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RISK ASSESSMENT FOR ACUTE EXPOSURE TO PYROGEN: A PYROTECHNICALLY-GENERATED FIRE EXTINGUISHING AEROSOL

by

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DEPARTMENT OF THE NAVY
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14. ABSTRACT

Pyrotechnically generated fire extinguishing aerosols represent an alternative to halon fire extinguishers in enclosed spaces. This study reviewed existing literature on one specific type (Pyrogen) and assessed the potential health risks associated with a short-term (15 min) exposure to a Pyrogen discharge. A fairly extensive study of vapor phase components by the Russian Academy of Science suggested that concentrations of potentially hazardous gases were generally below levels of concern. However, ammonia and NO_2 may pose some concern because concentrations exceeded recognized short-term exposure limits. Specific composition of the hydrocarbons produced also merit further study as they may or may not represent an additional inhalation risk. Concentrations of solid phase particulates generated by Pyrogen appear to be well below any levels of concern given their reported chemical composition.

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EXECUTIVE SUMMARY

Pyrotechnically generated fire extinguishing aerosols represent an alternative to halon fire extinguishers in enclosed spaces. This study reviewed existing literature on one specific type (Pyrogen) and assessed the potential health risks associated with a short-term (15 min) exposure to a Pyrogen discharge. A fairly extensive study of vapor phase components by the Russian Academy of Science suggested that concentrations of potentially hazardous gases were generally below levels of concern. However, ammonia and NO_2 may pose some concern because concentrations exceeded recognized short-term exposure limits. Specific composition of the hydrocarbons produced also merit further study as they may or may not represent an additional inhalation risk. Concentrations of solid phase particulates generated by Pyrogen appear to be well below any levels of concern given their reported chemical composition.

INTRODUCTION

PHYSIOLOGICAL MECHANISMS

Aircraft fires can result in large numbers of fatalities and serious property damage. Fire extinguishing systems designed to mitigate this threat include pyrotechnic elements that rapidly disperse fire-inhibiting chemicals throughout at-risk confined spaces. Discharge from a pyrotechnically generated fire extinguisher typically disperses large quantities of particulates and vapor within the protected confined space. While effective in suppressing a fire, these chemicals may make the confined space hazardous for unprotected individuals to enter because of exposure to the discharged materials.

Any potential health risk posed by the pyrotechnically discharged materials depends upon the chemical composition and time-weighted concentration of vapor and aerosol. Physiological factors include respiration rate, tidal volume, and the deposition profile within the airway. Inspired particulates deposit along the airway mucosa as a function of particle size, with larger particulates depositing along the extrathoracic airways (nasal and oral cavities, pharynx) and finer particles depositing either in the tracheobronchial tree or the pulmonary airways. A sizable percentage of the very finest particles (< 1.0 microns) never deposit.

Mucosal clearance (removal either by bulk mucus transport or macrophage removal) and solubility in both water and lipids determines how much deposited material diffuses across the airway epithelium and into the general circulation. Solubility can be a rate-limiting step in diffusion across the epithelium; high solubility permits rapid transport into the bloodstream. Trans epithelial diffusion is greatest for materials deposited in the pulmonary airways. Materials deposited within respiratory bronchi and alveoli are not removed by mucosal clearance (the mucociliary elevator does not extend beyond the conducting airways) and are cleared only through diffusion out of the airspace through the endothelium or by macrophage action. The actual dose received by target organs is determined not only by the deposition profile and transepithelial diffusion rate but also by exposure time. Cumulative dose over the course of an exposure plays a major role in determining potential risk.

FIRE EXTINGUISHER COMPOSITION

Pyrogen is a pyrotechnically generated fire extinguishing aerosol system intended for use in aircraft and other enclosed structures. It is designed to extinguish fires by both sequestering oxygen and cooling while imposing no atmospheric environmental hazard. Pyrogen is a nontoxic solid prior to detonation (table 1). Ignition of this material generates an aerosol comprised of approximately 40% solid particulates and 60% vapor by mass. Table 2 delineates combustion products generated after 1 min of pyrotechnic discharge of a Pyrogen canister at a concentration of 100 g/m^3 as measured by the Russian Academy of Sciences and the Department of Mineral Resources, Mine Safety Unit, Lidcombe, NSW, Australia ( ). A more detailed analysis of the solid phase components indicates the particulates are primarily compromised of K_2CO_3, $KHCO_3$, $NaHCO_3$, KI, and KCI ( ) along with the nitrocellulose and carbon.

