unlikely that significant quantities of this compound will be released from mattress barriers. The estimated HI values for these compounds are all less than one under all exposure conditions indicating that the compounds are not likely to present a risk to consumers. Since ammonium polyphosphate and melamine do not satisfy the FHSA definition of "toxic", these compounds also are not expected to pose any appreciable risk of health effects to consumers.

This risk assessment describes one approach that could be used to estimate exposure and risk from certain types of FR treatments. Based on the CPSC laboratory studies and assessments of exposure and risk for selected FR treatments described in this report, staff concludes that there are a number of FR treatments available including ammonium polyphosphate, antimony, boric acid, decabromodiphenyl oxide, melamine, and vinylidene chloride that are not expected to pose any appreciable risk of health effects to consumers who sleep on treated mattresses.

TABLES

Table 10. Dermal Exposure Tests - Antimony

Barrier Sample #	Filter Paper Extract	Reagent Extract	No Wt µg/cm² FRC	Avg µg/cm² FRC	1 PSI µg/cm²	1 PSI µg/cm²
			Sb_2O_3	Sb	Sb_2O_3	Sb
11	1	**Perspiration**	1.69	1.42	8.92	7.5
	2		0.67	0.56	2.25	1.89
	3		0.24	0.20	0.95	0.80
	4		0.17	0.14	0.57	0.48
	Total		2.77	2.32	12.69	10.67
	Mean/day				3.2	**2.7**
11	1	**Urine**	3.79	3.18	9.76	8.1
	2		0.52	0.43	1.14	0.96
	3		0.31	0.26	0.65	0.55
	4		0.31	0.26	0.58	0.49
	Total		4.93	4.13	12.13	10.1
	Mean/day				3.03	**2.6**

Numbers in bold are used in the risk assessment

Table 11. Dermal Exposure Tests - DBDPO

Barrier Sample #		Reagent Extract	No Weight Avg µg/cm^2	1 PSI Avg µg/cm^2
7	1	**Perspiration**	0.05	0.12
	2		0.02	0.01
	3		0.005	0.02
	4		0.03	0.04
	Total		0.1	0.19
	Mean(Day)		0.025	**0.05**
7	1	**Urine**	0.04	0.02
	2		0.01	0.03
	3		0.03	0.07
	4		0.48	0.02
	Total		0.56	0.14
	Mean(Day)		0.14	**0.035**
Numbers in bold are used as inputs for risk assessment				

Table 12a. Migration of Boric Acid From Mini-Mattresses

Barrier #9	Extract Series	Sweat $\mu g/cm^2$
	1	32.2
	2	22.6
	3	26.4
	4	12.4
	Total	93.6
	Mean/day	23.4
	Mean/day	**4.1 Boron**

Table 12b. Migration of Boric Acid From Full Scale (Twin) Mattresses

Twin Mattress	Extract Series	Sweat $\mu g/cm^2$	Urine $\mu g/cm^2$
	1	74.8	90.4
	2	33.3	42.1
	3	22.4	28.5
	4	22.5	28.8
	Total	153.0	189.7
	Mean/day	38.25	47.45
	Mean/day	**6.7 Boron**	**8.3 Boron**

Table 13. Impaction Test Results – Aged Mockups with Antimony-Treated Barriers

Barrier Sample #	Mockup ID	Filter Type	Filter Number	Sampling Time	Air Volume	Sb_2O_3 µg	Sb µg
9	4	Cellulose	3+3	28[1]	3360	0.15[2]	0.13
9	4	Cellulose	4	28	3360	0.15[2]	0.13
Total antimony Sampled Mockup 4						0.3	**0.26**
9	5	Cellulose	3	28	3360	0.15[2]	0.13
9	5	Cellulose	4	28	3360	0.15[2]	0.13
Total Sampled Mockup 5						0.3	0.26
Mean sampled/100,000 Cycles						0.3	**0.26**

[1]Results combined from 2 filters sampled at 8 and 20 Hours

[2]The amount is calculated from 1/2 the detection limit

Table 14. Impaction Test Results – Aged Mockups with Boric Acid-Treated Barriers

Barrier Sample #	Mockup ID	Filter Type	Filter Number	Sampling Time	Air Volume	Boron µg
9	4	PVC	1+1*	28*	3360	0.75*
9	4	PVC	2	28	3360	0.3
9	4	Cellulose	3+3	28*	3360	3.2*
9	4	Cellulose	4	28	3360	3.2
Total Boron Sampled Mockup 4						**7.5**
9	5	PVC	1	28	3360	0.46
9	5	PVC	2	28	3360	0.75
9	5	Cellulose	3	28	3360	4.4
9	5	Cellulose	4	28	3360	6.9
Total Boron Sampled Mockup 5						**12.5**
Mean Total Boron Sampled						**10.0** ug/100,000 impaction cycles

* - Results combined from 2 filters sampled at 8 and 20 Hours

Table 15. Impaction Test Results – Unaged Mockups with DBDPO-Treated Barriers

Barrier ID	Mockup ID	Filter ID and (Type)	Time (hrs)	Air Volume (l)	DB µg	DB µg
7	2 Unaged	1 glass fiber	28	3360	0.4	0.4
		2 glass fiber	28	3360	<0.2	0.1[1]
		3 glass fiber	28	3360	<0.2	0.1[1]
		4 glass fiber	28	3360	<0.2	0.1[1]
		Total sampled/100,000 Cycles				0.7
1 one-half detection limit used for samples						

Table 16. Risk Assessment of FR Chemicals in Mattress Barriers - Conservative Best Estimate - Adults

Parameter	Antimony	Boric acid	DBDPO
ADD Sweat mediated dermal absorption (mg)	0.7862	0.056114	0.07280
ADD Oral Ingestion (mg)	0.016200	0.02460	0.00030
ADD Inhalation (mg)	0.0000161718	0.0006215661	0.0000435394
ADD Total (mg/d)	0.802	0.081	0.07314
ADD Total (mg/kg/d)	0.011	0.00113	0.00101
ADI mg/kg/d	2.3	0.10	3.20
Hazard Index, HI	**0.005**	**0.01**	**0.0003**
Hazard Index Inhalation, HI(i)	**0.006**	**N/A**	**N/A**
Cancer Risk	**2.7E-08**	**N/A**	**N/A**

Table 17. Risk Assessment of FR Chemicals in Mattress Barriers - Conservative Best Estimate - Children

Parameter	Antimony	Boric acid	DBDPO
ADD Sweat mediated dermal absorption (mg)	0.46926	0.033491	0.04345
ADD Urine mediated dermal exposure (mg)	0.00392	0.000290	0.00026
ADD Oral Ingestion, (mg)	0.03510	0.053300	0.00065
ADD Inhalation (mg)	0.000014824	0.000569769	0.000039911
ADD Total (mg/d)	0.50829	0.08765	0.04440
ADD Total (mg/kg/d)	0.026	0.005	0.002
ADI mg/kg/d	2.3	0.10	3.2
Hazard Index, HI	**0.01**	**0.05**	**0.001**
Hazard Index Inhalation, HI(i)	**0.009**	**N/A**	**N/A**
Cancer Risk	**3.7E-08**	**N/A**	**N/A**

Table 18. Effect of Parameter Uncertainty and Variability for Selected Parameters

FR Chemical	ADI 50th percentile		ADI 95th percentile	
	Children	Adults	Children	Adults
Antimony	0.01	0.005	0.01	0.004
Boric acid	0.05	0.01	0.20	0.03
DBDPO	0.001	0.0003	0.001	0.0003

