Health Hazard Manual for

FIREFIGHTERS

Exposure to Chemicals and Toxic Gases

By Nellie J. Brown, M.S.

Chemical Hazard Information Program
Dr. James Platner, Toxicologist/Director
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For copies of manual call
(716) 852-4191
or write to
Cornell University NYSSILR
237 Main Street Suite 1200
Buffalo, New York 14203

I

PRINCIPLE HAZARDS OF CHEMICAL AND TOXIC GASES

Firefighters, as well as victims, can be exposed to a variety of toxic substances during a fire. Some of these toxicants are particularly insidious because they are produced by thermal decomposition before smoke makes a fire evident.

Although fireproof materials have been favored for shell construction -- wall and floor materials and furnishings tend to be synthetics. These can break down with heat to form toxic thermal degradation products; including asphyxiants, irritants, metal fumes and particulates, and gases and vapors which act on target organs.

Principle Hazards

Studies of the occupational health problems of firefighters indicate that the principal hazards (aside from asphyxiation) are irritation of the eyes and respiratory tract, polymer fume fever, pulmonary edema and long-term damage of lungs, heart and other internal organs. Approximately 20 states have "heart laws" which recognize that cardiovascular disease can be a work-related condition of firefighters. In addition to firefighters, incinerator operators may also be a work population at risk from the decomposition products discussed here. Exposure to toxic decomposition products can affect people who are unaware of a fire, especially if the smoke or fire is not readily visible or not perceived to be dangerous.

Case History: *Toxic fumes*

> "An alarm for a fire was reported on the sixth floor of Building 213 at the Washington, DC, Navy Yard. The floor was filled with heavy concentrations of toxic fumes, but the fire and smoke did not appear sufficiently excessive to prevent entry without masks, so masks were not worn. The fire was quickly extinguished, involving only an office copying machine made of PVC plastic and teflon parts. The fire companies placed several portable fans in the building for ventilation and left the scene about 20 minutes later.
>
> Upon returning to the firehouse, they experienced chest discomfort, including a searing, burning sensation, (shortness of breath), and a 'closing off' of the throat which persisted over several days. Also, severe eye irritation (tearing), and intense

headache occurred. All of these symptoms persisted for 24 hours after the fire in several firefighters. One firefighter collapsed and died about 24 hours after the fire of severe pulmonary hemorrhage and edema due to chemical pneumonia; he also had atherosclerosis found upon autopsy."

Interest has increased recently in the thermal decomposition products of building materials due to the realization that there has been a shift in the major cause of fire fatalities -- these deaths are principally due to the inhalation of toxic gases and smoke from synthetic building materials and furnishings, rather than from burns. Approximately 80% of fire fatalities in the United States result from "smoke inhalation."

Firefighters themselves have noticed that the nature of fires has changed in recent years; that they are encountering more fires involving combustible plastics in furnishings, electrical wiring and vehicle components.

In general, combustion may yield a variety of products, depending upon the composition of the fuel material:

- ❒ Particulate matter in the form of smoke particles, fluid droplets or silica
- ❒ Toxic Gases - generally carbon monoxide and dioxide; sulfur oxides; nitrogen oxides; hydrogen cyanide; hydrogen chloride, fluoride, or bromide; chlorine or bromine; simple hydrocarbons; amines; nitrites; and benzene.
- ❒ Irritant Gases - such as aldehydes, organic acids, ketones and acrolein
- ❒ Gases absorbed onto smoke particles or dissolved in liquid aerosols
- ❒ Heat
- ❒ Oxygen deficiency
- ❒ Trace compounds

Sampling studies of combustion products during real fires have shown that high concentrations of carbon monoxide and acrolein, capable of causing acute injury, have been found. Somewhat less hazardous levels have been found of HCl, HCN, NO_2, CO_2 and benzene. Benzene tends to be found in most fires,

but below levels expected to cause acute injury, although chronic injury may be possible. In one study, it was found in 181 of 197 samples analyzed.

Practically every building today contains plastics capable of producing, upon burning, gases which can damage the lungs. In the past, building codes in the United States tended to refer to fire gases from buildings using the phrase "no more toxic than wood." That is, building and furnishing materials were to be used whose combustion would result in toxic gases which were not any more toxic than wood itself when burned. Such phrases have been deleted recently since this level of toxicity was not well defined nor was there a standardized, accepted test method to assess the toxicity of these off-gases. Several states have attempted some form of regulatory activity; the approach of New York State was to enact a statute in 1982 to assess the feasibility of revising the NYS Chapter 552, Law of 1982, Uniform Fire Prevention and Building Code. Thus NYS requires that manufacturers of building materials conduct combustion tests and report the results.

This combustion toxicity information is forwarded to New York State so that such a database can be evaluated and used to develop performance standards. It will:

- ❒ make the public more aware of the problem
- ❒ give building designers and decision-makers more information on available products
- ❒ serve to push manufacturers in the direction of providing materials with lower toxic potency
- ❒ encourage economic competition and
- ❒ promote awareness of potential liability and thus bring about safer products in the marketplace

The building materials to be tested include: furniture upholstery; mattresses and bed pads; interior wall, ceiling and floor finishes; electrical wire insulation and conduit; and water distribution and sanitary pipes installed in buildings. This code would then prohibit the installation if testing was not done. Manufacturer disclosure is also required of the percentage of halogens in products as well as ignitability and flame spread characteristics.

