10 Toxicology of Fire and Smoke[1]

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CONTENTS

10.1 INTRODUCTION

Toxicology of fire and smoke is the study of the adverse health effects caused by exposure to combustion atmospheres. In this chapter, a combustion atmosphere is defined as all of the effluents generated by the thermal decomposition of materials or products regardless of whether that effluent is produced under smoldering, nonflaming, or flaming conditions. The objectives of combustion toxicity research are to identify potentially harmful products from the thermal degradation of materials, to distinguish those materials that produce unusual or more toxic quantities of toxic combustion products, to determine the best measurement methods for the identification of the toxic products and the degree of toxicity, to determine the effect of different fire exposures on the composition of the toxic combustion products, and to establish the physiological effects of such products on living organisms. The ultimate goals of this field of research are to reduce human fire fatalities due to smoke inhalation, to determine effective treatments for survivors, and to prevent unnecessary suffering of fire casualties who have inhaled toxic combustion products.

Seventy-six percent of the people who die in fires die of the inhalation of toxic combustion products, not of burns (Hall and Harwood, 1995). This percentage has been rising by about 1% per year since 1979. Although total deaths in fires are declining, the percentage attributed to smoke inhalation has increased. Levin (1996, 1998), Purser (1988, 1995), Nelson (1995), and the National Research Council (1995) have reviewed various aspects of this subject.

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10.2 FIRE DEATH STATISTICS IN THE UNITED STATES

The latest statistics from the National Fire Protection Association (NFPA) indicate that the fire death rate in the United States was 6.1 times higher (10.1 times more if the deaths from September 11, 2001 are included) than in the United Kingdom, 1.16 times more than in Sweden, 1.3 times less than Japan (or 1.3 times more if the deaths from September 11, 2001, are included), and about the same as that in Canada (Hall, 2003). Although the reasons are still being debated, the number of fire deaths per capita since 1977 has been higher in the United States and Canada than in most of the other industrialized countries outside the former Soviet Union (Hall, 2003). Fire statistics collected by NFPA reported that approximately 1,687,500 fires occurred in the United States in 2002, the latest year for which complete statistics are available (Karter, 2003). Calculated another way, these statistics translate into a fire occurring in the United States every 19 sec, in a property located outside every 38 sec, in a structure every 61 sec, in a residence every 67 sec, and in a motor vehicle every 96 sec. These fires caused approximately 3380 civilian deaths and 18,425 reported injuries in 2002. Excluding the World Trade Center disaster of September 11, 2001, in which 2326 civilian deaths occurred, the number of deaths in 2002 decreased by almost 10% from the preceding year. However, there still was one civilian fire death every 156 min and one fire injury every 28 min. The number for injuries is believed to be less than the actual number, because many injuries are not reported. The property loss due to fires in 2002 is estimated at 10.3 billion dollars and indicates a decrease of 2.2% from the preceding year (again excluding the World Trade Center disaster).

In 2002, residential fires accounted for only 24% of the total fires but were responsible for 79% of all fire deaths and 76% of the reported injuries. Although in the years 1977 through 2002, the number of civilian fire fatalities in homes dropped from 5865 to 2670, fires in homes still cause the greatest concern to the fire community. Statistics show that children under 5 and adults over 65 years of age are the most frequent casualties of residential fires. This is attributed to their inherent difficulties in trying to escape. Statistics also show that males are more likely to die in fires than females. More fires and higher fire death rates occur in the South than any of the other geographical areas of the United States; the geographical region with the next highest fire death rate and number of fires is the Northeast.

One must distinguish between the causes of fires and the causes of fire deaths. The primary causes of residential fires have been shown to be heating and cooking (Federal Emergency Management Agency [FEMA], 1982; Runyan et al.,1992). Lack of central heat and the incorrect use of portable space heaters are two of the reasons given for the high fire and death rates in the South. Heating fires result in the highest property losses, primarily because cooking fires are usually noticed and extinguished before getting out of control. Fire deaths, however, usually result from fires ignited by cigarettes (Hall, 2003; Runyan et al., 1992). The most common fire scenario leading to fire deaths is one in which a person (usually intoxicated) falls asleep in an upholstered chair while smoking (FEMA, 1982). The cigarette falls into a crevice and starts the upholstered chair smoldering. The individual awakes and goes to bed unaware of the danger. The chair can smolder for an extended period (an hour was not unusual in laboratory tests conducted at the National Institute of Standards and Technology [NIST]) before bursting into flames. Once the flaming starts, the smoke will fill the room and escape to the other rooms. It is common to find people who have died of smoke inhalation (not burns) in or near their beds, indicating that their making little or no effort to escape was probably due to absence of warning and incapacitation from the asphyxiants in the smoke. Smoke detectors in this scenario would save many lives. Statistics have shown that working smoke detectors double one's probability of escaping alive (Hall, 2003). A recent problem is that many homes have nonfunctioning smoke detectors because the batteries were removed after a false alarm (usually from smoke produced by a kitchen or wood stove) or because old batteries were never replaced (Hall, 2003).

10.3 GENERATION OF TOXIC GASES IN FIRES: ADVERSE EFFECTS OF PARTICULATES

The adverse effects from smoke inhalation are believed to result mainly from toxic gas exposures, although the role of the particulates alone and in combination with fire gases needs further investigation. The importance, therefore, of determining the identities and concentrations of toxic gases produced from materials thermally decomposed under various fire conditions is evident. In addition, the increased variety of plastics in buildings and homes has raised the issue of whether synthetic materials may produce extremely or unusually toxic[2] combustion products. In 1975, the journal *Science* documented a case in which an experimental rigid polyurethane foam containing a fire retardant produced a very unusual toxic combustion product identified as 4-ethyl-1-phospha-2,6,7-trioxabicyclo[2.2.2]octane-1-oxide (commonly referred to as a bicyclic phosphate ester) (Petajan et al., 1975). Bicyclic phosphate compounds have been shown to cause seizures at very low concentrations. Based on these test results, this fire-retarded rigid polyurethane foam never became commercially available. To a large extent, however, it was this case that generated the burgeoning interest in the field of combustion toxicology and the widespread concern about the potential formation of "supertoxicants." Although research since the 1970s has shown that this concern is largely unfounded, the bicyclophosphate ester case and at least one other product that generated extremely toxic combustion products have indicated the need to test new formulations or materials containing new combinations of compounds to ensure that extremely or unusually toxic products are not generated. In November 2000, John Hall, Jr., indicated that the field of combustion toxicology is not producing much new data (Hall, 2000). He concluded that most of the current international debate in this area is over the interpretation and application of past research.