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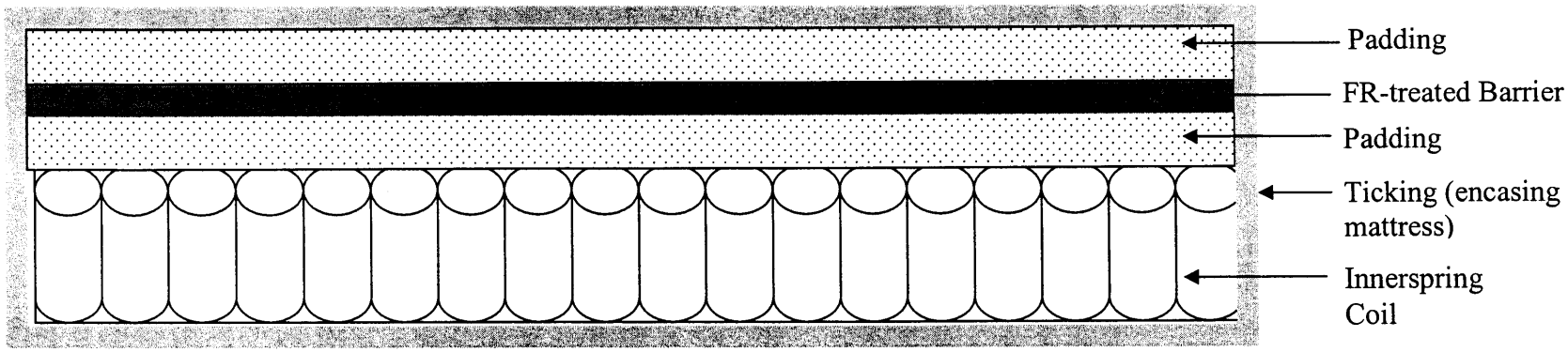
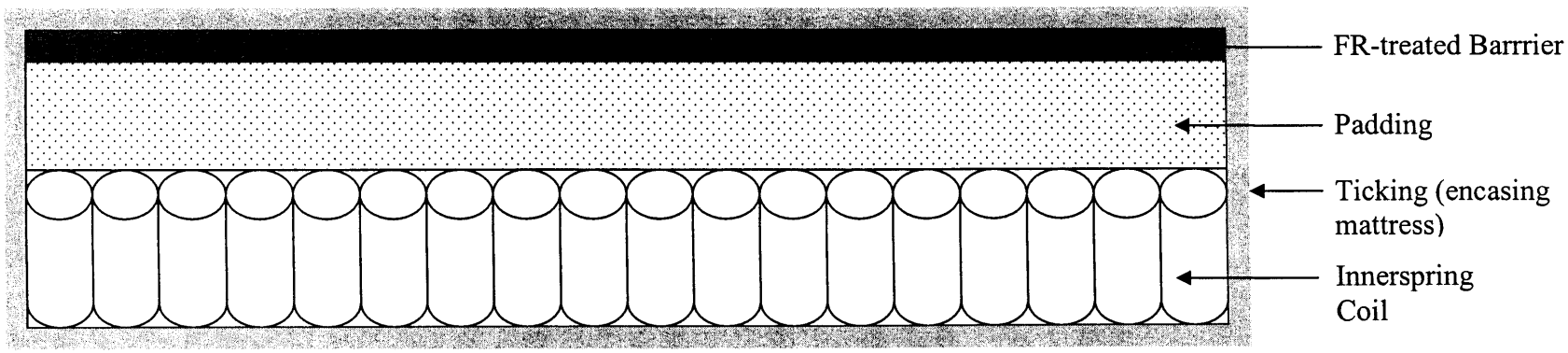
Appendix 1: Diagram of a Mattress

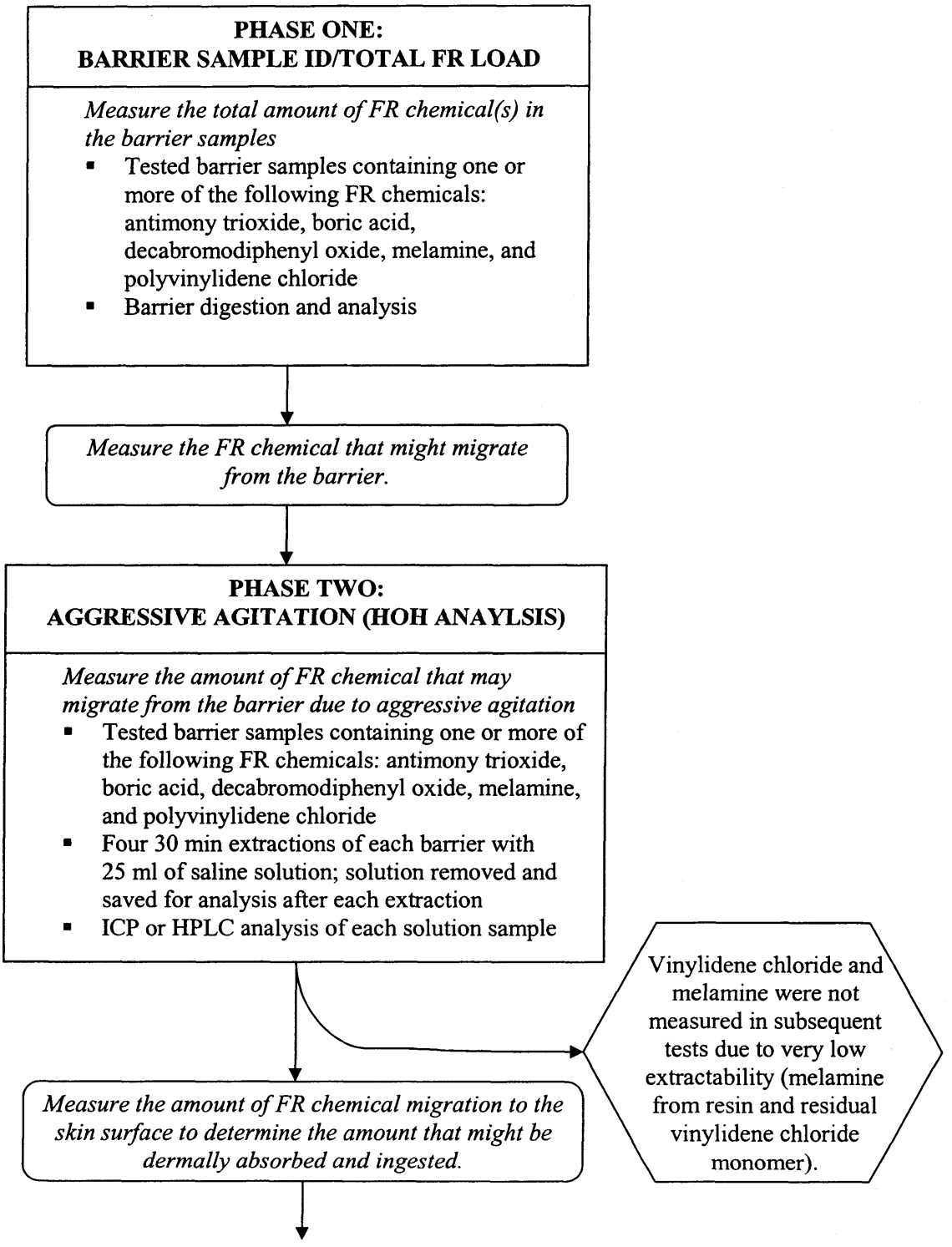
"Typical" Scenario:
FR-treated barrier under padding (i.e., foam or batting)