II

HOW THERMAL DEGRADATION PRODUCTS ARE FORMED

In most fires, little oxygen actually reaches the surface of the fuel. For oxygen-containing polymers (such as cellulose of polymethlene methacrylate), most of the oxygen atoms in the irritant decomposition products come from the polymers themselves – suggesting that the break-up of polymers, not oxidation, leads to the formation of irritants. Whether thermal degradation can occur depends upon the presence of additives.

As synthetic polymer chains heat up, they acquire vibrational energy until the solid plastic becomes a highly viscous liquid. (The temperature at which this occurs is called the glass-transition temperature.) With further heating, the excited chemical bonds break with several fragmentation modes possible:

1. Some polymers undergo
    a. RANDOM SCISSION in which the polymer cleaves in an irregular manner along the chain. The polymer melts and becomes viscous, and decomposes to form gas. This is followed by
    b. UNZIPPING in which the polymer loses terminal MONOMER units.

This type of thermal decomposition occurs in:

Polyurethanes (used in mattresses, sleeping bags, military clothing) which decompose to yield the isocyanate monomer(s) used in their synthesis.
Polystyrene
Poly(methylmethacrylate)
Fluorinated resins such as Teflon, polytetrafluoroethylene, and polychlorotrifluoroethylene, which decompose to yield hydrogen fluoride, carboxyfluoride or carbonylfluoride) (13), and perfluoroisobutylene as well as monomer
Polyamides, such as nylon which breaks down to yield ammonia
Cellulosic polymers such as viscose rayon, cellophane, cellulose acetate, cellulose nitrate, cellulose ethers, ethyl cellulose

2. Some polymers undergo
    a. RANDOM SCISSION (as described above) followed by
    b. LOW AMOUNTS OF MONOMER in the degradation products and a rapid drop in molecular weight. These products include volatile fatty acids (generally formic and acetic acids), formaldehyde, and acrolein, especially at smoldering combustion temperatures of 300 to 450^{o}C.

Ammonia and hydrogen cyanide are produced from polymers containing nitrogen.

This type of thermal decomposition occurs in:

Polyethylene and polypropylene which can produce acrolein
Some acrylics such as poly(methylacrylate), modacrylics, orlon

3. Other polymers undergo
   a. INTERCHAIN CHEMICAL REACTIONS, followed by
   b. Formation of a carbonaceous RESIDUE (or char) or random chain cleavage. This process has a high yield of VOLATILES, produces little monomer, and there is little or no loss of molecular weight during the initial degradation process.

This type of thermal decomposition occurs in:

Polyesters can produce carbon monoxide, carbon dioxide, methane, ethane, propylene, ethylene, pentanes, pentenes, hydrogen, butanes, pentadienes and benzene. At pyrolysis temperatures of 360 - 1200°C, 28 to 93% of the resin may be volatilized.
Polycarbonates
Heterocyclics such as polybenzimidizole, and polyimide, polyimide-imide
Phenolics such as phenol-formaldehyde resins which release phenol and formaldehyde as well as hydrogen, carbon monoxide, carbon dioxide, methane, acetylene, ethylene, acetone, propylene, propanols, butanols, cyclopentadienes benzene, toluene, and dimethyl benzene. At temperatures of 360 to 1200°C, 11 to 48% of the resin may be volatilized.
Amino resins such as urea or melamines with formaldehyde which release formaldehyde.
Poly(vinylchloride), or PVC, is used in plastics, electrical and telephone cables and equipment, and breaks down to form principally hydrogen chloride, carbon monoxide, chlorine, and phosgene. Other gases produced include chloromethane, benzene, toluene, divinylbenzene, methyl ethyl cyclopentane, and dioxane (miscellaneous others make up <2% of the products.) Vinyl chloride-vinylidene chloride copolymer also yields hydrogen cyanide, acrylonitrile, and acetonitrile. Decomposition tends to occur at temperatures above 150°C. The smoke concentration is very low, having about 2% solids; thus it is not much of a warning for the high concentration of toxic gases evolved. This should be considered when engaging in the work of extinguishing the fire and performing rescue operations when a room contains PVC objects. Moreover, PVC plastic used in telephone cables and electrical equipment installations are especially hazardous to firefighters because when a short circuit develops in a main switch, it is usually in a basement or other poorly ventilated area. Plastic-coated switches quickly heat in excess of 93°C and the heated conductors cause the plastic insulation to decompose and emit a white mist of HCL fumes resembling steam. At temperatures of 232 to 321°C, the gas may be invisible.