The gas composition of smoke depends on the chemical composition, the molecular structure and polymer formulation of the burning material, which may include a variety of additives, plasticizers, stabilizers, flame-retardants, cross-linking agents, fillers, and blowing agents. In addition, the conditions of thermal degradation, e.g., temperature, oxygen availability, and ventilation, will affect the nature of the combustion atmosphere. In a series of literature reviews by National Institutes of Standards and Technology (NIST) on the generation of combustion products and the combustion product toxicity from seven plastics (acrylonitrile-butadiene-styrenes [ABS], nylons, polyesters, polyethylenes, polystyrenes, poly(vinyl chlorides) [PVC], and rigid polyurethane foams) commonly found in materials and products, and decomposed under various thermal and atmospheric conditions, over 400 different decomposition products were noted (Rutkowski and Levin, 1986; Braun and Levin, 1986, 1987; Gurman et al., 1987; Huggett and Levin, 1987; Levin, 1987; Paabo and Levin, 1987a, 1987b). At about the same time, the Consumer Product Safety Commission reviewed the combustion products and the toxicity of the combustion products generated from acrylics, phenolics, polypropylene, and flexible polyurethane foam (Johnston et al., 1988a, 1988b; Purohit and Orzel, 1988; Orzel et al., 1989) and the National Academy of Sciences did the same type of literature review on an additional ten plastics, namely, acetal, aramid, cellulose acetate butyrate, epoxy resins, melamine-formaldehyde, polybutylene, polycarbonate, polyphenylene oxide, polytetrafluoroethylene, and urea-formaldehyde (National Research Council, 1987). Many of the combustion products were common to more than one plastic. In addition, many other combustion products probably exist that were not detected. At this time, the toxicity of most of these individual compounds is not known and little has been done to tackle the enormous problem of determining the toxicity of combinations of these compounds. The lack of detection of a specific combustion product from a material may only mean that the particular analytical techniques used were not suitable to detect that compound or that the investigator did

[2] In this chapter, the phrase "extremely toxic" is a relative term indicating that the effluent from the thermal decomposition of very small quantities of a material has been noted to cause death of experimental animals (usually rats or mice) under controlled laboratory conditions. "Unusually toxic" indicates that the toxic effect can not be totally attributable to the combustion gases (either singly or in combination) that are normally considered the main toxicants.

not specifically analyze the material for that combustion product. A limited amount of rodent testing becomes important to ensure that an unsuspected and therefore undetected toxic by-product has not formed or that synergism or antagonism of the generated products has not occurred.

Because the number of compounds one can reasonably analyze in any one test is limited, knowledge of the chemical composition, molecular structure, and formulation of the polymer can be used to provide some indication of the main gaseous products that may or may not be generated under specified experimental conditions. However, one needs to be cautious when predicting the combustion products from generic materials of unknown formulations. For example, one would expect nitrogen-containing materials (e.g., ABS, nylons, rigid and flexible polyurethanes) to produce hydrogen cyanide (HCN) and not expect HCN from a material like PVC. However, PVC containing zinc ferrocyanide[3] (an additive that was tested as a smoke suppressant) or a vinyl chloride-vinylidene chloride copolymer was found to generate HCN. In a similar fashion, based on the chemical composition, PVC is the only one of the seven plastics reviewed by NIST that would be expected to generate chlorinated combustion products. However, widespread usage of halogenated fire retardants in plastic formulations makes predicting the materials that will produce halogenated products extremely difficult. In addition, many of the fire-extinguishing agents contain halogens and can interact with the other combustion products in unpredictable ways when they are used to extinguish fires.

Temperature also plays an important role in influencing the production of decomposition products. In general, as the temperature and thus the rate of decomposition increase, the quantity of the more complex compounds and heavier hydrocarbons decreases and the concentrations of carbon monoxide (CO), carbon dioxide (CO_2), and nitrogen dioxide (NO_2) increase. The generation of HCN has also been shown to increase as a function of temperature. Another example is hydrogen chloride (HCl), the detection of which begins when stabilized PVC is heated to approximately 200°C; rapid dehydrochlorination then occurs at about 300°C (Huggett and Levin, 1987). On the other hand, more acrolein was generated from polyethylene under lower temperature, nonflaming conditions than under higher temperature flaming conditions (Paabo and Levin, 1987a).

As mentioned earlier, more work is needed to examine the adverse effects of the particulate matter that is also produced when materials are thermally decomposed. Examination of the smoke particulate and condensable matter is important for several reasons. First, many of the thermal degradation products may condense or be adsorbed by the soot particles and be transported along with the smoke into the body. Hydrogen chloride is one example of a compound that may be transported in such a fashion or can form a corrosive acid mist in moist air, such as air found in a person's lungs. One study of the particulate matter, which formed during the smoldering decomposition of rigid polyurethane foam, showed that many of the compounds detected in the soot fraction were not found in the volatile fraction (Joseph and Browner, 1980; Paabo and Levin, 1987b). Free radicals, which form in fires and are of toxicological concern due to their high reactivity (Westerberg et al., 1982; Lowry et al., 1985a, 1985b), are usually considered to have very short life spans; however, if adsorbed onto soot particles, their lifetimes can be considerably longer, and if the soot particle is the correct size, they can be inhaled deep into an individual's respiratory system. In addition, the particulate matter may interfere with the escape and rescue of individuals by causing the obscuration of vision, eye irritation (the eyes clamp shut and the victim is unable to see), and upper respiratory tract distress. An extreme case indicating the adverse effect of particulates was noted in experiments conducted at NIST. Rats exposed for 30 min to the smoke from flaming polystyrene died during the exposures and the concentration of CO in the blood, even in combination with CO_2, was too low to account for the deaths (Levin et al., 1987a). Pathological examination of these rats showed that their respiratory passages were completely blocked by soot and that suffocation was the likely cause of death (NIST, unpublished data).