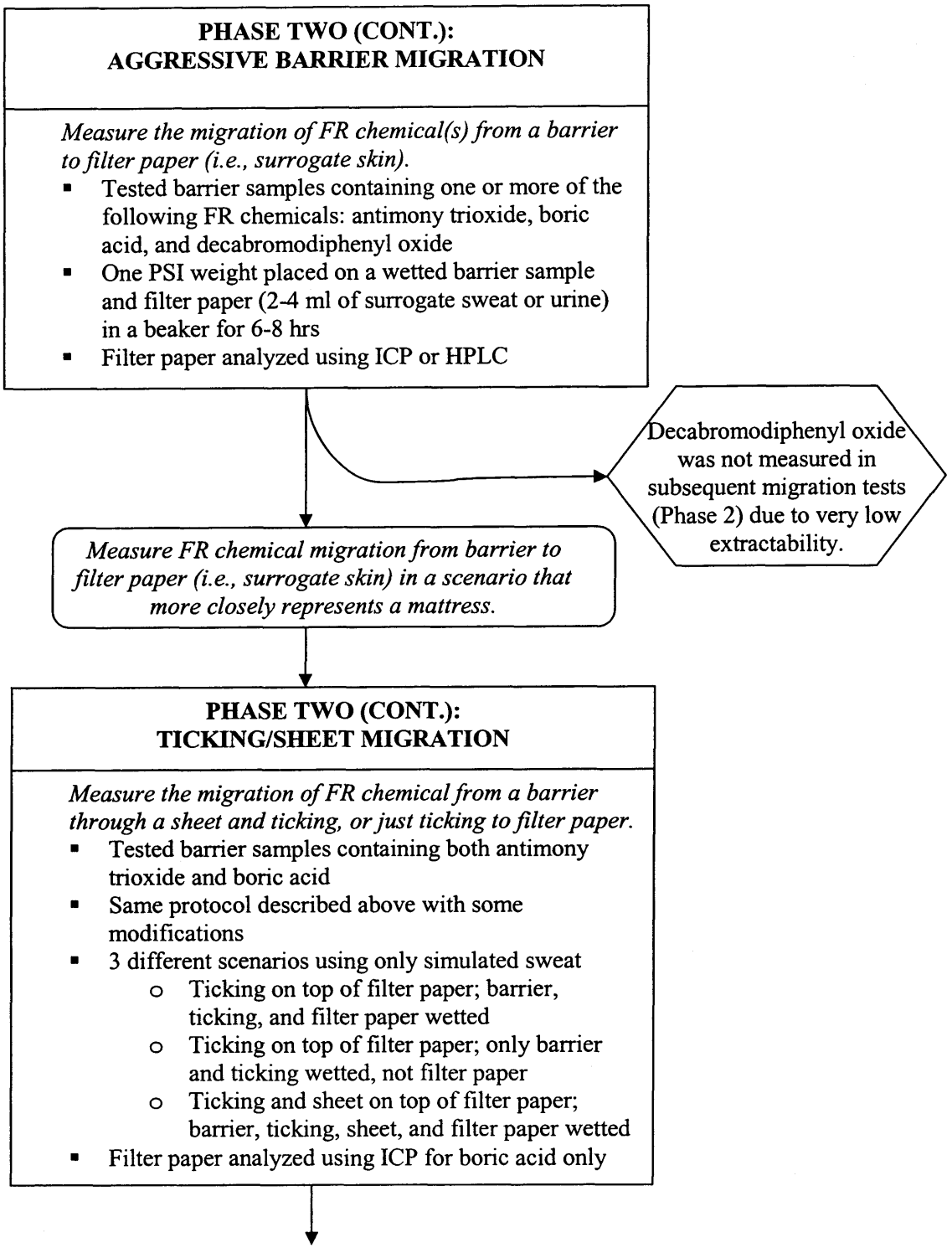


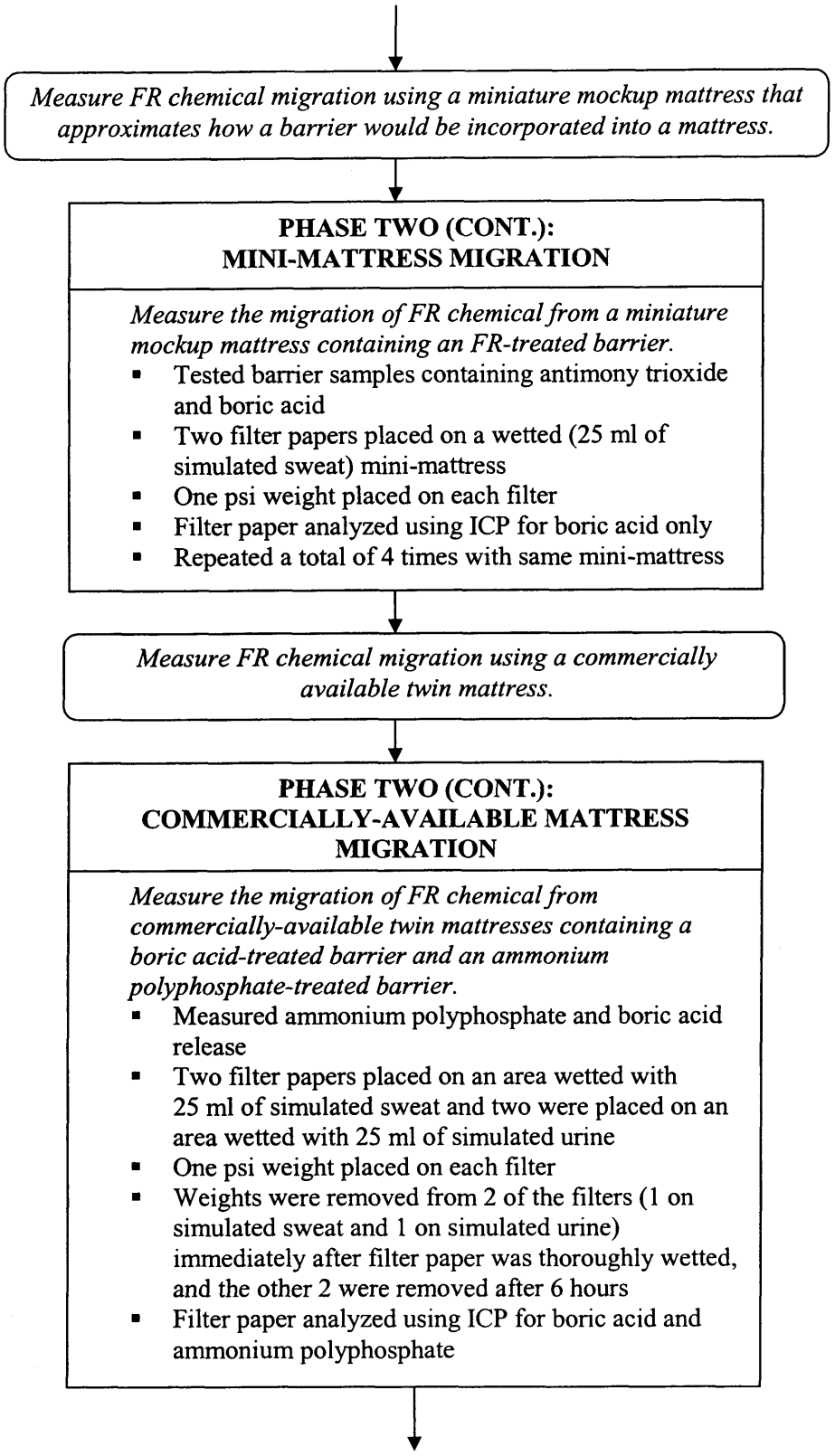
"Worst Case" Scenario:
FR-treated barrier directly under ticking