-continued-

Polychlorinated biphenyls (PCBs, especially when mixed with chlorinated benzenes as is common for transformer fluid) break down to form a soot containing polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), polychlorinated diphenylenes, polychlorinated pyrenes, polychlorinated chrysenes, and polychloroxanthenes.
Epoxy resins (halogenated) break down to release chlorine, bromine, hydrogen chloride, hydrogen bromide, carbon dioxide, acetone, benzene, oxygen, and minor amounts of hydrogen, carbon monoxide, carbon dioxide, methane, propylene, acetylene, ethylene, ethane, ethene, propane, methyl chloride, ethyl chloride, butanes cyclopentadiene, pentenes, pentanes, methylcyclopentadiene, hexanes, toluene, methylbromide, heptanes, ethyl benzene, ethyl bromide, allyl bromide, methylene bromide, and butyl bromide. At pyrolysis temperatures of 360 to 1200°C, 38 to 87% of the resin may be volatilized.
Silicone rubber (also used for wire insulation) breaks down to form carbon monoxide, hydrogen cyanide, amorphous silica, hydrogen, carbon dioxide, ethylene, acetone, propylene, butenes, butadienes, hexadienes, and benzene. At temperatures of 500 to 1200°C, 6 - 17% of the resin may be volatilized. It is possible that, at these temperatures, amorphous silica may be converted to crystalline form.

Decomposition products are further complicated by the presence of fire-retardants which also yield decomposition products such as hydrogen bromide, hydrogen chloride, and metals.

III

THE EFFECTS OF THERMAL BREAKDOWN PRODUCTS

An understanding of how thermal breakdown products produce their toxic effects is not only needed to assess the effects on firefighters, but also to assess the effects on the central nervous system and on muscular coordination since these determine the ability of victims to escape from a fire situation or survive long enough to be rescued. It is assumed that the routes of entry for toxins by either the firefighter or the victim will be by inhalation, eye contact, or skin contact.

Mechanisms for the toxicity of gases

The relative toxicities of gases are dependent upon their water solubility. Since the airways are lined with mucus, the inhaled toxicant must penetrate to reach underlying tissue. Mucus thickness ranges from 5 - 10 microns and is greatest in the upper airways, declining toward the smaller air sacs (alveoli) in the lungs. The alveoli are lined with surfactant, rather than mucus, at a thickness of ~0.5 - 1 microns. The airways divide into very small tubes towards the deep lung, but the total surface area increases due to the increasing number of airways. An inhaled toxicant enters these layers and mixes with other inhaled gases. Gases which are very soluble in water can dissolve rapidly in the mucus of the throat and upper airways. Poorly water-soluble gases are breathed more deeply into the lung, causing irritation or damage in the lower airways or in the alveoli. So long as the gas is not immediately lethal, several breaths are required before the gas is absorbed sufficiently to achieve a steady state.

Gases can dissolve in respiratory mucus to cause irritation, and also dissolve in eye moisture causing irritation of the eyes or conjunctivitis. These effects could inhibit firefighters in rescue and job performance or prevent escape by fire victims by tearing, burning and generally interfering with vision.

An inhaled toxicant gas may:

❖ Dissolve in or react with lung mucus or surfactant. Mucus is removed at a rate of about 10ml/24 hrs and is moved up and out the lung by airway cilia, to be swallowed or expectorated. Irritation, such as by aldehydes, acrolein or acids, may cause airway constriction or edema. Pulmonary edema results when damage to airway cells or to alveoli causes increased permeability, thus allowing fluid to fill the space of the airways and alveoli. Since major edema may take several hours to develop, the individual may not be aware at the time

of exposure of the extent of damage possible. Infection may increase the damage. A feeling of shortness of breath (dyspnea) is caused by an inability to breathe deeply or quickly enough to satisfy respiratory needs since the tissue swelling increases the thickness through which oxygen must diffuse to reach the blood.

❖ Diffuse through the lung and saturate the blood. When this occurs, the direct effects of a toxicant tend to occur in other body organs; this is the case with some metals and benzene.

❖ React with the pulmonary cells of the lung tissue itself causing necrosis and edema. Less water-soluble materials such as anhydrous acids and strong oxidants (NO_2, SO_2, chlorine, bromine) affect tissue permeability causing edema or damage lung proteins (elastin or collagen) causing fibrosis. The transition region between bronchioles and alveoli is particularly susceptible to NO_2, since this area receives the greater toxicant dose.

❖ Damage the alveolar macrophages. The macrophages die, releasing enzymes which break down the alveolar walls causing decompartmentalization and possibly emphysema.

For water-soluble gases, such as HCl or HBr, the scrubbing action of the mucus in the nose protects the upper airways. However, upon exercise or exertion, humans become an obligatory mouth-breather and thus lose this protective mechanism. Mouth-breathing is probably also the mode used during the fear/panic/exertion of the fire scenario for the victim as well. Unfortunately, the bioassay (experimental) studies used for fire simulation employ animals who are obligatory nose-breathers only, thus animal testing does not duplicate the actual extent of human exposure.

If the toxicant is an aerosol or is absorbed onto a particle (as with HCl from PVC combustion). the site of action is determined by particle size (see below), since smaller particles are inhaled more deeply into the lungs.

Besides actual damage to the respiratory system or other organs, the inhalation of toxicants can also cause bronchoconstriction either due to direct action on the smooth muscles of the upper airways or by histamine release. Anhydrides of acids tend to produce bronchoconstriction and upper airway tissue death; while less water-soluble compounds reach the lower airways and can damage the alveoli. Also, more extensive constriction occurs during exercise than at rest due to the different ventilation of the lungs with exercise -- thus, these effects are more pronounced with the rapid breathing of the firefighter or excited fire

victim under exertion. Pulmonary function testing can be useful for medical monitoring.

