

# HALOGENATED FLAME RETARDANTS: DO THE FIRE SAFETY BENEFITS JUSTIFY THE RISKS?

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## Introduction

Since the 1970s, an increasing number of regulations have rapidly expanded the global usage of brominated and chlorinated flame retardants. When the regulations leading to their use were implemented, the potential adverse health and environmental impacts of flame retardant chemicals were not recognized or fully understood. The five brominated flame retardants (BFRs) that have been used most extensively are tetra-bromobisphenol A (TBBPA), hexabromocyclo-dodecane (HBCD), and three commercial mixtures of polybrominated diphenyl ethers (PBDEs)—decabromodiphenyl ether (decaBDE), octabromo-diphenyl ether (octaBDE), and pentabromo-diphenyl ether (pentaBDE)<sup>1</sup>. Although penta and octaBDE have been withdrawn from the market and decaBDE is being phased out, the overall production of halogenated flame retardants (HFRs) continues to rise rapidly<sup>2-6</sup>. Chlorinated flame retardants (CFRs) in current use in the United States (U.S.) include TDCPP, [tris(1,3-dichloro-2-propyl) phosphate] also called TDCP or chlorinated Tris; TCEP or tris(2-chloroethyl) phosphate; TCPP or tris(1-chloro-2-propyl) phosphate, Dechlorane Plus, and chlorinated paraffins.

HFRs enter the environment through multiple pathways, such as emission during manufacturing, from products in use, and combustion, leaching from landfills, or recycling at the end of the product's life. Since their introduction, HFRs have become widespread global contaminants associated with a wide range of adverse effects in animals and humans, including endocrine disruption, immunotoxicity, reproductive toxicity, effects on fetal/child development, thyroid and neurologic function, and cancer<sup>3,7,8</sup>. At the end of life, hazardous flame retardants are often exported to developing countries and countries in transition such as China in electronic waste (e-waste). E-waste is frequently recycled with primitive technologies such as open burning, resulting in severe human and environmental contamination by HFRs and their combustion products<sup>9-11</sup>. Firefighters are also exposed to such combustion products, especially during cleanup after fires. Studies show elevated rates of cancers that are thought to be related to dioxin exposure among firefighters<sup>12,13</sup>.

After 30 years of widespread use, some flame retardants such as PBDEs have been banned or voluntarily phased out by manufacturers because of their environmental persistence and toxicity, only to be replaced by other organohalogens of unknown toxicity<sup>3</sup>. Despite restrictions on further production in some countries, consumer

products previously treated with banned retardants are still in use and continue to release toxic chemicals into the environment, and the worldwide use of halogenated flame retardants (HFRs) continues to increase exponentially worldwide.

### Materials and Methods

A comprehensive review of current literature was conducted by a cross-disciplinary group of scientists to determine whether the health and environmental risks of halogenated flame retardants are justified by fire safety benefits. The major findings and conclusions of this review were incorporated into the San Antonio Statement on Brominated and Chlorinated Flame Retardants<sup>14</sup> that has been signed by 200 scientists from 22 countries.

### Results and Discussion

Our analysis revealed that adding HFRs to household and consumer products results in no proven fire safety benefits. In contrast, there is mounting evidence that exposure to these chemicals is increasing worldwide and is associated with a wide range of adverse effects in animals and humans, including endocrine disruption, immunotoxicity, reproductive toxicity, diabetes, effects on fetal/child development, thyroid and neurologic function, and cancer<sup>3,7,8</sup>.