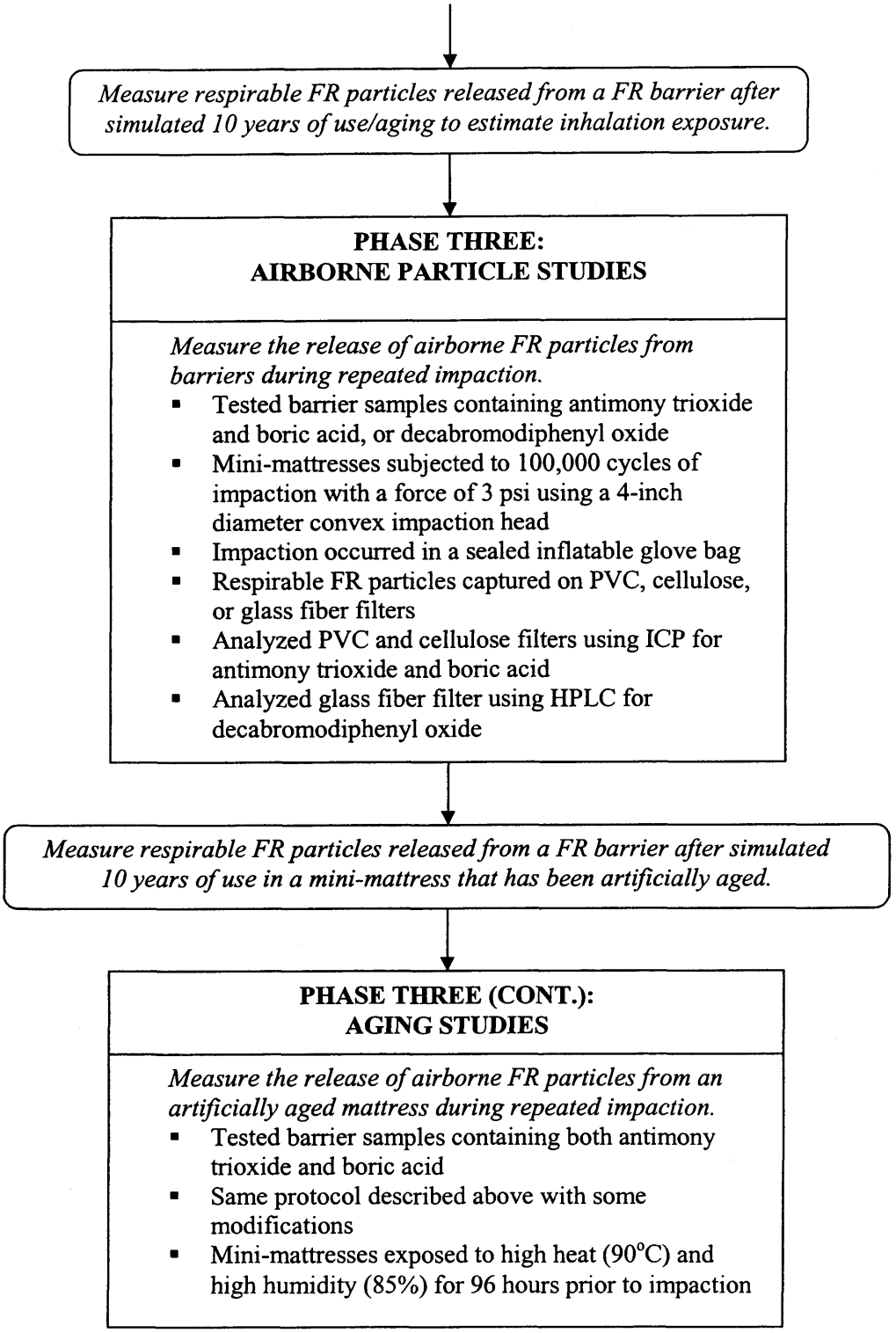


Appendix 2: Experimental Protocol Flow Chart

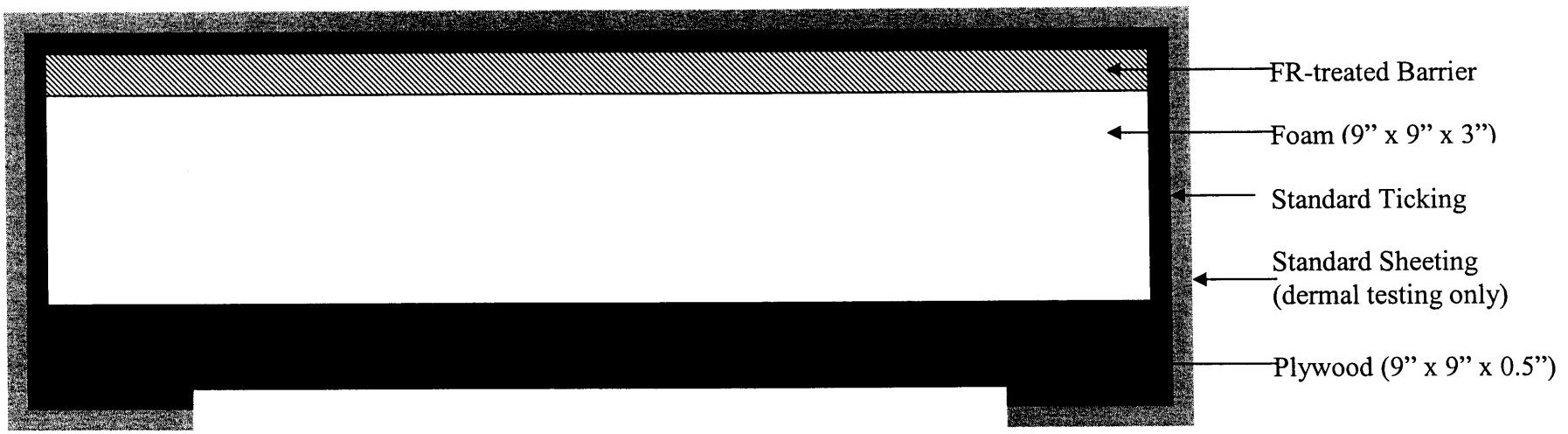



PHASE TWO (CONT.):
AGGRESSIVE BARRIER MIGRATION
Measure the migration of FR chemical(s) from a barrier to filter paper (i.e., surrogate skin).
Tested barrier samples containing one or more of the following FR chemicals: antimony trioxide, boric acid, and decabromodiphenyl oxide
One PSI weight placed on a wetted barrier sample and filter paper (2-4 ml of surrogate sweat or urine) in a beaker for 6-8 hrs
Filter paper analyzed using ICP or HPLC
Decabromodiphenyl oxide was not measured in subsequent migration tests (Phase 2) due to very low extractability.
Measure FR chemical migration from barrier to filter paper (i.e., surrogate skin) in a scenario that more closely represents a mattress.
PHASE TWO (CONT.):
TICKING/SHEET MIGRATION
Measure the migration of FR chemical from a barrier through a sheet and ticking, or just ticking to filter paper.
Tested barrier samples containing both antimony trioxide and boric acid
Same protocol described above with some modifications
3 different scenarios using only simulated sweat
Ticking on top of filter paper; barrier, ticking, and filter paper wetted
Ticking on top of filter paper; only barrier and ticking wetted, not filter paper
Ticking and sheet on top of filter paper; barrier, ticking, sheet, and filter paper wetted
Filter paper analyzed using ICP for boric acid only


Measure FR chemical migration using a miniature mockup mattress that approximates how a barrier would be incorporated into a mattress.
PHASE TWO (CONT.): MINI-MATTRESS MIGRATION
Measure the migration of FR chemical from a miniature mockup mattress containing an FR-treated barrier.
Tested barrier samples containing antimony trioxide and boric acid
Two filter papers placed on a wetted (25 ml of simulated sweat) mini-mattress
One psi weight placed on each filter
Filter paper analyzed using ICP for boric acid only
Repeated a total of 4 times with same mini-mattress
Measure FR chemical migration using a commercially available twin mattress.
PHASE TWO (CONT.): COMMERCIALLY-AVAILABLE MATTRESS MIGRATION
Measure the migration of FR chemical from commercially-available twin mattresses containing a boric acid-treated barrier and an ammonium polyphosphate-treated barrier.
Measured ammonium polyphosphate and boric acid release
Two filter papers placed on an area wetted with 25 ml of simulated sweat and two were placed on an area wetted with 25 ml of simulated urine
One psi weight placed on each filter
Weights were removed from 2 of the filters (1 on simulated sweat and 1 on simulated urine) immediately after filter paper was thoroughly wetted, and the other 2 were removed after 6 hours
Filter paper analyzed using ICP for boric acid and ammonium polyphosphate


Measure respirable FR particles released from a FR barrier after simulated 10 years of use/aging to estimate inhalation exposure.
PHASE THREE:
AIRBORNE PARTICLE STUDIES
Measure the release of airborne FR particles from barriers during repeated impaction.
Tested barrier samples containing antimony trioxide and boric acid, or decabromodiphenyl oxide
Mini-mattresses subjected to 100,000 cycles of impaction with a force of 3 psi using a 4-inch diameter convex impaction head
Impaction occurred in a sealed inflatable glove bag
Respirable FR particles captured on PVC, cellulose, or glass fiber filters
Analyzed PVC and cellulose filters using ICP for antimony trioxide and boric acid
Analyzed glass fiber filter using HPLC for decabromodiphenyl oxide
Measure respirable FR particles released from a FR barrier after simulated 10 years of use in a mini-mattress that has been artificially aged.
PHASE THREE (CONT.):
AGING STUDIES
Measure the release of airborne FR particles from an artificially aged mattress during repeated impaction.
Tested barrier samples containing both antimony trioxide and boric acid
Same protocol described above with some modifications
Mini-mattresses exposed to high heat (90°C) and high humidity (85%) for 96 hours prior to impaction

Appendix 3: Diagram of Miniature Mockup Mattress



Appendix 4: Calculation of Mattress Moisture Content

At temperate temperatures below 30°C (86°F), approximately 900ml of imperceptible water loss occurs as a result of passive diffusion of pure water from the skin (~500ml) and water vapor released in humidified expired air (~400ml) (Brown and Stubbs, 1983, Bell, Emslie-Smith and Patterson, 1976). The insensible loss of water from the skin does not involve the sweat glands. Additional water loss via sweat glands occurs, with sweat production and volume being controlled by neurally-activated mechanisms in response to different stimuli (e.g., elevated ambient temperatures, physical activity, emotional status, febrile illnesses).

Staff has assumed a conservative average estimate of 300 ml of water lost via the skin during an 8 hour sleep period (~150 ml through passive diffusion plus ~150 ml through active sweating). Profuse active sweating (resulting in visible drops) usually occurs suddenly, affecting sweat glands all over the body – regional differences in volume of sweat produced are based on proportional distribution of the number of sweat glands in different skin areas. For someone lying on a mattress, sweat that cannot effectively evaporate (e.g., in high humidity conditions and/or during copious sweat production) is likely accumulate at points where the body is in contact with the mattress. CPSC staff used a total of 1.82 m^2 (18,200 cm^2) as the default body surface area and assumed approximately one half of the body surface would be in contact with the mattress at any one time. Staff calculated that 300 ml released on a 0.91 m^2 (18,200 cm^2) mattress area would result in approximately 0.03ml water and sweat/cm^2.

During the liquid-mediated dermal migration test, CPSC laboratory staff applied 2 ml of simulated sweat onto various combinations of barrier, ticking, sheet matrices with a surface area of 20.3 cm^2. When 2 ml is applied to this area and is assumed to spread out evenly, there is approximately 0.09 ml of moisture per square centimeter of "bed" surface. These tests were also conducted on mini-mattresses, where 25 ml of simulated sweat was poured on the 522.6 cm^2 surface of the mini-mattress resulting in approximately 0.05 ml of moisture per square centimeter of surface area.