Table 1: Major components of Pyrogen prior to pyrotechnic dissemination (reference 1)

Ingredient	Mass %
Potassium nitrate	62.3
Plasticized nitrocellulose	22.4
Carbon	9
Additives	6.3

Table 2: Major components of Pyrogen subsequent to pyrotechnic dissemination in a 1 m^3 test chamber (reference 1)

Ingredient	Mass %
Potassium carbonates, solid	~ 7,000 mg/m^3
Nitrogen, gas	~ 70 vol %
Carbon dioxide, gas	~ 1.2 vol %
Carbon monoxide, gas	~ 0.4 vol %
NO, gas	109 ppm
NOx, gas	146 ppm
Ammonia, gas	~ 0.075 vol %
CHx, gas	212 ppm
Aldehydes	9 ppm
Ketones	12 ppm
HCN, aqueous	9 mg/m^3
HCN, gas	< 1.0 ppm

Fire extinguishing is accomplished primarily by a physical mechanism (heat removal) but also employs chemical removal of oxidants. Endothermic decomposition of potassium bicarbonate:

[1] $$KHCO_3\,(s) \rightarrow K_2CO_3\,(s) + CO_2(g) + H_2O(g)$$

extracts considerable energy from the combustion process. Solid potassium carbonate subsequently undergoes an endothermic phase change:

[2] $$K_2CO_3\,(s) \rightarrow K_2CO_3\,(g)$$

that further reduces energy available to maintain combustion (reference 2).

Gas phase reactions are intended to isolate the fire from oxidizing materials by the reactions:

[3] $$K^+ + OH^- = KOH$$

[4] $$K^+ + O^{2-} = KO^-$$

[5] $$KOH + H^+ = K^+ + H_2O$$

TOXICOLOGICAL ANALYSIS

PYROTECHNIC COMPOSITION

Use of Pyrogen is anticipated only for unoccupied spaces because of the obscuration created at discharge. However, accidental or emergency exposure to Pyrogen in an enclosed space or external to such a space (e.g., next to a burning engine nacelle) can occur. Pyrogen literature (reference 1) suggests 100 g/m^3 as a normal extinguishing concentration of aerosol based on extinguishing class B fires as set forth in UL 1058 Area Coverage Test.

GAS PHASE

Table 3 shows the relationship of anticipated vapor phase concentrations to accepted regulatory exposure levels based on table 2 data and corresponds to prior analysis performed by the Russian Academy of Science (reference 1). Note that only CO and NH_3 concentrations in the Pyrogen discharge exceed short-term exposure limits (STEL) but do not achieve levels immediately dangerous to life or health (IDLH). This suggests that short duration exposures (< 15 min) consistent with inadvertent entry into a space with a discharged Pyrogen canister would be tolerable and unlikely to cause irreversible injury based on gas phase toxicity

Table 3: Vapor phase products generated during pyrotechnic discharge of a Pyrogen canister (reference 1). Regulatory exposure limits for each material are also provided.

Combustion Product	Measured Concentration ppm	STEL ppm[††]	IDLH ppm[‡]
Carbon dioxide	400	30,000	50,000
Carbon monoxide	196	400	1,500
NO_2[†]	37	5	50
Ammonia	65	35	500
CHx*	212	N/A	N/A
Aldehydes**	9	150	10,000
Ketones**	12	885	3,000
HCN	< 1	10 (ceiling)	50
STEL: Short-term exposure limit (maximum concentration of a substance in air to which workers can be exposed for 15 min for four exposure periods per day with at least 60 min between exposure periods).			
IDLH: Immediately dangerous to life or health (maximum concentration from which in the event of respirator failure one could escape within 30 min without experiencing irreversible health effects).			
Notes: [†] - difference between NOx and NO, * - nonspecific (straight chain hydrocarbons have very low toxicity, cyclic hydrocarbons generally more toxic), ** - nonspecific (toxicity values: used methyl ethyl ketone for ketones, acetaldehyde for aldehydes), [††] – ACGIH (reference 4) , [‡] - NIOSH (reference 5).			

SOLID PHASE (PARTICULATES)

Pyrotechnic generation produces a polydispersed aerosol primarily consisting of $KHCO_3$ and $K2CO_3$ (table 2). Particulate size characteristics generated by pyrotechnically generating aerosols over the course of 42 separate trials are given in figure 1. Median particle size was 5.54 μm (mass), 0.85 μm (number), and 2.52 μm (surface area), i.e., the bulk of the solid phase mass is in larger (> 2 μm) particles.