HYDROGEN HALIDES: HCl, HBr and HF produce the acute effects of irritation of the upper airways and hemorrhagic pulmonary edema when inhaled.

ISOCYANATES: Animal studies show blockage of the upper respiratory tract is the cause of death from inhaled isocyanates. Toluene diisocyanate (TDI) is an irritant and sensitizer which, upon inhalation, causes an immune response and the release of inflammatory cells, particularly polymorphonuclear leukocytes and eosinophils. Hypersensitivity, an asthma-like syndrome, can occur from isocyanates even at levels below 0.02 parts per million (ppm) in air, causing acute changes in lung capacity.

PHOSGENE: Delayed pulmonary edema is also caused by phosgene. It dissolves in the moisture of the lungs to form HCl which irritates the nose and upper respiratory passages, and, upon penetrating to the deep lung, destroys the permeability of the alveoli. This effect may be delayed is about 24 hours, so exposed firefighters should be monitored and oxygen should be provided for about 48 hours as a preventive measure.

ALDEHYDES AND ACROLEIN: Although these cause irritation of the eyes and the mucus membranes of the respiratory tract, acrolein is more irritating than formaldehyde since it is an unsaturated aldehyde. High concentrations or prolonged exposure can cause bronchoconstriction, pulmonary flow resistance and decreased respiration rate. Inhalation of acrolein can also affect the liver, causing increased production of alkaline phosphatase and tyrosine-ketoglutarate-transaminase enzymes (useful for medical monitoring), as well as, cause myocardial and vascular fibroses by denaturing protein and disrupting nucleic acid synthesis. Formaldehyde could also be carcinogenic.

NITROGEN DIOXIDE: NO_2 is a deep lung irritant which causes pulmonary edema if inhaled in high concentrations for even a short exposure of 15 minutes. Adverse effects on the deep lung include: damage to mast cells, alterations in lung collagen, lesions in the alveoli, septal breaks, expansion of the alveoli and wall thinning and possible collapse of the lung.

Hydroxylysine glycosides from the breakdown of lung collagen have appeared in the urine of humans with pulmonary damage (useful for medical monitoring) after having been exposed to 250 ppm NO_2 for 4-5 minutes. Both acute and chronic exposure can cause biochemical alterations in collagen and elastin in the lungs; it is not surprising, therefore, that repeated exposure can cause

irreversible changes in lung proteins and alter lung structure. Nitrogen dioxide causes measurable alterations in pulmonary function such as an increase in respiration rate and a decrease in compliance and respiratory volume. These changes do not occur immediately, but are delayed. For example, in humans, a 10-minute exposure to 5 ppm of NO_2 caused an increase in airway resistance of 92% which did not appear until after 30 minutes had passed. There is evidence that this kind of exposure may increase sensitivity to other bronchoconstrictive agents. Also, both short- and long-term exposures increase susceptibility to respiratory infection by bacterial pneumonia or influenza virus.

SULFUR DIOXIDE: SO_2 is very water-soluble; thus, at air concentrations of less than 1 ppm, it readily dissolves in the mucus of the upper respiratory system at efficiencies greater than 90%. At less than 1 ppm it dissolves poorly in the mucus of the upper airways so it reaches the lower lung. Thus it is more damaging to the alveoli at air pollution concentrations. Also, SO_2 is more likely to reach the lower lung by mouth breathing than by nose breathing. The body absorbs SO_2. This occurs more efficiently when the lungs are exposed than if only the trachea or upper airways are exposed. Sulfur dioxide causes a mild bronchial constriction resulting in a measurable increase in flow resistance; this is due to changes in smooth muscle tone since SO_2 acts upon the sympathetic nervous system.

METALS: It is unknown to what extent adverse health effects in firefighters are caused by inhaled metals produced from fires or from fire retardants. Antimony, molybdenum, zinc, bismuth, aluminum and tin are found in fire retardants. Based upon soot samples taken from the lungs of fire victims, metals found include antimony, cadmium, lead, manganese, copper and zinc.

Inhaled molybdenum tends to be deposited in liver, kidney, fat, bones and blood. It can cause anemia because it reduces sulfide oxidase in the liver so that copper sulfide forms inducing a copper deficiency.

Inhaled zinc vapor causes metal fume fever. Symptoms appear about 4-8 hours after exposure consisting of chills, fever, sweating and weakness lasting about 24-48 hours. It is thought to be produced by fever-causing agents released when lung cell rupture from metal fume exposure.

Bismuth causes runny nose (rhinitis) and acute pulmonary edema. Chronic exposures may cause rhinitis irritation of throat, trachea or bronchial tubes, and eventually pneumoconiosis (with obstructive lung disease) and emphysema.