Appendix 5: Uncertainty and Variability of Selected Risk Assessment Model Parameters

Many of the values used in the parameters in the risk models are based on experimental results, published literature, or expert judgment. Although these values may be used to estimate the risk for a significant portion of the population, it may not represent the full range of possible values for the entire population. In general, the staff's analysis applied conservative assumptions in areas of scientific uncertainty, that is, assumptions that may overestimate, rather than underestimate exposure and risk. The laboratory experiments for the liquid-mediated release of FR chemicals from treated mattresses were conservative in nature, and are believed to be higher than would be experienced during most consumer use scenarios. These results were used to estimate the amount of FR chemical that would migrate to the mattress and skin surface and be either dermally absorbed, or ingested as a result of mouthing the skin or mattress surface. Estimates of body surface area and mouthing areas were determined using a combination of published literature and expert judgement. In the risk assessment calculations, values for body surface and mouthing area were selected to represent the typical consumer or "50th percentile". In the uncertainty analysis, values were selected to represent a consumer that would have much higher than average or 95th percentile values.

Mouthing Area

The suggested mouthing rate and area (1 hour daily, 50 cm^2) originated with the NAS's NRC study of flame-retardant chemicals (2000) for use in upholstered furniture. That estimate assumed exposures of a 1-year old child to furniture designed for day-time use. The CPSC's mattress exposure estimate requires consideration of furniture designed for night-time use when children are primarily asleep, and therefore interacting less vigorously with their environment. Furthermore, CPSC staff has chosen to examine older children (5 year olds) because younger children's mattresses are more likely to be waterproofed due to their higher likelihood of bed wetting. This waterproofing, either with fluid-resistant ticking or mattress covers, could provide more containment of FR particles, and so would be inappropriate for an estimate of exposures at the high end of the range of possibility. Also, mouthing of non-body-part objects decreases across the lifespan, and notably after the age of 3 years. However, staff acknowledges that some mouthing of sheets and covers may occur in 5 to 15 year old children, but believes this event would be infrequent and slight. The NRC scientists state that the actual oral exposures that they used are "hard to imagine" and could be "100-fold less" (page 51) than their mouthing parameter (50 cm^2). Because mattresses have a different use pattern, and the CPSC estimates focus on an older child, it seems reasonable to include the NRC's estimate in a modified form. Assuming that the 50 cm^2 was 100-fold less than actual exposures, then the actual exposures would be about 0.5 cm^2. If this actual estimate were increased 10 times to be conservative, this would yield an oral exposure of 5 cm^2 a day. This estimate of actual mouthing of the mattress has been added to the current hand-to-mouth estimates for a total of 13 cm^2 of mattress and body surfaces that would be mouthed by children. An additional 5-fold factor was applied to the 13 cm2 mouthing area to estimate the 95th percentile mouthing area. The increased mouthing area of 65

cm2 has been incorporated into the uncertainty analysis along with other conservative assumptions and 95th percentile factors in the models. The 95th percentile adult mouthing area of 30 cm2 was estimated for adults by multiplying the 6 cm2 are by the same 5-fold factor.

Skin Surface Area

During sleep, consumers of all ages will often toss and turn in bed which potentially allows nearly the complete surface area of the body to touch the mattress and sometimes wrap tightly in sheets and pajamas that have also touched mattress surface residues. Tossing and turning has the potential to distribute dusts from the mattress surface throughout the bedclothes and sleepwear. Agitation is expected to result in the distribution of FR particulates over large section of the body, particularly when consumers sleep without nightwear. A conservative analysis estimates FR deposition over the entire surface area of the body. The EPA exposure factors handbook was used to determine the average skin surface area of adults and children. The combined-gender average of the total skin surface area of 50th percentile adults is 1.82 m^2 and 0.79 m^2 for 50th percentile 5-year children (EPA, 1997). The combined-gender average of the total skin surface area of 95th percentile adults is 2.19 m^2 and 0.935 m^2 for 95th percentile 5-year children (EPA, 1997).

Boric Acid Liquid-Mediated Migration

Migration tests were conducted on twin mattresses that estimated liquid-mediated exposure to boric acid (Cobb, 2005). A surrogate urine and sweat were applied to these mattresses to estimate exposure that may occur as a result of sweating for adults and children, and urinating in the bed by children. The results of the tests were higher in twin mattresses than in mini-mattresses (Tables 12a and 12b), however, there were fewer replicates for the twin mattress than the mini-mattress. The results from the twin mattress were 6.7 ug/cm2 for sweat and 8.3 ug/cm2 for urine- mediated migration. These results were used in the upper bound risk calculations only.

Results

The combination of conservative values for exposure and 95th percentile values for selected parameters resulted in risk estimates that are expected to be significantly higher than would be expected in the majority of consumers sleeping on treated mattresses. Table 18 compares the risk parameters for FR chemicals where the 50th and 95th percentile values were used. The hazard indices were slightly elevated for antimony and boric acid, but no effects were seen with DBDPO. These results suggest that in circumstances where unusual or extreme consumer behavior may result in elevated exposure to FR chemicals, the increased exposure is not expected to result in an unacceptable risk of adverse health effects in children or adults.

UNITED STATES
CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, DC 20207

Memorandum

Date: January 9, 2006

TO : Margaret Neily, Project Manager for Mattresses and Bedding
Directorate for Engineering Sciences

THROUGH: Mary Ann Danello, Ph.D., Associate Executive Director for Health Sciences
Lori E. Saltzman, M.S., Director, Division of Health Sciences

FROM : Treye A. Thomas, Ph.D., Toxicologist, Division of Health Sciences
Patricia A. Brundage, Ph.D., Pharmacologist, Division of Health Sciences

SUBJECT : Response to TERA Comments on Mattresses—Toxicity of Flame Retardant Chemicals

This memorandum provides the Directorate for Health Sciences staff responses to comments made to the U.S. Consumer Product Safety Commission (CPSC) staff on the CPSC staff risk assessment of selected flame retardant (FR) chemicals that may be used to meet a flammability standard for mattresses (CPSC 2004). In September 2005, CPSC contracted with Toxicology Excellence in Risk Assessment (TERA) to review the CPSC staff risk assessment and provide written comments. Included are written comments received from TERA.

General Comments

Comment 1. All calculations and algorithm details should be checked.

Answer. The authors have checked all calculations and spreadsheets. A Health Sciences staff person not associated with this risk assessment, but with expertise in using models in spreadsheets has checked all models and calculations.

Comment 2. A table of contents should be added. The risk assessment sections could be re-organized.

Answer. A table of contents has been added. CPSC staff is comfortable with the organization of the paper.

Comment 3. The worst case scenarios should be included (95^{th} percentile).

Answer. The worst case scenario has been addressed in the uncertainty analysis section of this report where the 95^{th} percentile and other potential factors were incorporated into the calculations. This is in addition to the already conservative nature of the exposure assessment.

Comment 4. Inhalation dose calculation for antimony versus boric acid should be re-calculated.

Answer. The calculations have been adjusted by the CPSC staff.

Comment 5. Data on the inhalation exposure to DBDPO should be included, or more explanation on the lack of experimental inhalation data.

Answer. DBDPO releases into the air from the impaction experiments have been quantified. The results have been included in the risk models for DBDPO.

Comment 6. Differences between PVC5 and Mixed Cellulose Ester Fiber (MCEF) are not accurately presented.

Answer. CPSC staff has made the appropriate changes regarding the discussion of the two filters.

Comment 7. The total mass of airborne particles should be included in the risk assessment rather than the respirable fraction. In the absence of data, a 5- or 30- fold correction should be made.

Answer. The staff has adjusted the estimate of the particle exposure by applying a 20-fold correction factor. The 20-fold factor was agreed upon during a telephone discussion with the expert reviewers.

Comment 8. The volume of air that will contain particles should be reduced.

Answer. The volume of air that contains the particles has been reduced to a considerably smaller volume that largely encompasses the breathing zone.

Comment 9. Mouthing area should be increased to include 50 cm^2 of direct mouthing of sheets.

Answer. TERA's suggested mouthing rate and area (1 hour daily, 50 cm^2) originated with the National Academy of Sciences' (NAS) National Research Council (NRC) study of flame-retardant chemicals (2000) for use in upholstered furniture. That estimate assumed exposures of a 1-year old child to furniture designed for day-time use. However, CPSC staff's mattress exposure estimate requires consideration of furniture designed for night-time use when children are primarily asleep, and therefore interacting less vigorously with their environment. Additionally, CPSC staff has chosen to examine older children (5 year olds) because younger children's mattresses are more likely to be waterproofed due to their higher likelihood of bedwetting. This waterproofing, either with fluid-resistant ticking or mattress covers, is expected to reduce contact with FR chemicals, and so would be inappropriate for an estimate of exposures at the high end of the range of possibility. Also, mouthing of non-body-part objects decreases across the lifespan, and notably after the age of 3 years. Staff acknowledges that some mouthing of sheets and covers may occur in 5 to 15 year old

children, but believes this event would be infrequent and slight. The NRC scientists state that the actual oral exposures that they used are "hard to imagine" and could be "100-fold less" (page 51) than their mouthing parameter (50 cm^2).