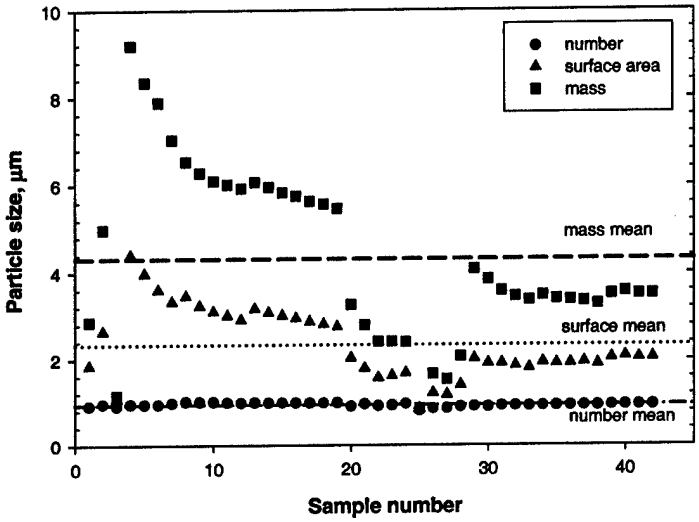

Figure 1: Experimentally measured Pyrogen aerosol particle size after pyrotechnic dispersion. Consistent number particle size indicates the majority of particles are fine (1.0 mm) though the larger particles represent the bulk of the aerosol mass.

Particle deposition in the human airway was modeled with a three-compartment model of the airway (reference 3). Figure 2 shows human airway deposition profiles during 15 min of *nasal* breathing based on the particle sizing data from figure 1. Predicted mean deposition in the three airway compartments was: extrathoracic (nasal cavity, pharynx, larynx) = 345 ± 53 μg (mean ± standard error of mean); tracheobronchial = 23 ± 4 μg; and pulmonary = 37 ± 6 μg. Total predicted deposition (sum of the three compartments) during nasal breathing = 408 ± 63 μg. Likewise, figure 3 shows human airway deposition profiles during 15 min of *oral* breathing where predicted mean deposition in the airways was: extrathoracic = 121 ± 19 μg (mean ± standard deviation); tracheobronchial = 87 ± 13 μg; and pulmonary = 95 ± 15 μg. Total predicted deposition during oral breathing = 319 ± 49 μg.

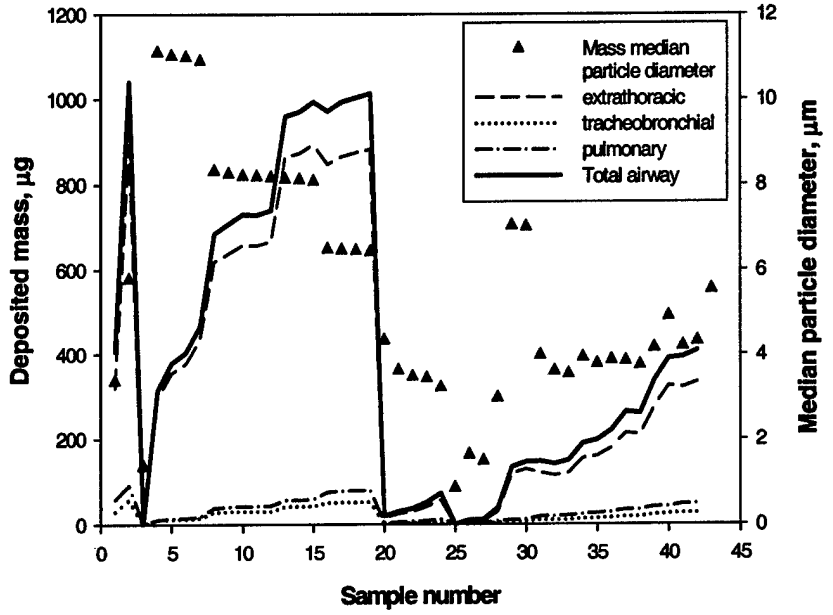

Figure 2: Predicted deposited particulate mass during nasal breathing in three airway regions (and cumulative mass) based on experimental measurement of mass median particle diameter determined after discharge of a Pyrogen canister. Most of the deposited particulates were predicted to impact extrathoracic surfaces.