Particulates have also been implicated in the toxicity of polytetrafluoroethylene (PTFE). Using the National Bureau of Standards (NBS) Cup Furnace Test Method (Figures 10.1 and 10.2; see Section 10.7), the combustion products of PTFE were found to be 300 times more toxic than other

[3] This material was never made commercially available after toxicity testing indicated that its combustion products produced very rapid deaths of experimental animals (rats).

materials decomposed in a similar fashion (Levin et al., 1982, 1983). Further studies showed that inhalation of the combustion products of PTFE decomposed in the NBS cup furnace caused focal hemorrhages, edema, and fibrin deposition in the lungs of rats (Zook et al., 1983). These same studies showed that, with time, focal interstitial thickenings developed because of hypertrophy and hyperplasia of alveolar cells and that macrophages accumulated in the alveoli. Thrombosis of pulmonary capillaries and disseminated intravascular coagulation also occurred. Renal infarcts were common. The exact reason for the unusual toxicity of PTFE's combustion products is unknown and has generated a great deal of interest (Alarie and Stock, 1984a; Birky, 1984; Williams and Clarke, 1983, 1984). The specific heating conditions of the NBS Cup Furnace Test Method[4] (Williams and Clarke, 1983; Williams et al., 1987) and the particulate fraction from PTFE have been cited (Clarke et al., 1990; Baker and Kaiser, 1991; Lee and Seidel, 1991). However, thermal decomposition in the University of Pittsburgh I (U. Pitt I) Test Method also produced their highest toxicity classification of "much more toxic than wood" (Alarie and Anderson, 1981; Alarie and Stock, 1984a). The other classifications of the U. Pitt I method are "less toxic than wood", "as toxic as wood," and "more toxic than wood." Decomposition in the German Din System produced a toxicity at least 10 times more toxic than wood (Purser, 1992). Full-scale fire tests seem to implicate hydrogen fluoride or carbonyl fluoride as the toxic agents (Clarke et al., 1992). Another compound that was suspected is perfluoroisobutylene (PFIB), which is toxic at extremely low levels and can be generated during the combustion of polymerized fluorocarbon compounds (e.g., PTFE) (Wang et al., 2001). The Occupational Safety and Health Administration (OSHA) reports that the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV) is 0.01 ppm (0.082 mg/m^3) (www.osha.gov/dts/chemicalsampling/data/CH_260750.html, accessed April 15, 2004). However, analysis of the combustion atmospheres from PTFE at NIST did not detect any PFIB. An extensive review on the toxicology of soot was published in 1983 (Barfknecht, 1983). This review discusses the effect of soot on the induction of respiratory diseases and carcinogenicity.

10.4 TOXIC POTENCY VERSUS FIRE HAZARD VERSUS FIRE RISK

Death in a fire may be caused by:

1. Carbon monoxide (CO)
2. Toxic gases in addition to CO
3. Oxygen (O_2) at levels too low to sustain life
4. Incapacitation—either physical (inability to escape) or mental (incorrect decision making)
5. Bodily burns from flame contact
6. Very high air temperatures
7. Smoke density or irritants in smoke that affect vision and interfere with ability to escape
8. Psychological effects (e.g., fear, shock, and panic)
9. Physical insults (e.g., building or ceiling collapses, broken bones from jumping from upper floors)

Research in the field of combustion toxicology is primarily concerned with items 1 through 4, all of which are related to the toxic potency of the fire gas effluent. Toxic potency as applied to inhalation of smoke or its component gases is defined by American Society for Testing and Materials (ASTM) as "A quantitative expression relating concentration (of smoke or combustion gases) and exposure time to a particular degree of adverse physiological response, for example, death on exposure of humans or animals"(ASTM, 2004). This definition is followed by a discussion, which states, "The toxic potency of smoke from any material, product, or assembly is related to the composition of that smoke which, in turn, is dependent upon the conditions under which the smoke is generated." One should add that

[4] Further explanation of the combustion toxicity test methods are in Section 10.7.

the LC_{50}[5] is a common end point used in laboratories to assess toxic potency. In the comparison of the toxic potencies of different compounds or materials, the lower the LC_{50} (i.e., the smaller the amount of material necessary to reach the toxic endpoint), the more toxic the material is.

A toxicity assessment based on lethality due to toxic gases is only part of the total fire hazard that needs to be evaluated, especially when one is making choices as to the best material for a specific end use. ASTM defines "fire hazard" as the potential for harm associated with fire (ASTM, 2004). The discussion that follows this definition states, "A fire may pose one or more types of hazard to people, animals, or property. These hazards are associated with the environment and with a number of fire-test-response characteristics of materials, products, or assemblies including but not limited to ease of ignition, flame spread, rate of heat release, smoke generation and obscuration, toxicity of combustion products, and ease of extinguishment." Other factors that need to be evaluated when considering a material for use in a given situation include the quantity of material needed, its configuration, the proximity of other combustibles, the volume of the compartments to which the combustion products may spread, the ventilation conditions, the ignition and combustion properties of the material and other materials present, the presence of ignition sources, the presence of fire protection systems, the number and type of occupants, and the time necessary to escape.