CARBON MONOXIDE: CO decreases the oxygen transport capability of blood causing oxygen starvation. Oxygen and carbon monoxide compete for the same binding sites on hemoglobin. Even low CO levels can cause considerable carboxyhemoglobin formation: in air with 21% oxygen by volume and 0.1% CO, the blood would have a 50% carboxyhemoglobinemia (COHb). However, upon exposure to low CO concentrations, there is a poor correlation between blood content of COHb and the signs and symptoms of exposure -- thus there may be little or no warning that a serious exposure is occurring. As hypoxia develops, the heart compensates by increasing output; this lowers blood pressure so fainting is more common than shortness of breath. Consciousness may be lost for long periods of time before death occurs; this probably explains the trapping of fire victims. Other symptoms are headache, weakness, nausea, dizziness and dimness of vision; at 50 - 80% hemoglobin saturation, there is unconsciousness, convulsion and death. Additional heart stress may also result due to the increased cardiac output and thus increased heart rate caused by CO exposure. The cherry red color of COHb in capillary blood, may give an abnormal red color to the skin, mucus membranes and nail beds. Carbon monoxide is not really a cumulative poison since COHb will dissociate when exposure has terminated, reversion to oxyhemoglobin occurs and the CO is excreted by the lungs. Smoking adds CO stress since the nonsmoking adult has less than one percent COHb, but heavy smokers may be as high as 5 - 10% saturation. This may decrease the reaction time of fire victims as well as stress the firefighter who is a smoker, especially if he smokes after a fire or after the stress of having fought a fire wearing a Self-Contained Breathing Apparatus (SCBA).

HYDROGEN CYANIDE: Death can occur within minutes or seconds of the inhalation of high concentrations of cyanide. The effects on the heart tend to bring on low blood pressure (hypotension). There can also be chronic effects from hydrogen cyanide: chemically-induced hypoxia may involve changes in the cell microtubules and neurofibrils which are particularly abundant in axons and other cell processes of the nerves. The heart is also particularly susceptible to chronic hypoxia due to the changes in acid-base balance and electolytes which result from anoxia in cells.

BENZENE: In animals, chronic inhalation of benzene has induced lymphomas, solid neoplasms, aplastic anemia and leukemia. The mode of action is unknown, but may involve the action of a benzene metabolite, such as catechol, suppressing the immune system and causing acute myelogenous leukemia in which the white blood cell count is high (>30,000/cubic mm). Aplastic anemia (pancytopenia) eventually causes tissue death and fatty replacement of bone marrow. Epidemiological evidence supports that leukemia can be benzene-induced in humans.

Mechanisms for the toxicity of particulates, smoke and aerosols

Inhalation and clearance of particulates and aerosols depends upon particle size. Gases may be absorbed onto the surface of solid particles or dissolved in liquid droplet aerosols. Particles trapped in the nasopharyngeal region range in size from 5 - 30 microns. Here they are caught by colliding with mucus-coated tissues due to the sharp change in direction of the nasal passages and mouth at the 90 degree bend in the throat. Large particles fail to make these turns and thus impact and adhere to the mucus in this area. The mucus and any trapped particles in the front third of the nose may be blown out and the remainder is swallowed. Particles of 1 - 5 microns in size tend to be carried into the airways where they are caught in the mucus of the trachea and bronchioles. Due to the repeated branching of the passages, air velocity decreases with branching and particles settle out by sedimentation; again being trapped in mucus. Mucus is continuously moved toward the throat by the hair-like cells of the airway and thus these particles are also swallowed.

The small particles of less than 1 micron can reach the alveoli by diffusion where they are removed by:

- ❒ Engulfment by alveolar macrophages which take them to the bronchioles where the macrophages are carried up and out the airways with the mucus, or

- ❒ Engulfment by alveolar macrophages which take them out via the lymph, or

- ❒ Dissolution of the particles or material on the particle which, depending upon the dissolved species, may damage the alveolar walls or pass into the bloodstream or lymph.

For example, water-soluble HCl is normally scrubbed in the upper respiratory system, but its penetration to the deep lung is enhanced if it is attached to small aerosol particles. The soot particles themselves show no crystalline structure and are considered amorphous with properties resembling carbon blacks. Soot particles of respirable size (0.04 - 0.11 microns) from burning PVC do absorb HCl. Studies of this HCl absorbion show that it is easily removable and thus could dissolve in lung moisture to form HCl and cause edema.

Simulated versus actual combustion

A major problem in assessing the toxicity of combustion products is the lack of a standard analytical method. When selecting building or furnishing materials to reduce fire hazards, it would be useful to have information concerning

- ❒ The relative toxicity of such materials
- ❒ The quantity and toxicity of smoke generation, so that the threat of fire can be reduced, and sufficient escape time given to potential victims.

When the fire does occur:

- ◆ How is the fire fighter at risk?
- ◆ What protective equipment is appropriate?
- ◆ How serious is the long-term occupational hazard?

Among the most commonly used methods to determine thermal breakdown products and their toxicity are those developed by the National Bureau of Standards, the University of Pittsburgh and the American Society for Testing and Materials. These share a common approach, consisting of

- ❒ A combustion system for burning the test sample
- ❒ A chemical analytical system for continuous monitoring of oxygen, carbon dioxide, and carbon monoxide; and the ability to sample other gases for later analysis
- ❒ An animal exposure system

Each method has its particular advantages and limitations, but the principle problems shared by them involve the measurement of acute, not chronic, toxicity. This is of particular interest to the firefighter whose exposure is both acute and chronic. As will be discussed later the occupational hazards for this group appear to be directly related to the number of fires fought; this information on the effects of long-term, repeated exposure are important. For example, polymeric materials produce benzene or polyaromatic hydrocarbons which have little or no acute effects, whereas these substances have significant potential for producing chronic effects.