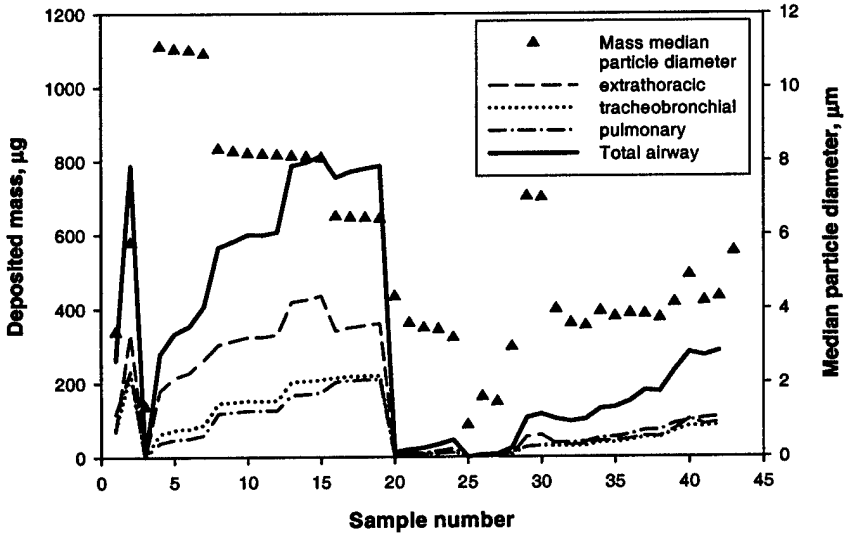

Figure 3. Predicted deposited particulate mass during oral breathing in three airway regions (and cumulative mass) based on experimental measurement of mass median particle diameter determined after discharge of a Pyrogen canister. Deposited particulates were predicted to impact more broadly along conducting and pulmonary airway surfaces.

Though the total deposited mass is predicted to be 22% greater during nasal breathing, most of this mass is estimated to impact the extrathoracic surfaces rather than enter the lower airways as predicted for oral breathing. This is due to inspired particles encountering greater surface area and more convoluted surface structures in the nasal cavity than the oral cavity and is consistent with experimental data (figure 4). These differences are attributable to the larger inspired particles (> 1 μm); finer particles are generally unaffected by nasal filtration or impaction within the nasal cavity, nasopharynx, or oropharynx (during oral breathing). In addition, lower airstream velocities are expected to contribute to greater deposition during strictly nasal breathing since oronasal breathing does not generally initiate until minute volumes exceed roughly 30 L/min (reference 6).

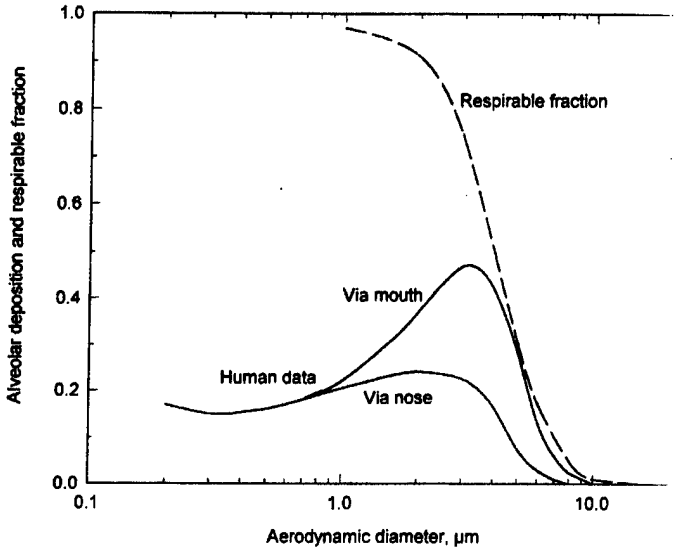

Figure 4: Experimental measurements depicting the relationship between the orifice of entry for inspired particulates and subsequent alveolar deposition for particles within the ACGIH respirable fraction criterion (reference 10).

The role of solubility also affects how deposited materials interact with respiratory tract tissue. Highly water-soluble materials (such as potassium carbonates) are much more likely to be absorbed by upper airway (extrathoracic, proximal tracheobronchial airways) mucosa and underlying tissues while lipid-soluble materials are more readily absorbed in the lower tracheobronchial and pulmonary airways (reference 7). Highly water-soluble materials will also dissolve in the periciliary fluid layer of respiratory mucus; mucosal clearance will transport these dissolved materials into the gastrointestinal tract.

The solid particulates in the Pyrogen aerosol appear to consist primarily of $KHCO_3$ and K_2CO_3 (table 1). High water solubility suggests that the entire deposited mass represents a systemic dose since upper airway absorption, mucosal clearance, and pulmonary absorption will likely transport the bulk of the solubilized $KHCO_3$ and K_2CO_3 into lung tissue and the bloodstream for transport to other organ systems. Assuming this is the case, one can use the maximum predicted cumulative total airway deposited mass to approximate systemic dose. This assumes that localized effects (e.g., on nasal mucosa) are negligible compared to overall systemic effects.