"Fire risk" is defined as "An estimation of expected fire loss that combines the potential for harm in various fire scenarios that can occur with the probabilities of occurrence of those scenarios" (ASTM, 2004). The discussion following the definition of fire risk states, "Risk may be defined as the probability of having a certain type of fire, where the type of fire may be defined in whole or in part by the degree of potential harm associated with it, or as potential for harm weighted by associated probabilities. However it is defined, no risk scale implies a single value of acceptable risk. Different individuals presented with the same risk situation may have different opinions on its acceptability." A simple way to explain the difference between fire hazard and fire risk is to compare the fire with sky diving, a very hazardous sport; however, if one never goes sky diving, no risk is incurred.

10.5 TOXICITY ASSESSMENT: ANIMAL EXPOSURES

In most combustion toxicology experiments, the biological end point has been lethality or incapacitation of experimental animals, usually rats or mice. Incapacitation (sublethal effects) in a fire can be as perilous as lethality if an individual becomes incapable of correct decision making or physically unable to move. Under these circumstances, the ability to escape will be lost and death will occur unless the individual is rescued. Therefore, many fire scientists are concerned with the levels of combustion products or amounts of materials that, when combusted, will cause incapacitation. However, an incapacitation model for use in laboratory testing has been especially difficult to develop. Most of the tests for incapacitation that have been designed are based on the physical-motor capability of an experimental animal to perform some task (e.g., running in a motorized wheel, jumping onto a pole or lifting a paw to escape a shock, running in a maze, or pushing the correct lever to open a door to escape an irritating atmosphere) (Purser and Berrill, 1983; Kaplan and Hartzell, 1984; Kaplan et al., 1985). The concentration of toxic combustion products that cause the loss of these types of physical-motor capabilities is usually close to the concentration that is lethal and does not usually add much additional information. Other attempts to measure incapacitation have included the examination of neurological end points (e.g., measuring the increased number of errors by humans doing mathematical problems while exposed to low levels of CO [Arthur Callahan, private communication] or exposing rats and pigeons to a complete neurobehavioral battery of 25 tests) (Richie et al., 1995).

Whether one needs to examine incapacitation or lethality depends on the problem one is trying to solve. To determine the best material for a particular end use application, the lethality end point has

[5] The LC_{50} value is the result of a statistical calculation based on multiple experiments, each with multiple animals, and indicates the concentration at which 50% of the experimental animals exposed for a specific length of time would be expected to die either during the exposure time or the postexposure observation period.

proven to be more definitive and will flag the materials that produce extremely toxic combustion products better than an incapacitation end point. There are at least two reasons for this. (1) Incapacitation is only measured during the exposure that is usually 30 min or less, but lethality can also occur during the postexposure observation period, which can be two weeks or longer. A material that only causes delayed effects during the postexposure period (e.g., a material that generates HCl) can thus have an LC_{50} value that is lower (more toxic) than the incapacitation EC_{50}[6] value (i.e., the amount of thermally decomposed material necessary to cause postexposure deaths is less than the amount needed to cause incapacitation during the exposure). (2) In many cases in which the combustion products contain high concentrations of irritant gases, the animals would only appear to be incapacitated (i.e., they would stop responding to the incapacitation test indicator because of the high irritant quality of the smoke), but when removed from the combustion atmosphere, they would immediately start responding normally.

10.6 TOXICITY ASSESSMENT: PREDICTIVE MODELS

In the 1970s, there were essentially two experimental strategies to examine the issues raised by the field of combustion toxicology: (1) the analytical chemical method and (2) the animal exposure approach. In the analytical chemical method, investigators thermally decomposed materials under different experimental conditions and tried to determine every combustion product that was generated (Levin, 1987; ASTM, 2001). This approach generated long lists of compounds. The toxicity of most of these individual compounds was unknown and the concept of examining the toxicity of all the various combinations of compounds was and still is considered an impossible task. An additional problem with the analytical method was that, as mentioned earlier, one could not be certain that every toxic product was detected and identified. This approach enabled one to identify many of the multiple products that were generated but not know the toxic potency of all the identified compounds, especially when combined.

In the animal exposure approach, the animals (usually rats or mice) serve as indicators of the degree of toxicity of the combustion atmospheres (Kaplan et al., 1983; Levin et al., 1982, 1991b, 1992a). The materials of concern are thermally decomposed under different combustion conditions and the animals are exposed to the combined particulate and gaseous effluent. Multiple animal experiments (each with multiple animals) with different concentrations of material are conducted to determine an EC_{50} (incapacitation) or an LC_{50} (lethality) for a specific set of combustion conditions. Each material would then have a particular EC_{50} or an LC_{50} value that can be used to compare the toxicities of different materials decomposed under the same conditions. The lower the EC_{50} or LC_{50}, the more toxic the combustion products from that material. In this approach, one knows the relative toxicity of a material as compared with another material but does not know which of the toxic gases are responsible for the adverse effects.

In the 1980s, investigators began examining the possibility of combining the analytical chemical method with the animal exposure approach to develop empirical mathematical models to predict the toxicity (Levin, 1996; Levin et al., 1985a, 1987a, 1987b). These predictions were based on actual experiments with animals and their response to each of the main toxic combustion gases, CO, CO_2, low O_2, HCN, NO_2, HCl, and hydrogen bromide (HBr) and various combinations of these gases. The advantages of these predictive approaches are (1) the number of test animals is minimized by predicting the toxic potency from a limited chemical analysis of the smoke; (2) smoke may be produced under conditions that simulate any fire scenario of concern; (3) fewer tests are needed, thereby reducing the overall cost of the testing; and (4) information is obtained on both the toxic potency of the smoke (based on the mass of material burned) and the responsible gases (based on the primary toxic gases in the mixture). The prediction is checked with one or two animal tests to ensure that an unexpected gas or toxic combination has not formed. The results of using these empirical mathematical models indicated that, in most cases, one could predict the toxic potency of a combustion atmosphere with the main toxic gases and did not need to worry about the effects of minor or more obscure gases.

[6] The definition of the EC_{50} is essentially the same as that of the LC_{50} except incapacitation rather than lethality is the end point and incapacitation is monitored only during the exposure and not during the postexposure period.