Although real fires consist of both nonflaming pyrolysis and flaming combustion, it is reasonable to suppose that if the worst case condition of each mode is reproduced in the laboratory, then an objective comparison of the toxicity of different materials can be made. Certainly the toxicity of the offgases should be assessed individually for each mode.

Certainly a lab-scale test cannot reproduce exposure which would represent all fire conditions. Self-propagating smoldering decomposition in particular is the most difficult to simulate. Besides the evolution of toxic gases, oxygen deficient conditions may also be present. It is difficult to separate the effects of toxic gases and oxygen-deficiency.

The principal problem of reproducing combustion in the laboratory is the complex nature of the fire scenario and of combustion itself. If combustion was complete and oxygen always abundant, fully oxidized gases would be produced, such as carbon dioxide, nitrogen and sulfur oxides. The result might be irritating to the respiratory system, but is probably not likely to be fatal.

Combustion, however, is generally oxygen-limited and incomplete, producing carbon monoxide and potent irritants and toxic gases. The nature of the gases produced depends upon the composition of the combustion mixture. This is a highly material-specific chemical problem since a synthetic product usually includes additives such as plasticizers, stabilizers, flame retardants, cross-linking agents, fillers and blowing agents.

During a fire involving synthetic materials, the solid fuel evolves volatile gases which then burn. Heat decomposes the material at the surface, generating volatile fuel which burns to generate heat which generates more volatile fuel -- and thus the flammability cycle occurs. Thus the combustion zone consists of a burning front, a volatile diffusion zone, and a low temperature pyrolysis zone. The resulting combustion products may be smaller (gaseous species) or larger (soot or char) then the parent molecules of the synthetic.

In order to pass flammability tests, many materials require the addition of a fire retardant. The retardant may break down with heat to release toxics, although it doesn't burn. When comparing the toxicity of the fire-retarded versus nonfire-retarded material, one must weigh the risks of reduced ignition and flame spread versus smoke-induced toxicity.

For example, in a lab-scale combustion test, a new nonfire-retarded polyurethane foam produced sufficient carbon monoxide to yield a carboxyhemoglobin level between 28 - 33% in the test animal. The same foam with a fire-retardant added, (in this case, 0,0-diethyl-N,N-bis-(2-hydroxyethyl-phosphate)) caused grand mal seizures and death in the test animals from a toxic combustion product (4-ethyl-1-1-phospha-2,6,7-trioxabicyclo[2,2,2.]octane-1-oxide). Compounds like this one have caused seizures at concentrations of under 7 ppm -- levels which could go undetected by gas chromatography in complex smoke mixtures. This case illustrates the reason for using animal bioassays, not just chemical analysis, of combustion products.

IV

OCCUPATIONAL HEALTH HAZARDS OF FIREFIGHTERS AND THEIR RELATION TO THE THERMAL BREAKDOWN PRODUCTS OF SYNTHETICS

Many epidemiological studies of firefighters show a "healthy worker" effect. This is not surprising and may reflect selectivity in the recruitment process for greater physical strength, endurance and pulmonary reserve (also higher initial degree of pulmonary function); as well as in job assignment and retirements.

Thus, firefighters, at the time of retirement, may have impaired ventilatory capacity, but this may not be listed as the cause of death on subsequent death certificates. Early retirees tend to have greater prevalence of chronic bronchitis, loose cough and impairment of lung function.

Workers with higher pulmonary function tend to have more adverse fire exposures since healthier firefighters tend to stay on the job while others seek less active duty, transfer, promotion, retirement or support functions. Therefore, health problems may be underestimated.

Occupational health hazards can be divided into acute and chronic -- repeated acute episodes could eventually lead to a chronic conditions.

ACUTE HEALTH HAZARDS OF FIREFIGHTERS

Acute symptoms usually involve:

sore throat	dyspnea (shortness of breath)
chest tightness	wheeze
nasal discharge	cough
increased sputum production	nausea
headache	dizziness
palpitations	muscle pain

Irritancy of gases may provide a warning mechanism to prevent voluntary acute exposure. However, for fire victims such warnings may not be useful since carbon monoxide inhalation may result in loss of attention and failure to respond to stimuli. For example, a victim detecting the sharp odor of hydrogen chloride may not actually be able to walk away and is thus susceptible to HCl effects.

Moreover, in a sealed environment such a as centrally heated house, office, high-rise, or hospital, the fumes from plastic decomposition could kill even if the fire was fought successfully. It takes a sleeping person 12-15 minutes to awaken, react to danger and save him/herself; therefore escape from these fumes may be impossible.

Acute hazards from thermal decomposition products include:

- ❖ Asphyxiation from carbon monoxide and dioxide. A blood COHb of 65% is generally fatal due to depression of respiration and circulation. Death has occurred from lower COHb levels and tends to involve acute myocardial hypoxia which causes ventricular fibrillation when a severe pre-existing coronary vascular disease was present.