Because mattresses have a different use pattern than upholstered furniture, and because the CPSC staff estimates focus on an older child, CPSC staff will include the NRC's estimate in a modified form. Assuming that the 50 cm^2 was 100-fold less than actual exposures, then the actual exposures would be about 0.5 cm^2. If this estimate were increased 10 times to provide a conservative estimate, this would yield an oral exposure of 5 cm^2 a day. This estimate of actual mouthing of the mattress has been added to the current hand-to-mouth estimates. The increased mouthing area of 50 cm^2 has been incorporated into the uncertainty analysis where more conservative assumptions and 95th percentile factors have been used in the models.

Comment 10. The rationale for extrapolating the aging results to a 10 year mattress lifetime should be substantiated or presented as indeterminate aging.

Answer. The mattresses that have been subjected to the aging process are classified as "aged" without regard to any specific time period.

Comment 11. CPSC staff should consider harmonizing methods of calculating ADI's with other organizations.

Answer. CPSC staff is obligated to assess the potential hazards of chemicals using the methodology outlined in the Federal Hazardous Substances Act (FHSA) and the supporting Chronic Hazard Guidelines (CPSC, 1992). While there are several methods for calculating an ADI[1], in many cases, the use of different methods does not ultimately result in substantial differences in risk. Pros and cons exist for the use of different methods. The method that the CPSC staff uses to calculate ADIs for the flame retardant chemicals that may be used with mattresses versus use of another methodology (e.g., benchmark dose methodology) does not result in substantial differences in risk as compared to that used by other organizations.

Comment 12. Comments on specific chemical assessments

Comment 12a. Derivation of the ADI for decabromodiphenyl oxide (DBDPO) should consider new studies.

Answer. CPSC staff reviewed the new studies on DBDPO. The new studies did not alter the DBDPO ADI.

Comment 12b. The possible carcinogenicity of DBDPO should be discussed.

Answer. CPSC staff previously determined that DBDPO is a possible carcinogen. Staff reviewed and discussed the evidence on the carcinogenicity of DBDPO and maintains

[1] The acceptable daily intake (ADI) is the amount of a compound that one may be exposed to on a daily basis without posing a significant risk of health effects.

that DBDPO is a possible carcinogen in humans according to the CPSC's Chronic Hazard Guidelines based on the minimal evidence of carcinogenicity in animals, along with the lack of genotoxicity. This means that DBDPO is not considered "toxic" by virtue of its carcinogenicity under the FHSA.

Comment 12c. Chemical specific adjustment factors could be applied to the ADI derivation for boric acid.

Answer. In accordance with the CPSC's Chronic Hazard Guidelines, chemical specific adjustment factors (i.e., safety factors) are not applied. For the derivation of the ADI for boric acid, CPSC staff followed the Chronic Hazard Guidelines and applied a 100-fold safety factor to account for possible differences between animals and humans, and for differences in the sensitivity among individuals.

Comment 12d. An inhalation ADI for boric acid should be calculated.

Answer. An inhalation ADI for boric acid was not calculated by CPSC staff. ADIs are calculated when a given chemical is considered "toxic" due to its chronic effects and sufficient toxicity information is available. In accordance with the guidance provided in the CPSC's Chronic Hazard Guidelines on how to evaluate toxicity studies, the CPSC staff determined that there is not sufficient evidence of systemic toxicity in humans caused by chronic inhalation exposure. Thus, staff only developed an oral ADI for which there was sufficient evidence of developmental toxicity due to oral exposure.

Comment 12e. Slow clearance of antimony from the lung could be considered, but it is unlikely to have a major impact on systemic exposure.

Answer. The impact of the slow clearance of antimony from the lung was considered by CPSC staff in its assessment of the health effects of antimony trioxide.

Comment 12f. The derivation of the vinylidene chloride ADI should be reconsidered.

Answer. No adjustments to the vinylidene chloride ADI were made. CPSC staff based its ADI on a study conducted by National Toxicology Program (NTP) (1982). Staff did not use the Quast et al. study (1983) chosen by other organizations. However, recalculation of the ADI using the Quast et al. study (1983) would not significantly affect the risk characterization as no vinylidene chloride monomer was extracted in detectable concentrations from the barriers in the aggressive migration studies.

Comment 12g. An inhalation ADI for vinylidene chloride could be developed since the compound is volatile.

Answer. Inhalation exposure to vinylidene chloride is expected to be negligible and staff concludes that it would not be sufficient to result in an unreasonable risk of health effects.

Comment 13. An expanded risk calculation including an uncertainty analysis would be useful.

Answer. An uncertainty analysis section has been added to the risk assessment. Values that represent the 95th percentile were used in the calculations in addition to the already conservative estimates of exposure.

Comment 14. Exposures from other sources (e.g., upholstered furniture) and their potential impact on risk should be mentioned.

Answer. CPSC staff estimates the potential risks resulting from the exposure from a specific consumer product. Aggregate exposures resulting from the use of other products that may contain the same FR chemical are not considered.

Comment 15. Please explain the statement (P. 33, in the context of the inhalation-specific ADI and related risk) that the effects of antimony (trioxide) inhalation are "not cumulative," particularly in light of the long half-life described above. This appears to be a non-conservative assumption.

Answer. There was a misinterpretation of the text by the reviewers which was addressed in a telephone discussion with the reviewers.

The inhalation effects of antimony are assessed by CPSC staff based on daily exposures. An inhalation average daily exposure (ADE) is calculated, and exposures are estimated to determine whether they would exceed the acceptable daily exposure. The cancer effects are cumulative. Every exposure contributes to the overall lifetime risk of developing cancer.

Comment 16. Information on the ADE for antimony and comparison to ADI and cancer risk should be included in the summary tables.

Answer. This information has been added to the tables.

Comment 17. The logic regarding the exposure to vinylidene chloride is not clear. While the volatility of the monomer would minimize the oral and dermal exposure, one might expect the volatility to increase the inhalation exposure to this chemical, particularly for a new mattress.

Answer. The volatile phase of this compound is not detectable and therefore was not measured. However, CPSC staff believes that inhalation exposure to vinylidene chloride would be negligible based on the other data collected on vinylidene chloride. CPSC staff does not consider the potential exposure to be sufficient enough to result in an unreasonable risk of health effects.

Comment 18. Report the ADIs to one significant figure.

Answer. The ADIs have been changed to one significant figure.

Editorial Comments

1. P. 8 – The description of the worst case scenario (FR-treated barrier directly under the ticking) does not appear to match the worst-case scenario diagram in Appendix 1.

   Answer. The diagram was reviewed by the CPSC staff and staff believes that the description of the worst case scenario matches the diagram in Appendix 1.

2. P. 16 – Please define and explain the extraction efficiency, as well as the rationale for the choice of 1 for this parameter.

   Answer. Extraction efficiency was explained by CPSC staff in the discussion of the oral exposure parameters.

3. Pp. 15 and 17 – It would also be useful to have a clear explanation of the dermal FR load (L_D). It would appear to be the amount of FR substance that is available, dislodgable or transferable to the skin as a per unit area property of the surface material. In the context of equation 1.7 it appears to be the assumed amount that would be injected into the air per unit area. It is suggested that this be explicitly explained, along with the rationale for using the same factor in equation 1.7 and equations 1.1-1.3.

   Answer. CPSC staff further explained dermal FR load in the discussion of dermal exposure parameters.

   CPSC staff eliminated equation 1.7. In lieu of using data from surface migration tests, data on the airborne particle release of DBDPO from a barrier in a mini-mattress was used to estimate inhalation exposure. Therefore, it was unnecessary explain the rational for using dermal FR load to determine the amount of DBDPO available for inhalation.

4. P. 20, line 7 – would be clearer if phrased as "was observed when 6 nanomoles or less ( ) was applied…

   Answer. CPSC staff made the recommended change.

5. P. 27, melamine – line 11 ff: Would be clearer if phrased as "barriers that contained melamine in a form other than as a resin did not release…" (The current wording could be read as "barriers that did not contain melamine [in any form] did not release…)

   Answer: CPSC staff made the recommended change.

UNITED STATES
CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, DC 20207

Memorandum

Date: January 9, 2006

TO : Margaret Neily, Project Manager for Mattresses and Bedding
Directorate for Engineering Sciences

THROUGH: Mary Ann Danello, Ph.D., Associate Executive Director for Health Sciences
Lori E. Saltzman, M.S., Director, Division of Health Sciences

FROM : Michael A. Babich, Ph.D., Chemist, Division of Health Sciences

SUBJECT : Response to Public Comments on Mattresses—Toxicity of Flame Retardant Chemicals

This memorandum provides the Directorate for Health Sciences staff responses to comments made to the U.S. Consumer Product Safety Commission (CPSC) on the use of flame retardant (FR) chemicals to meet a flammability standard for mattresses (CPSC 2004). Included are oral comments presented to the Commission on March 3, 2005 and written comments received from February 1, 2005 through March 31, 2005 (CPSC 2005). In the comments discussed below, numbers in parentheses refer to the list of individuals or groups that provided written comments (CPSC 2005). Names in parentheses refer to individuals who provided oral comments.