10.6.1 Primary Toxic Combustion Gases

Complete combustion of a polymer containing carbon, hydrogen, and oxygen in an atmosphere with sufficient O_2 yields CO_2 and H_2O. It is during incomplete combustion under various atmospheric conditions in either flaming or nonflaming modes that compounds of greater toxicological concern are generated. When O_2 is limited, the primary gases formed during the combustion of most materials are CO, CO_2, and H_2O. If the materials contain nitrogen, HCN and NO_2, two principal thermo-oxidative products of toxicological concern, are also likely to be generated. Halogenated or flame-retarded materials generally produce HCl or HBr. Other commonly found fire gases include nitrogen oxides (NO_x), ammonia (NH_3), hydrogen sulfide (H_2S), sulfur dioxide (SO_2), and fluorine compounds. One also needs to consider that in fire situations, O_2 levels drop and exposure to low O_2 atmospheres will have additional adverse physiological effects. Some of these toxic combustion gases (e.g., CO, HCN, low O_2) produce immediate asphyxiant symptoms, whereas others (e.g., HCl, HBr, NO_2) fall into an irritant category and produce symptoms following the exposures.

10.6.2 The N-Gas Models

The N-Gas Models for predicting smoke toxicity were founded on the hypothesis that a small number ("N") of gases in the smoke account for a large percentage of the observed toxic potency. These predictive models were based on an extensive series of experiments conducted at NIST on the toxicological interactions of the primary gases found in fires (Levin, 1996; Levin et al., 1985a, 1987a, 1987b, 1989c, 1990a, 1991a, 1995). Both the individual gases and complex mixtures of these gases were examined. To use these models, materials are thermally decomposed by using a bench-scale method that simulates realistic fire conditions, the concentrations of the primary fire gases—CO, CO_2, low O_2, HCN, HCl, HBr, and NO_2—are measured, and the toxicity of the smoke is predicted by using the appropriate N-Gas Model. The predicted toxic potency is checked with a small number of animal (Fischer 344 male rats) tests to ensure that an unanticipated toxic gas was not generated or an unexpected toxicological effect (e.g., synergism or antagonism) did not occur. The results indicate whether the smoke from a material or product is extremely toxic (based on mass consumed at the predicted toxic level) or unusually toxic (the toxicity can not be explained by the combined measured gases). These models have been shown to correctly predict the toxicity in both bench-scale laboratory tests and full-scale room burns of a variety of materials of widely differing characteristics chosen to challenge the system (Levin et al., 1987a; Babrauskas et al., 1991). The six-gas model (without NO_2) is now included in two national toxicity test method standards—ASTM E1678-02 approved by the American Society for Testing and Materials (ASTM, 2002) and NFPA 269 approved by the National Fire Protection Association (2003). It is also included in an international standard (ISO 13344:1996) that was approved by 16 member countries of the International Organization for Standardization (ISO), Technical Committee 92 (TC92). All three of these standards were first published in 1996.

The objectives of developing the N-Gas Models were:

- To establish the extent to which the toxicity of a material's combustion products could be explained and predicted by the interaction of the major toxic gases generated from that material in the laboratory or whether minor and more obscure combustion gases needed to be considered.
- To develop a bioanalytical screening test and a mathematical model that would predict whether a material would produce extremely toxic or unusually toxic combustion products.
- To predict the occupant response from the concentrations of primary toxic gases present in the environment and the time of exposure.
- To provide data for use in computer models designed to predict the hazard that people will experience under various fire scenarios.

10.6.2.1 The Six-Gas N-Gas Model

The six-gas model (see Equation [10.1]) was based on studies at NIST on the toxicological interactions of six gases, CO, CO_2, HCN, low O_2 concentrations, HCl, and HBr. First, individual gases in air were tested to determine the concentrations necessary to cause 50% of the laboratory test animals (Fischer 344 male rats) to die either during the exposure (within exposure LC_{50}) or during the exposure plus a 14-day postexposure observation period (within plus postexposure LC_{50}). The studies on HCl and HBr were conducted at Southwest Research Institute (SwRI) under a grant from NIST (Hartzell et al., 1990). Similar measurements for various combinations of these gases indicated whether the toxicity of the mixtures of gases was additive, synergistic, or antagonistic.

Based on these empirical results, the following six-gas N-Gas model was developed:

$$\frac{m[CO]}{[CO_2]-b}+\frac{[HCN]}{LC_{50}HCN}+\frac{21-[O_2]}{21-LC_{50}O_2}+\frac{[HCl]}{LC_{50}HCl}+\frac{[HBr]}{LC_{50}HBr}=\text{N-Gas Value} \qquad (10.1)$$

where the numbers in brackets indicate the time-integrated average atmospheric concentrations during a 30-min exposure period [(ppm × min)/min or for O_2 (% × min)/min] (Levin, 1996, 1998; Levin et al., 1991b, 1995; International Organization for Standardization, 1996; ASTM, 2002; NFPA, 2003). The other terms are defined in the following paragraphs.

Under the experimental conditions used at NIST and with Fischer 344 male rats, the 30-min LC_{50} value of CO_2 is 47% (470,000 ppm or 846 g/m^3) with 95% confidence limits of 43 to 51% (Levin et al., 1987b,1989c, 1991a)[7]. No deaths occurred in rats exposed to 26% CO_2 for 30 min. In a real fire, the highest theoretically possible concentration of CO_2 is 21%, a concentration that could only occur if all the atmospheric O_2 were converted to CO_2, a highly improbable event. Therefore, CO_2 concentrations generated in fires are not lethal. However, CO_2 is a respiratory stimulant causing an increase in both respiratory rate and tidal volume. It also increases the acidosis of the blood. When combined with any of the other tested gases, CO_2 has a synergistic toxicological effect, i.e., the toxicity of the other gases is increased in the presence of CO_2 (Table 10.1). Empirically, however, we found that the effect of the CO_2 can only be added into the N-Gas equations once. Therefore, we included the CO_2 effect with the CO factor because we had more data on the combined effect of CO and CO_2, and CO is the toxicant most likely to be present in all fires. The results on the synergistic effect of CO_2 on CO indicated that, as the concentration of CO_2 increases (up to 5%), the toxicity of CO increases. Above 5% CO_2, the toxicity of CO starts to revert back toward the toxicity of the CO by itself. The terms m and b in Equation (10.1) define this synergistic interaction and equal −18 and 122,000, respectively, if the CO_2 concentrations are 5% or less. For studies in which the CO_2 concentrations are above 5%, m and b equal 23 and –38,600, respectively.