- ❖ Irritation of mucous membranes and upper respiratory tract from hydrogen chloride, hydrogen bromide, ammonia, acrolein and aldehydes.

- ❖ Polymer fume fever, particularly from PTFE or teflon decomposition products. After a latent interval, symptoms include a sense of oppression or discomfort in the chest (rather than pain) which may or may not be accompanied by a dry cough. After a few hours, a gradual increase in body temperature (not exceeding 104°F) and pulse rate occur, followed generally by chills and sweating. Other symptoms may include joint pains, headache, nausea, weakness and shortness of breath. Recovery is fairly rapid and usually complete within two days. Individuals with a history of cigarette smoking appear to have worse symptoms.

- ❖ Chemical pneumonia from chlorine, bromine, nitrogen oxides, sulfur oxides, hydrogen fluoride, carboxy fluoride, carbonyl fluoride, phosgene, occurs when these irritant gases are absorbed onto small particles and inhaled into the deep lung. Chemical pneumonia has complicated the medical treatment of people exposed to combustion products. Carbon monoxide does not cause serious pulmonary injury, so that before the introduction of massive fire loads of plastics capable of thermal decomposition, fire surgeons would consider a carbon monoxide victim safe after four hours from the time of exposure. However, the edema arising from chemical exposure tends to involve a delay of 1 - 6 hours or more after exposure before the onset of severe respiratory symptoms. Delays of 24 hours before onset of symptoms are well-known; treatment is most effective when provided during this latent period. It may be too late to prevent severe damage or death if

difficult breathing, cyanosis or other severe symptoms appear without medical intervention.

- Lung function or capacity decreases; such as acute bronchoconstriction. This has been measured in firefighters within two hours of firefighting and can last up to 18 hours after a fire. This does not appear to be related to any hyperresponsiveness of individuals' airways. It occurs in home and hotel fires involving mattresses and room contents; it does not have to involve only exotic chemical fires. Exhaled carbon monoxide measurements indicate a blood carboxyhemoglobin level of 17 - 22% -- clinically significant carbon monoxide intoxication. This airway response can be induced in people exposed to chemicals causing airway injury and inflammation. Reduced lung function is associated with prolonged mucous production and general malaise after a fire exposure, especially in smokers (loose cough). The lung appears to recover from acute episodes, such as being overcome, which may require oxygen or hospitalization for smoke inhalation. These do not necessarily lead to chronic decreased lung function. Lung function loss seems to occur if acute episodes also involve cough or eye irritation. Changes in FEV upon second exposure to smoke in the same tour of duty are greater when smoke exposure was heavy at the previous fire. A respirator (demand type) is not protective if put on after exposure or removed before safe to do so.

- Heart problems can occur due to irritant gases. Chlorine affects the myocardium more rapidly and severely than carbon monoxide. Hydrogen chloride is also a potent myocardial irritant (worse in conjunction with pulmonary edema) and it can interfere with oxygen supply to coronary arteries and lead to sudden death syndrome. A clinical study of firefighters exposed to PVC decompositon products showed, within a few hours after exposure, extra systoles, cardiac arrhythmia and premature ventricular contractions. Some had a recurrence of extra systoles throughout the year following exposure. A history of cigarette smoking did not appear to affect the severity of these symptoms.

- Heart and circulatory problems due to carbon monoxide. Some firefighters show normal arteries, but also indicate direct damage to the left ventricular muscle, which may reflect frequent CO exposures and the resulting limited oxygen delivery to the heart. Studies have shown that fires with heavy, noxious smoke have CO levels of 100 ppm and can reach as high as 3000 ppm; only in a few instances was

heavy smoke accompanied by minimal CO. CO levels remained elevated after the fire had been "knocked down" but was still smoldering, before overhaul had started. Carbon monoxide has been shown to cause damage to the endothelial lining of the arteries, leading to accelerated development of atherosclerosis. Strenuous work during firefighting further stresses the heart – pointing to the need for lighter, more efficient models of breathing apparatus'.

CHRONIC HEALTH HAZARDS OF FIREFIGHTERS

Chronic health hazards include:

- ❖ Lung damage, particularly chronic nonspecific nonmalignant respiratory disease or loss of pulmonary function, forced vital capacity (FVC) and forced expiration volume of air (FEV). This may involve annual loss rates that are related to the number of fires fought, not simply the length of employment as a firefighter. These losses may be due to long-term effects of those gases which do deep-lung damage, such as those causing chemical pneumonia. Silica exposure from silicone rubber fires is an example. The problem appears even in studies controlled for smoking, as well as for age and height which also affect lung capacity.

- ❖ Heart problems, particularly coronary heart disease, ischemic heart disease, and arteriosclerosis. These problems may reflect exposure to carbon monoxide, oxygen deficiency, chlorine, or PCBs. PCBs can also affect fat distribution in blood vessels. Carbon monoxide (CO) is suspected of advancing ateriosclerosis and damaging the heart through oxygen deprivation. The myocardium in ateriosclerotic persons is especially susceptible to impaired oxygen release. The body tries to make up for oxygen deficiency by increasing coronary flow; unfortunately ateriosclerotic persons are unable to do so. Arteriosclerotic disease appears highest among workers with few years on the job, possibly because the physical/psychological stress or carbon monoxide quickly affect those most susceptible. Even in healthy firefighters, CO exposure may be followed by disturbances of cardiac rhythm as seen on an electrocardiogram.