Disruption of thyroid hormone homeostasis is proposed to be a primary effect of many BFRs, of which PBDEs are the best-studied. Increasing data suggest that PBDE exposure adversely affects the developing nervous system resulting in behavioral and IQ deficits in children<sup>15</sup>. Recent *in vitro* data suggest that pentaBDE congeners may profoundly affect the development of fetal human neural progenitor cells via the endocrine disruption of cellular thyroid hormone signaling<sup>16</sup>. Adverse human reproductive/developmental outcomes related to PBDE exposure have also been reported including a longer time to pregnancy<sup>17</sup> and adverse birth outcomes<sup>18,19</sup>. Consistent with the anti-androgenic effects of PBDEs observed in experimental animals, elevated PBDE levels in human breast milk have been correlated with cryptorchidism (undescended testicles)<sup>20</sup>, and with decreased sperm count and decreased testes size<sup>21</sup>. A recent study in the U.S. reported a relationship between altered hormone levels in men and PBDE levels in house dust<sup>22</sup>. Although not well studied in humans, the adverse effects of other BFRs (HBCD, TBBPA) appear to be similar to those of PBDEs, notably disruption of thyroid hormone homeostasis and developmental effects in animals<sup>3,4,7</sup>.

Many chlorinated and brominated flame retardants have been banned or voluntarily phased out<sup>3,7</sup>, only to be replaced by others having similar characteristics. For example, in 1977 the U.S. Consumer Product Safety Commission (CPSC) banned brominated Tris or Tris-BP/tris (2,3-dibromopropyl) phosphate/ from children's sleepwear after the chemical was found to be a mutagen<sup>23</sup>. Brominated tris was also found to be absorbed into children's bodies<sup>24</sup>. The main replacement for brominated Tris was chlorinated Tris or TDCPP. After being found to be a mutagen as well<sup>25</sup>, chlorinated Tris was voluntarily removed from use in sleepwear in 1978. Both compounds are also probable human carcinogens<sup>26,27</sup>. Nevertheless, TDCPP is currently used as a flame retardant in polyurethane foam in furniture and juvenile products. A recent study showed that men living in homes with high amounts of the organophosphate (OP) flame retardants TPP and TDCPP in household dust had reduced sperm counts and altered levels of hormones related to fertility and thyroid function<sup>28</sup>.

The uncontrolled burning of products containing HFRs (such as in primitive e-waste recycling) can result in highly toxic exposure when byproducts such as brominated and chlorinated dioxins and furans are released during combustion<sup>29,30</sup>. A recent study reported adverse birth outcomes in infants of pregnant women involved in e-waste recycling in Guiyu, China<sup>18</sup>. Firefighters are exposed to brominated and chlorinated dioxins and furans during and after fire events<sup>30</sup>, and have elevated rates of four types of cancer that are potentially related to their exposure<sup>12,13</sup>.

### Conclusions

To date, many flame retardant chemicals have been produced and used without evaluation of their health and environmental impacts, resulting in human and wildlife exposure and associated adverse health outcomes. Our

major conclusions are: (1) HFR chemicals can pose a potentially greater hazard than the risk from the fires they are supposed to prevent. (2) The current options for end-of-life disposal of products treated with HFRs are problematic. (3) Life-cycle analyses evaluating benefits and risks should consider the health and environmental effects of the chemicals, as well as their fire safety impacts. (4) Most fire deaths and most fire injuries result from inhaling carbon monoxide, irritant gases, and soot. The incorporation of organohalogens can increase the yield of these toxic by-products during combustion. (5) Fire-safe cigarettes, fire-safe candles, child-resistant lighters, sprinklers, and smoke detectors can prevent fires without the potential adverse effects of HFRs. (6) Policy solutions and use of alternatives to HFRs including less flammable materials, design changes, and safer chemicals are recommended.

It is clear that the health and environmental risks of HFRs outweigh their putative fire safety benefits, and a more systematic approach to the regulation of HFRs is needed. Reducing the use of toxic or untested flame retardant chemicals in consumer products can protect human and animal health and the global environment without compromising fire safety.