Comment

Numerous commenters stated that they were concerned about the possible toxicity of flame retardant (FR) chemicals in general (1-8, 10-501, 511, 513, 514, 518, 537-539, and 541). One of these (35) is a specialty mattress manufacturer who is also affiliated with the non-governmental organization People for Clean Beds. This organization and the manufacturer are opposed to the proposed flammability requirements for mattresses and have solicited comments to be sent directly to CPSC. Many of the commenters concerned about toxicity are directly associated with the organization and manufacturer (44-47, 51-53, 57-64, 68-76, 84, 85, and 493) and many more used identical or essentially similar language.

Some commenters, including manufacturers of mattresses or mattress components, stated that there are FR chemicals that can be used without presenting a hazard to consumers, workers, or the environment (9, 502, 518, 519, 526, 527, S. Wolf, T. Wolf, and W. Younts).

Response

In the view of the CPSC staff, there are inherently flame resistant materials and FR chemicals available that can be used to meet the proposed mattress standard and that are not expected to pose any appreciable risks to consumers, workers, or the environment. The CPSC and Environmental Protection Agency (EPA) staffs are continuing to evaluate the potential hazards of FR treatments to ensure that they do not pose any appreciable risks to consumers, workers, or the environment.

Mattress manufacturers would be free to choose the means of complying with the CPSC staff's draft flammability standard. Options available to manufacturers include the use of inherently flame resistant materials, FR barriers, and FR chemicals. To meet the draft standard, FR chemicals would most likely be applied to components inside the mattress, such as batting or barriers. However, FR chemicals might be applied to mattress ticking (cover fabric) in some cases. The potential risk presented by any chemical, including FR chemicals, depends on both toxicity and exposure. To the extent that FR chemical treatments remain bound to or within the mattress, exposure and its attendant risk would be minimized.

In addressing the hazards associated with mattress fires, the CPSC staff is working to develop a performance standard without creating additional health hazards to consumers or the environment. The CPSC staff has considered the potential chronic health risks associated with FR chemicals that may be used in mattresses to comply with the proposed standard (Thomas and Brundage 2004) and continues to study the potential exposures to FR chemicals that may occur over the lifetime of a mattress (Cobb 2005; Thomas and Brundage 2005). The staff concludes that there are inherently flame resistant materials, FR barriers, and FR chemical treatments that can be used without posing any appreciable risks of health effects to consumers (Thomas and Brundage 2004, 2005).

The CPSC staff is also working with the EPA to ensure that the use of FR chemicals does not endanger consumers, workers, or the environment. EPA has broad statutory authority over chemical substances that address potential risks to consumers, workers, and the environment. EPA has several programs such as the Design for the Environment (DfE), High Production Volume (HPV) Chemical Challenge, and Voluntary Children's Chemical Exposure Program (VCCEP) to evaluate the potential hazards of flame retardants and other chemicals to consumers, workers, and the environment. In addition, the CPSC staff is cooperating with EPA in developing a significant new use rule (SNUR) for FR chemicals that could be used to comply with CPSC or state flammability requirements for upholstered furniture. EPA's programs and statutory authority can be used to obtain additional toxicity or exposure data where needed, and complement the activities of the CPSC staff and the statutory authority of the Commission.

Comment

Some individuals commented that the "precautionary principle" should be applied to FR chemicals, that is, they should not be used until proven safe (7, 26, 44, 47, and 51).

Response

All of the statues that provide regulatory authority to the CPSC explicitly require risk-based decision making, thus precluding application of the "precautionary principle."

Comment

Several commenters recommended including in the standard a requirement that mattresses provide a label listing FR chemicals used or a statement warning of health risks (37, 38, 52, 92, 112, 130, 145, 312, 477, 504, 530, S. Baldwin). These comments included: "it will allow the consumer to make a decision regarding whether the potential hazard is a factor to be considered when purchasing these products," mattresses should be treated similar to food items, where ingredients are required to be listed, and "It is the consumer's right to have a warning label of health risks on a mattress. . . . deserves as much attention as the tobacco industry."

Response

The staff has found that numerous FR materials are available that will enable mattresses to meet the draft standard without posing any appreciable risks of health effects to consumers. Moreover, the FHSA itself would require a hazard warning label if a mattress were a "hazardous substance", as that term is defined in the FHSA. The potential health hazard associated with any chemical depends on both toxicity and exposure. A label stating the names of any FR chemicals used in the mattress would not likely provide useful information to the consumer because the mere presence of an FR chemical is not an indication that the mattress containing that chemical poses any health risk.


nment

A number of commenters were specifically concerned about the toxicity of boric acid, which is used to treat cotton batting (3, 18, 19, 21, 24, 28, 35, 99, 123, 135, 163, 166, 168, 170, 172, 198, 199, 204, 208, 220, 221, 225, 226, 235, 262, 327, 362, 373, 390, 432, 446, and 487). Some of these commenters also cited the use of boric acid as an insecticide as purported proof of its toxicity. As above, many of these comments are associated with one particular manufacturer and non-governmental organization.

Other commenters, including manufacturers of mattresses, mattress components, and chemicals, noted that boric acid has been used in mattresses for many years and that their employees have not suffered any ill effects (9, 502, 526, 527, S. Wolf, and T. Wolf). Some of these commenters also pointed out that the EPA recently increased their reference dose (RfD) for boric acid. (This means that a greater daily exposure to boric acid would be considered acceptable by EPA.)

Response

Since the publication of the Notice of Proposed Rulemaking (NPR), the CPSC staff has performed studies to estimate the potential for exposure (Cobb 2005) as well as the potential health risk (Thomas and Brundage 2005) associated with the use of boric acid as a flame retardant for mattresses. The staff's studies and analysis applied conservative assumptions in areas of scientific uncertainty, that is, assumptions that may overestimate, rather than underestimate, exposure and risk. The staff concluded that the estimated exposure to boric acid was substantially below the ADI (Thomas and Brundage 2005). Thus, boric acid is not expected to pose any appreciable risk of health effects to consumers who sleep on treated mattresses. Some manufacturers have stated that they are also developing exposure data.

Comment

One commenter specifically mentioned fiberglass as a potentially hazardous FR treatment due to inhalation of glass fibers (3).

Response

The type of fiberglass used in textiles and FR barriers (continuous filament) is not considered hazardous. Fiberglass textiles are made from "continuous filament," which contains longer, larger diameter fibers that are too large to be inhaled. Fiberglass textiles are not considered hazardous to consumers or workers (IARC 1988, 2002; Shannon et al. 1990).

Comment

Some commenters argued that the risk of dying in a fire is lower than the risk of adverse health effects from exposure to FR chemicals (3, 11, 14, 15, 21, 25, 32, 34-37, 39, 42, 49, 50, 55, 57, 98, 142, 143, 147, 150, 151, 157, 173, 175, 179, 181, 185, 218, 221, 222, 231, 238, 241, 302, 310, 311, 313, 322, 3215, 343, 347, 424, 456, and 478).

Response

The CPSC staff disagrees with the claim of some commenters that the risk of dying in a fire is lower than the risk of adverse health effects from exposure to FR chemicals. Commenters did not provide supporting data to substantiate this claim.

There are approximately 15,300 fires per year in the U.S. in which mattresses or bedding were the first item ignited, resulting in about 1,750 injuries and 350 deaths per year (Smith and Miller 2005). Thus, the risk of injury or death in a fire involving mattresses or bedding is substantial. The results of the CPSC staff's exposure and risk assessment suggest that there are a number of commercially available FR-treated barriers that can be used to meet the staff's draft mattress flammability standard that will not pose any appreciable risks of health effects to consumers who sleep on treated mattresses (Thomas and Brundage 2004, 2005).

Comment

Numerous commenters stated that they have multiple chemical sensitivity (MCS), allergies, or other health conditions that could be exacerbated by exposure to FR chemicals (2-4, 6, 14, 16, 19, 21, 31, 22, 32, 35, 42, 43, 46, 48-51, 57, 65, 83, 97, 100, 104, 105, 107, 108, 115, 121, 123-125, 129, 131, 133, 135, 137, 138. 141, 147, 152, 153, 158, 160, 163, 167, 169, 176, 178, 180-184, 189, 190, 192, 194, 196, 198, 200, 204, 205, 209, 210, 214-216, 219, 221-223, 225-227, 229, 233, 234, 237, 240, 245, 248, 249, 251, 254, 257, 258, 261, 264, 267-269, 280, 281, 286-288, 291, 293, 297, 298, 300, 307-310, 312, 313, 315-318, 321, 325, 332, 334, 336, 339, 341, 342, 345, 348, 353, 354, 364, 367, 370, 375, 384, 387, 389, 395, 403, 409, 415, 417, 420, 437, 439, 442-444, 454, 459, 461, 470-472, 474, 479, 480, 482, 484, 486, 488, 491, and 538).