In rats, the 30-min LC_{50} for CO is 6600 ppm (7560 mg/m^3) and with 5% CO_2, this value drops to 3900 ppm (4470 mg/m^3). Exposure to CO in air only produced deaths during the actual exposures and not in the postexposure observation period; however, exposures to CO plus CO_2 also caused deaths in the postexposure period. Carbon monoxide is a colorless, odorless, tasteless, and nonirritating poisonous gas. The toxicity of CO comes from its binding to the hemoglobin in red blood cells and the formation of carboxyhemoglobin (COHb). The presence of CO on the hemoglobin molecule prevents the binding of O_2 to hemoglobin (O_2Hb) and results in hypoxia in the exposed individual. Because the binding affinity of hemoglobin for CO is 210 times greater than its affinity for O_2, only 0.1% CO (1000 ppm) (1150 mg/m^3) is needed to compete equally with O_2, which is normally present at 20.9% in air (20.9% ÷ 210 ≈ 0.1%). Thus, only 1000 ppm of CO in the atmosphere is enough to generate 50% COHb, a value commonly quoted (but not necessarily

[7] *Caution:* The values given for use in Equations (10.1) and (10.3) depend on the test protocol, on the source of test animals, and on the rat strain. It is important to verify these values whenever different conditions prevail and, if necessary, to determine the values that would be applicable under the new conditions.

TABLE 10.1 Synergistic Effects of CO_2

Gas[a]	LC_{50} Values[b]	
	Single Gas	With 5% CO_2
CO_2	470,000 ppm (846 g/m³)	—
CO	6600 ppm (7560 mg/m³)	3900 ppm (4470 mg/m³)
NO_2	200 ppm (376 mg/m³)	90 ppm (169 mg/m³)
O_2	5.4%	6.4%

[a] All gases were mixed in air.

[b] Thirty-minute exposures of Fischer 344 rats. Deaths occurred during and after the exposure.

proven) as the concentration that is lethal to humans. The time to get to 50% COHb at 1000 ppm CO would be longer than 30 min.

The LC_{50} value of HCN is 200 ppm (221 mg/m³) for 30-min exposures or 150 ppm (166 mg/m³) for 30-min exposures plus the postexposure observation period. HCN caused deaths both during and after the exposures.

The 30-min LC_{50} of O_2 is 5.4%, which is included in the model by subtracting the combustion atmospheric O_2 concentration from the normal concentration of O_2 in air, i.e., 21%.

The LC_{50} values of HCl or HBr for 30-min exposures plus postexposure times are 3700 ppm (5450 mg/m³) and 3000 ppm (9940 mg/m³), respectively. HCl and HBr at levels found in fires only cause postexposure effects.

The pure and mixed gas studies showed that if the value of Equation (10.1) is 1.1 ± 0.2, then some fraction of the test animals would die. Below 0.9, no deaths would be expected and above 1.3, all the animals would be expected to die. Because the concentration–response curves for animal lethalities from smoke are very steep, it is assumed that if some percentage (not 0 or 100%) of the animals die, the experimental loading is close to the predicted LC_{50} value. Results using this method show good agreement (deaths of some of the animals when the N-gas values were above 0.9) and the good predictability of this approach.

This model can be used to predict deaths that will occur only during the fire exposure or deaths during and after the fire. To predict the deaths that would occur both during and after the exposures, Equation (10.1) is used as presented. To predict deaths only during the exposures, HCl and HBr, which only have postexposure effects, should not be included in Equation (10.1). In small-scale laboratory tests and full-scale room burns, Equation (10.1) was used successfully to predict the deaths during and after exposures to numerous materials (Braun et al., 1990; Babrauskas et al., 1991). In the case of PVC, the model correctly predicted the results as long as the HCl was greater than 1000 ppm (1470 mg/m³); therefore, it is possible that HCl concentrations smaller than 1000 ppm may not have any observable effect on the model even in the postexposure period. More experiments are necessary to show whether a true toxic threshold for HCl does exist.

Although most of the work at NIST concentrated on deaths during or after 30-min exposures, the LC_{50}s of many of these gases both singly and mixed were determined at times ranging from 1 to 60 min and in all the cases examined, the predictive capability of Equation (10.1) holds if the LC_{50}s for the other times are substituted into the equation.

10.6.2.2 The Seven-Gas Model: Addition of NO_2 to the N-Gas Model

Nitrogen dioxide is an irritant gas that will cause lachrimation, coughing, respiratory distress, increases in methemoglobin levels, and lung edema (Lee, 1980). Single brief exposures to less than lethal concentrations can cause lung damage, emphysema, or interstitial fibrosis. Low levels have

been alleged to increase one's susceptibility to respiratory infections and aggravate one's reactions to allergens. Impairment of dark adaptation has also been noted. Delayed serious effects can be observed as late as two to three weeks following exposures. In the lungs, NO_2 forms both nitric (HNO_3) and nitrous (HNO_2) acids, which are probably responsible for the damage to the lung cells and connective tissue.