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### References

1. Bromine Science and Environmental Forum (BSEF), (2003); <http://www.bsef.com>.
2. Fink U, Hajduk F, Wei Y, Mori H. (2008).
3. Shaw SD, Kannan K. (2009); *Rev Environ Health* 24:157-229.
4. Covaci A, Gerecke AC, Law RJ, Voorspoels S, Kohler M, Heeb NV, et al. (2006); *Environ Sci Technol.* 40:3679-88.
5. Law RJ, Allchin CR, de Boer J, Covaci A, Herzke D, Lepom P, et al. (2006) *Chemosphere* 64:187-208.
6. Tanabe S, Ramu K, Isobe T, Takahashi S. (2008); *J Environ Monit.* 10:188-97.
7. Birnbaum LS, Staskal DF. (2004); *Environ Health Perspect.* 112:9-17.
8. Costa LG, Giordano G. (2007); *NeuroToxicology* 28:1047-67.
9. Leung AO; Luksemburg WJ, Wong AS; Wong MH. (2007); *Environ Sci Technol* 41(8):2730-7.
10. Ma J, Addink R, Yun SH, Cheng J, Wang W, Kannan K. (2009); *Environ Sci Technol* 43:7350-6.
11. Wong MH, Wu SC, Deng WJ, Yu XZ, Luo Q, Leung AO, et al. (2007); *Environ Pollut* 149:131-40.
12. Kang D, Davis LK, Hunt P, Kriebel D. (2008); *Am J Ind Med.* 51: 329-35.
13. LeMasters GK, Genaidy AM, Succop P, Deddens JA, Sobeih T, Berriera-Viruet H, et al. (2006); *J Occup Environ Med* 48:1189-1202.
14. DiGangi, J., Blum, A., Bergman A., De Wit, C., Lucas, D., Mortimer, D. Schecter, A., Scheringer, M., Shaw, S.D., Webster, T. (2010). *Environ. Health Persp.* 18: A516-A518.
15. Herbstman JB, Sjodin A, Kurzon M, Lederman SA, Jones RS, Rauh V, et al. (2010); *Environ Health Perspect* 118:712-9.
16. Schreiber T, Gassmann K, Götz C, Hübenthal U, Moors M, Krause G, et al. (2010); *Environ Health Perspect* 118:572-8.
17. Harley KG, Marks AR, Chevrier J, Bradman A, Sjödin A, Eskenazi B. (2010); *Environ Health Perspect* 118:699-704.
18. Wu K, Xu X, Liu J, Guo Y, Li Y, Huo X.(2010); *Environ Sci Technol* 44:813-9.
19. Chao H-R, Wang S-L, Lee W-J, Wang Y-F, Pöpke O. (2007); *Environ Int* 33:239-45
20. Main KM, Kiviranta H, Virtanen HE, Sundqvist E, Tuomisto JT, Tuomisto J, et al. (2007); *Environ Health Perspect* 115:1519-26.

21. Akutsu K, Takatori S, Nozawa S, Yoshiike M, Nakazawa H, Hayakawa K, et al. (2008); *B Environ Contam Tox* 80:345-50.
22. Meeker JD, Stapleton HM. (2010); *Environ Health Perspect* 118: 318-23.
23. Blum A, Ames BN. (1977); *Science* 195:17-23.
24. Blum A, Gold MD, Ames BN, Kenyon C, Jones FR, Hett EA, et al. (1978); *Science* 201:120-3.
25. Gold MD, Blum A, Ames BN. (1978); *Science* 200:785-7.
26. Babich MA, Thomas TA, Hatlelid KM. (2006); Consumer Product Safety Commission.  
<https://www.cpsc.gov/library/foia/foia06/brief/uhff1.pdf>
27. National Toxicology Program (NTP). (2005); <http://ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s061tris.pdf>.
28. Meeker JD, Stapleton HM. (2010); *Environ Health Perspect* 118: 318-23.
29. World Health Organization (WHO). (1998); <http://www.inchem.org/documents/ehc/ehc/ehc205.htm>.
30. Birnbaum LS, Staskal DF, Diliberto JJ. (2003); *Environ Int* 29:855-60.