- ❖ Cancer: Skin cancer risk appears to increase with the number of years on the job. This may be due to the sun or to soot which can contain polynuclear aromatics, many of which are skin carcinogens. Buccal (mouth), pharyngeal, intestinal, and rectal cancers appear to be associated with fires involving dry cleaning establishments, especially

those using carbon tetrachloride (now discontinued) or perchloroethylene (perc). Other potential cancers could include mesotheliomas linked to asbestos exposure from the destruction of ceilings and walls containing asbestos during firefighting.

- ❖ Leukemia, generally preceded by aplastic anemia, shows significant increase in firefighters over the general population. This may reflect exposure to benzene from plastics combustion since benzene has been found in combustion gas from virtually all household and industrial fires.

- ❖ Chloracne: reported cases may be related to silicone tetrachloride fires or to PCBs from transformer fires.

- ❖ Bone disease or disorders.

- ❖ Nonmalignant digestive disease. Cirrhosis appears to increase with the number of years on the job. While alcohol abuse is a possible cause, this condition has been shown to increase with increased chemical industry employment in the fire district service area.

Case History: *Polyurethane fire*

Thirty-five firefighters responded to a fire involving a factory manufacturing polyurethane foam. Two large storage tanks of toluene diisocyanate (TDI) were damaged in the fire causing massive spillage. After an hour, the smoke assumed a distinctive metallic taste and smell; at this time, the firefighters in the production area put on breathing apparatus'. Valves on the tanks were then discovered to be leaking and not repairable. About 4500 liters of TDI escaped. The firemen later described how their uniforms had become soaked with TDI which dried to leave a white powdery coating of polyurethane. Below are the number of respiratory symptoms present before, during and after the fire:

Present before the fire	cough	12
	sputum	6
Developed during the fire	chest tightness	8
	cough	13
	sputum	2

-continued-

Developed during the first three weeks after the fire	chest tightness	28
	cough	25
	sputum	9
	breathlessness	12
Present at six months	chest tightness	3
	cough	11
	sputum	8
	breathlessness	5
Present at 44 months	chest tightness	2
	cough	20
	sputum	14

Smokers experienced more immediate and delayed coughing symptoms than non- or ex- smokers and also more immediate and delayed chest tightness and breathlessness. Most of them experienced symptoms during the fire and 20 men experienced symptoms for as long as 44 months later. Ventilatory capacity measurements showed a decline in the first six months, but improved after this period.

V

PROTECTION AND PREVENTION

It would appear that the fire scenario is hazardous to firefighters (as well as victims) due to the acute and chronic health effects of the thermal decomposition products of synthetic materials. Firefighters have many health conditions which appear occupationally-related; therefore, personal protective equipment is a must to prevent firefighter exposure.

All fires should be considered potentially toxic.

The term "smoke inhalation" should be abandoned and instead, the more realistic expression "inhalation of toxic combustion products" should be used. Respiratory equipment should be used as a standard procedure, not just when smoke is apparent or when its odor or characteristics change. To maintain this standard, a "mandatory mask rule" should be instituted.

Other protective clothing should be used as needed. Special protective procedures should be maintained during the overhaul phase when firefighters clean up the fire ground since toxic smoke and fumes are still being emitted. Hazardous gases can be retained in heat sinks such as porous building materials, concrete and block foundations or in confined spaces; these are released after a fire. Highly toxic concentrations of hydrogen chloride (HCl) have been found to persist in concrete for one hour after a fire has been extinguished.

When the city of Boston Fire Department adopted a mandatory mask rule, the use of new SCBAs brought about a 52% reduction in smoke inhalation the first year and an 80% reduction over the first two years.

However, there are disadvantages to the use of such equipment besides their limited air/oxygen supply. An SCBA and protective clothing are heavy to carry and so they increase cardiorespiratory strain during heavy exercise and decrease the maximal working capacity. Thus they require individuals who are healthy and in good physical condition. The equipment weight (~25 kg) adds stress to the body under conditions of the existing stress from the fire itself, coupled with the exertion of demolishing doors or walls, climbing ladders/stairs and rescuing people.

In addition, respirators alter the breathing pattern of the user, therefore may disturb respiratory gas exchange and tend to increase psychophysiological stress. There is resistance to inhalation and exhalation, increased by equipment

dead space. Repetitive use of equipment should be limited and separated by rest periods.

Also consider the need for other protective equipment such as impervious or disposal clothes for fires involving PCBs, acid fumes, or unusual chemical/spills where respiratory protection alone is insufficient or the atmosphere would damage the equipment, preventing its proper functioning.

Finally....

Firefighting is a physically demanding occupation which requires sudden exertion without prior warm-up, as well as work under thermal stress and heavy pollution consisting of toxicant gases and particulates. Without use of proper work practices and protective equipment, decomposition of synthetic materials are toxic and could bring about the acute and chronic health effects seen in firefighters.