In fires, NO_2 may arise from atmospheric nitrogen fixation, a reaction that is material independent, or from the oxidation of nitrogen from nitrogen-containing materials. To examine the generation of NO_2 from nitrogen fixation, a small study was undertaken at NIST. In two full-scale fires of rooms in which the main source of fuel was polystyrene-covered walls, only low levels of NO_x (10 and 25 ppm, respectively) were found, indicating little nitrogen fixation under these conditions (Levin et al., 1989c). A real example of burning nitrogen-containing materials was the 1929 Cleveland Clinic fire in which 50,000 nitrocellulose x-ray films were consumed (Gregory et al., 1969). The deaths of 97 people in this fire were attributed mainly to NO_x. An additional 26 people died between 2 h and one month after the fire, and 92 people were treated for nonfatal injuries. In laboratory tests of nitrogen-containing materials under controlled conditions, 1 to 1000 ppm of NO_x were measured (Lieu et al., 1981; M. Paabo, NIST, unpublished data; Tsuchiya, 1984; Babrauskas et al., 1991). In military tests of armored vehicles penetrated by high-temperature ammunition, NO_2 levels above 2000 ppm (3760 mg/m^3) were found (Mayorga et al., 1995).

10.6.2.2.1 Individual and Binary Mixtures

In small-scale laboratory tests of NO_2 in air, deaths of Fischer 344 male rats occur only in the postexposure period, and the LC_{50} value following a 30-min exposure is 200 ppm (376 mg/m^3) (Levin et al., 1989c). Carbon dioxide plus NO_2 show synergistic toxicological effects (Levin et al., 1989c). The LC_{50} for NO_2 following a 30-min exposure to NO_2 plus 5% CO_2 is 90 ppm (169 mg/m^3) (postexposure deaths) (i.e., the toxicity of NO_2 doubled).

The details of the following research are provided in Levin et al., 1995. As mentioned above, CO produces only within-exposure deaths and its 30-min LC_{50} is 6600 ppm (7560 mg/m^3). In the presence of 200 ppm (376 mg/m^3) of NO_2, the within-exposure toxicity of CO doubled (i.e., its 30-min LC_{50} became 3300 ppm [3780 mg/m^3]). An exposure of approximately 3400 ppm (3890 mg/m^3) CO plus various concentrations of NO_2 showed that the presence of CO would also increase the postexposure toxicity of NO_2. The 30-min LC_{50} value of NO_2 went from 200 ppm (376 mg/m^3) to 150 ppm (282 mg/m^3) in the presence of 3400 ppm (3890 mg/m^3) of CO. A concentration of 3400 ppm of CO was used because that concentration is not lethal during the exposure, thus permitting any postexposure effects of CO on NO_2 to become evident; the LC_{50} of CO (6600 ppm) (7560 mg/m^3) would have caused deaths of the animals during the 30-min exposure.

The 30-min LC_{50} of O_2 is 5.4% and the deaths occurred primarily during the exposures. In the presence of 200 ppm (376 mg/m^3) of NO_2, the within-exposure LC_{50} of O_2 and its toxicity increased to 6.7%. In the case of O_2, increased toxicity is indicated by an increase in the value of the LC_{50} because it is more toxic to be adversely affected by a concentration of O_2 ordinarily capable of sustaining life. Exposure of the animals to 6.7% O_2 plus various concentrations of NO_2 showed that the NO_2 toxicity doubled (i.e., its LC_{50} value decreased from 200 ppm to 90 ppm [376 mg/m^3 to 169 mg/m^3]).

One of the most interesting findings was the antagonistic toxicological effect noted during the experiments on combinations of HCN and NO_2. As mentioned above, the 30-min LC_{50} for NO_2 alone is 200 ppm (376 mg/m^3) (postexposure) and the 30-min within-exposure LC_{50} for HCN alone is also 200 ppm (221 mg/m^3). These concentrations of either gas alone are sufficient to cause the death of the animals (i.e., 200 ppm HCN or 200 ppm NO_2 would cause 50% of the animals to die either during the 30-min exposure or after the 30-min exposure, respectively). However, in the presence of 200 ppm of NO_2, the within-exposure HCN LC_{50} concentration increases to 480 ppm (530 mg/m^3) or, in other words, the toxicity of HCN decreases by 2.4 times.

The mechanism for this antagonistic effect is believed to be as follows. In the presence of H_2O, NO_2 forms nitric acid (HNO_3) and nitrous acid (HNO_2) (Goldstein et al., 1980). These two acids are

the suspects most likely responsible for the lung damage leading to the massive pulmonary edema and subsequent deaths noted following exposure to high concentrations of NO_2. Nitrite ion (NO_2^-) formation occurs in the blood when the nitrous acid dissociates. The nitrite ion oxidizes the ferrous ion in oxyhemoglobin to ferric ion to produce methemoglobin (MetHb) (Equation [10.2]) (Rodkey et al., 1976). MetHb is a well-known antidote for CN^- poisoning (Klaassen, 1996). MetHb binds cyanide-forming cyanmethemoglobin, which keeps the cyanide in the blood and prevents it from entering the cells. In the absence of MetHb, free cyanide will enter the cells, react with cytochrome oxidase, prevent the utilization of O_2, and cause cytotoxic hypoxia. If, on the other hand, cyanide is bound to MetHb in the blood, it will not be exerting its cytotoxic effect. Therefore, the mechanism of the antagonistic effect of NO_2 on the toxicity of cyanide is believed to be due to the conversion of oxyhemoglobin [$O_2Hb(Fe^{2+})$] to methemoglobin [$MetHb(Fe^{3+})$] in the presence of nitrite (see Equation [10.2]).

$$2H^+ + 3NO_2^- + 2O_2Hb\left(Fe^{2+}\right) = 2MetHb\left(Fe^{3+}\right) + 3NO_3^- + H_2O \qquad (10.2)$$