Modeling nasal breathing over a 15-min period produced an estimated maximum of 1,040 μg potassium carbonates particulates deposited over the entire airway. Likewise, oral breathing estimates produced a maximum 815 μg of deposited particulates. Assuming this mass is entirely transported to the bloodstream via absorption through both the respiratory and gastrointestinal tracts gives a worst-case systemic exposure.

One approach to estimating the equivalent human LD_{50} for potassium carbonate to the rodent LD_{50} (table 4) is proposed by Hallenbeck (reference 11). The equivalent human dose, D, is given by:

$$[6] \qquad D = (W_{hum} * L_{hum} * C_{rat} * I_{rat} * T)/(L_{rat} * W_{rat} * F)$$

where:

W_{hum} = body weight, human (kg); L_{hum} = life span, human (hr); C_{rat} = exposure concentration, rat (mg/kg); I_{rat} = consumption rate, rat (kg/hr); T = exposure time (hr); L_{rat} = life span, rat (hr); W_{rat} = body weight, rat; and F = uncertainty (safety) factor. Table 5 lists the values used to solve equation [6]. The worst case for ingested potassium carbonates was used to assess equivalent human dose. An ingested dose was used for calculations because of little of the simulated deposition occurs in the pulmonary airways during nasal breathing.

Table 4: Regulatory exposure limits for potassium carbonates typically found in Pyrogen aerosol

Material	STEL mg/m^3	LC_{50} mg/L†	LD_{50} mg/kg‡	TWA mg/m^3*
K_2CO_3 (reference 8)	3	> 4.96	1,870	-
$KHCO_3$ (reference 9)	-	>2,000	2,825	3
† - rat: inhalation, 4 hr exposure, ‡ - rat: oral delivery, * - ACGIH respirable particulates for nuisance dust				

Table 5: Standard values used to calculate equivalent human dose for rat LD_{50}.

Variable	Units	Values
Human: Weight	Kg	70
Life span	Years	75
Rat: Weight	Kg	0.5
Life span	Years	2.5
Consumption rate (food and water)	g/day	45
Exposure concentration	mg/kg	1,870
Exposure time	Hours	4
Uncertainty factor	Nondim.	10

The computed equivalent dose for a 4-hr human exposure (i.e., an estimated human LD_{50}) based on these values equals 845 mg/kg. Recalculating for a 15-min exposure (equal to the modeled airway deposition), an estimated human LD_{50} = 53 mg/kg. Predicted total airway deposition is roughly 3,500X less than this (15 μg/kg) suggesting a negligible response to exposure.

This is a simplistic approach but the apparent precision offered by more complex methods for estimating equivalent dose (e.g., physiologically-based pharmacokinetic model) often suffer for lack of sufficient knowledge to adequately describe the biological processes (e.g., biokinetics of absorbed materials, transport mechanisms, and specific toxic effects at site of action) (reference 11). In addition, the uncertainty associated with species response to irritant or toxic materials (reference 7) makes such complex modeling problematic.

Some transient localized effects may be anticipated based on an earlier study of a pyrotechnically generated fire suppressant (reference 12). Smith et al. (reference 12) exposed rats to an aerosol (2.1-2.6 μm mass mean aerodynamic diameter) of potassium chloride, the principal fire suppressant decomposition product. An observed time- and dose-dependent pulmonary edema was attributed to changes in the hydrostatic pressure gradient between the periciliary fluid and epithelial cells. Increased periciliary fluid osmotic pressure, likely caused by dissociation of potassium chloride particulates deposited onto the mucosal surface, probably led to an increased pressure gradient. Inspiring Pyrogen combustion products will likely result in a similar edematous process due to $KHCO_3$ and K_2CO_3 dissociating and markedly increasing periciliary fluid electrolyte concentration. Morbidity in rats, however, was found to be reversible after removal from the contaminant. Likewise, while humans probably risk edema during exposure to pyrolized Pyrogen, chronic pulmonary tissue damage appears unlikely.

CONCLUSIONS

1. Vapor phase contaminants are generally below levels of concern; NO2 and ammonia are above STEL levels and indicate that exposures are not without risk. Hydrocarbons generated during Pyrogen discharge may also pose some health risks; this cannot be ascertained without better characterization of these combustion products.

2. Particulates do not appear to pose a systemic health risk during short-term (15 min) exposures.

3. Localized edema is possible but appears unlikely to pose chronic health risks.

4. This toxicological assessment depended upon data provided by Pyrogen to quantitatively characterize Pyrogen combustion products. Validation of these results, especially particulate composition and mass, would be useful in better defining potential health risks.

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