Some commenters disagreed with statements that boric acid could contribute to allergies or MCS, noting that boric acid and other forms of boron have been used safely in consumer products for many years (9, S. Wolf, T. Wolf).

Response

The CPSC staff concludes that there is no evidence to suggest that FR chemicals would contribute to the causation or exacerbation of allergies, asthma, or multiple chemical sensitivity (MCS). For the most part, the materials and FR chemicals that will be used to comply with the proposed flammability standard do not share the characteristics of the types of exposures associated with the conditions noted by the commenters.

MCS is a "condition in which a person reports sensitivity or intolerance (as distinct from 'allergic') to a number of chemicals and other irritants at very low concentrations" (EPA 2005). The chemicals include both recognized pollutants—for example, formaldehyde, volatile organic compounds (VOC's), and environmental tobacco smoke—as well as agents generally considered to be innocuous, such as fragrances (ALA 1994). Health professionals and biomedical scientists differ in their views regarding the underlying causes and physiological processes of this condition (ALA 1994; Ashford and Miller 1991; Fiedler and Kipen 1997; Ziem and McTamney 1997). Non-allergic asthma and rhinitis are generally associated with exposure to respiratory irritants such as combustion products, environmental tobacco smoke (ETS), dusts, and solvents, while allergic asthma and rhinitis symptoms are most often associated with exposures to airborne biological substances, such as animal dander, insect wastes, molds, and pollen (Creticos 2000). The FR materials or chemicals under consideration are generally non-volatile, are not associated with fragrances or odors, and are not derived from biological materials.

Furthermore, the potential risks presented by FR chemicals depend on both toxicity and exposure. In most cases, FR chemicals would be applied to components inside the mattress, such as batting or barriers. To the extent that FR chemical treatments remain bound to or within the mattress, exposure and its attendant risk would be minimized.

Comment

Some commenters claimed that FR chemicals may cause sudden infant death syndrome (SIDS) (12, 64, and 283).

Response

The CPSC staff disagrees with the claim that antimony compounds or any other FR chemicals may cause sudden infant death syndrome.

The CPSC staff previously addressed this issue in detail (Bittner and Babich 2001). Following a four year study in the United Kingdom (Cullen et al. 2000; Gates et al. 1997; Jenkins et al. 1998a,b, 2000; Lehr et al. 2003; Lyon et al. 2002; Pearce, et al. 1998; Warnock et al. 1995) and reviews by a number of expert panels in the UK and the U.S., the expert panels concluded that there is no credible evidence that antimony compounds or any other FR chemical contribute to sudden infant death syndrome (SIDS). The CPSC staff concurs with the findings of the expert panels.

Comment

Some commenters were specifically concerned about the toxicity of polybrominated diphenyl ethers (PBDE's), including decabromodiphenyl oxide (DBDPO) (5, 24, 35, 36, 38, 39, 130, 289, 358, 399, and 457). Many of these commented that PBDE's have been found in breast milk and that they are the primary FR chemicals used in mattresses. Some of these commenters also noted that some states have banned the use of pentabromodiphenyl ether (penta-BDE) and octabromodiphenyl ether (octa-BDE).

Response

Pentabromodiphenyl oxide, which was used to treat flexible polyurethane foam, is no longer manufactured. The CPSC staff concluded that decabromodiphenyl oxide used in barriers for mattresses is not likely to present a hazard to consumers (Thomas and Brundage 2005). The European Chemicals Bureau concluded that there is no reason to ban DBDPO (ECB 2003). The U.S. EPA and the European Chemicals Bureau continue to review the potential environmental effects of DBDPO (see Babich 2004).

Polybrominated diphenyl ethers (PBDE's) are a family of FR chemicals that have been used in some components of upholstered furniture and mattresses, as well as other products (reviewed in Babich 2004). Octabromodiphenyl ether (octa-BDE) was a relatively minor product that was never used in mattresses or upholstered furniture. Pentabromodiphenyl ether (penta-BDE) is no longer in use. It was one of the primary FR treatments for flexible polyurethane foam (PUF), which is used in mattresses, upholstered furniture, and other applications. However, most non-California residential mattresses and upholstered furniture do not require FR-treated PUF to pass current flammability requirements.

The European Union (E.U.) and some states have banned the use of pentabromodiphenyl ether (penta-BDE) and octabromodiphenyl ether (octa-BDE). Penta-BDE and octa-BDE are no longer manufactured in the U.S. or Europe. The only manufacturer of penta- and octa-BDE voluntarily ceased production. Recently, the U.S. EPA issued a significant new use rule (SNUR), which would require any manufacturer or importer to notify EPA if they plan to produce or import either product. Thus, penta- and octa-BDE are no longer relevant to any rulemaking activities on mattresses and bedding. The banning and withdrawal of penta- and octa-BDE were primarily due to concerns about their persistence and accumulation in the environment, animals, and in human tissue. The E.U. concluded that the risk to consumers from direct exposure to penta-BDE in upholstery foam is negligible (ECB 2000). Other FR chemicals can be used to treat PUF. The U.S. EPA, through its Design for the Environment program (DfE), in which CPSC staff are participating, is working to ensure that penta-BDE substitutes do not present a hazard to consumers, workers, or the environment. However, FR-treated PUF is not necessarily needed to comply with the proposed flammability standard.

Decabromodiphenyl oxide (DBDPO), also referred to as decabromodiphenyl ether (deca-BDE), is primarily used in housings for televisions and other electronic equipment (reviewed in Babich 2004). DBDPO is less toxic and less bioaccumulative than penta- and octa-BDE. DBDPO is generally found less frequently and at lower levels than penta- and octa-BDE in the environment, animals, and human tissue. However, not all researchers routinely test for DBDPO along with other PBDE's. The E.U. has assessed the potential environmental effects of DBDPO (ECB 2002), and the U.S. EPA is reviewing DBDPO under its VCCEP program. Thus far, neither the U.S. EPA nor the E.U. has taken any steps to ban the use of DBDPO. EPA, the E.U., and the CPSC staff continue to monitor new information relating to DBDPO and, if necessary, to make appropriate changes to their risk assessments.

DBDPO can be applied to barrier materials for use in mattresses and upholstered furniture. DBDPO may also be applied in the form of a polymeric back-coating to upholstered furniture cover fabrics. However, it will not necessarily be one of the primary means of FR-treating mattresses, as suggested by some commenters.

The CPSC staff has considered the potential risks to consumers from the use of DBDPO in mattresses (Thomas and Brundage 2005). Although there is some uncertainty in the staff's analysis, the estimated exposure to DBDPO was several orders of magnitude below the ADI level. Therefore, the staff concludes that DBDPO in mattresses is not expected to prose any appreciable risk of health effects to consumers.

Comment

Some individuals commented that there is no guidance for manufacturers to consider toxicity and exposure when selecting FR chemicals (38 and 188).

Response

Under the Federal Hazardous Substances Act (FHSA), manufacturers are responsible for ensuring that their products either do not present a hazard to consumers or, if they are hazardous, that they are properly labeled according to the requirements of the FHSA. In 1992, the Commission issued chronic hazard guidelines to assist manufacturers in complying with the FHSA (CPSC 1992). The guidelines address carcinogenicity, neurotoxicity, reproductive and developmental toxicity, exposure, bioavailability, and risk assessment.

Comment

One manufacturer commented that the CPSC staff should use realistic exposure scenarios, rather than overly conservative ones (W. Younts).

Response

In assessing chronic health hazards, the goal of the CPSC staff is to determine whether "reasonably foreseeable handling and use" of a product or substance may be hazardous to consumers. Therefore, the staff generally attempts to make best estimates of exposure under realistic conditions (CPSC 1992). However, in the absence of adequate data, the staff applies "conservative" assumptions, that is, assumptions that might overestimate, rather than underestimate risk.

The CPSC chronic hazard guidelines describe various approaches to exposure assessment (CPSC 1992). Direct measures of exposure such as field studies are generally preferred over laboratory studies and mathematical modeling. However, field studies are not always practical for technical or economic reasons. Thus, the staff frequently relies on a combination of laboratory data and mathematical models.

The CPSC staff developed laboratory methods and exposure scenarios to assess the potential exposure to FR chemicals in mattresses. These methods are conservative in that they may overestimate, rather than underestimate, the potential risk.

Comment

Some commenters expressed concern about legal liabilities they felt that retailers and manufacturers could face due to the use of FR chemicals used in mattresses to meet the draft standard (88, 238, 239, 328).

Response

As discussed in the briefing package and memos, the staff believes that numerous FR materials are available that will enable mattresses to meet the draft standard without posing any appreciable risk of health effects to consumers.

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