10.6.2.2.2 Tertiary Mixtures of NO_2, CO_2, and HCN

Earlier work indicated that the presence of 5% CO_2 with either HCN or NO_2 produced a more toxic environment than would occur with either gas alone (Levin et al., 1987a, 1989c). The antagonistic effects of NO_2 on HCN indicate that the presence of one LC_{50} concentration of NO_2 (~200 ppm) (376 mg/m^3) will protect the animals from the toxic effects of HCN during the 30-min exposures, but not from the postexposure effects of the combined HCN and NO_2. Thus, it was of interest to examine combinations of NO_2, CO_2, and HCN (Levin et al., 1995). In this series of experiments, the concentrations of HCN were varied from almost 2 to 2.7 times its LC_{50} value (200 ppm) (221 mg/m^3). The concentrations of NO_2 were approximately equal to one LC_{50} value (200 ppm) (376 mg/m^3) if the animals were exposed to NO_2 alone and approximately one-half the LC_{50} (90 ppm) (169 mg/m^3) if the animals were exposed to NO_2 plus CO_2; the concentrations of CO_2 were maintained at approximately 5%; and the O_2 levels were kept above 18.9%. The results indicated that CO_2 does not make the situation worse but rather provides additional protection even during the postexposure period. In each of six experiments, some or all of the animals lived through the test even though they were exposed to greater than lethal levels of HCN plus lethal levels of NO_2 and in four tests, some of the animals lived through the postexposure period even though the animals were exposed to combined levels of HCN, NO_2, and CO_2 that would be equivalent to 4.7 to 5.5 times the lethal concentrations of these gases. One possible reason that CO_2 seems to provide an additional degree of protection is that NO_2 in the presence of 5% CO_2 produces four times more MetHb than does NO_2 alone (Levin et al., 1989c).

10.6.2.2.3 Mixtures of CO, CO_2, NO_2, O_2, and HCN

The initial design of these experiments was to look for additivity of the CO/CO_2, HCN, and NO_2 factors keeping each at about one-third of its toxic level, while keeping the O_2 concentration above 19% (Levin et al., 1995). When these initial experiments produced no deaths, we started to increase the concentrations of CO up to one-third of the LC_{50} of CO alone (6600 ppm) (7560 mg/m^3), HCN was increased to 1.3 or 1.75 times its LC_{50} depending on whether the within-exposure LC_{50} (200 ppm) (221 mg/m^3) or the within- and postexposure LC_{50} (150 ppm) (166 mg/m^3) was being considered, and NO_2 was increased up to a full LC_{50} value (200 ppm) (376 mg/m^3). The results indicated that just adding a NO_2 factor (e.g., $[NO_2]/LC_{50}$ NO_2) to Equation (10.1) would not predict the effect on the animals. A new mathematical model was developed and is shown as Equation (10.3). In this model, the differences between the within-exposure predictability and the within-exposure and postexposure predictability are the following: (1) the LC_{50} value used for HCN is 200 ppm

(221 mg/m³) for within-exposure or 150 ppm (166 mg/m³) for within-exposure and postexposure and (2) the HCl and HBr factors are not used to predict the within-exposure lethality, only the within-exposure and postexposure lethality. According to Equation (10.3), animal deaths will start to occur when the N-Gas Value is above 0.8, and 100% of the animals will die when the value is above 1.3. Results indicated that in those few cases where the values were above 0.8 and no deaths occurred, the animals were severely incapacitated (close to death) as demonstrated by the absence of the righting reflex or the eye reflex.

$$\text{N-Gas Value} = \frac{m[\text{CO}]}{[\text{CO}_2]-b} + \frac{21-[\text{O}_2]}{21-\text{LC}_{50}(\text{O}_2)} + \left(\frac{[\text{HCN}]}{\text{LC}_{50}(\text{HCN})} \times \frac{0.4[\text{NO}_2]}{\text{LC}_{50}(\text{NO}_2)}\right) + 0.4\left(\frac{[\text{NO}_2]}{\text{LC}_{50}(\text{NO}_2)}\right) + \frac{[\text{HCl}]}{\text{LC}_{50}(\text{HCl})} + \frac{[\text{HBr}]}{\text{LC}_{50}(\text{HBr})} \tag{10.3}$$

10.6.2.2.4 The N-Gas Model Including NO_2

For an explanation of these terms, see the paragraphs following Equation (10.1). Equation (10.3) should be used to predict the within-exposure plus postexposure lethal toxicity of mixtures of CO, CO_2, HCN, reduced O_2, NO_2, HCl, and HBr. The LC_{50} values will be the same as those given for Equation (10.1) using 150 ppm (166 mg/m³) for HCN and 200 ppm (376 mg/m³) for NO_2. If one wishes to predict the deaths that will occur only during the exposure, the LC_{50} value used for HCN should be 200 ppm (221 mg/m³) and the HCl and HBr factors should not be included. To predict the lethal toxicity of atmospheres that do not include NO_2, Equation (10.1) should be used.

10.7 COMBUSTION TOXICITY TEST METHODS

The toxicity of the combustion products from any new material formulation or product containing additives or new combinations of additives needs to be examined. Material and polymer chemists keep trying to develop new "fire-safe" materials (Nelson, 1995). The terms "fire safe" or "fire resistant" are not the same as noncombustible. Unless these new materials are truly noncombustible, some thermal decomposition will occur when the materials are exposed to fire conditions. Both the toxic gases and the irritants that are present in all smoke need to be considered potential dangers. The toxic products can cause both acute and delayed toxicological effects. It is the acute and extremely short-term effects that prevent escape from burning buildings by causing faulty judgment, incapacitation, and death. The irritants in the smoke can also interfere with one's ability to escape by causing severe coughing and choking and by preventing one from keeping one's eyes open long enough to find the exits. In addition, the delayed effects, such as tissue or organ injury, mutagenicity, carcinogenicity, and teratogenicity need to be studied because they may ultimately lead to permanent disability and postexposure deaths. Some of the issues involved in delayed effects are addressed in an article by Lewtas (1994) and a review by Barfknecht (1983). The current advances in the field of genetics provide investigators with new opportunities to examine the effects of combustion products at the molecular level. One objective could be to determine whether these toxic products cause DNA damage and mutations. Specific problems of interest include: Does the damage occur in nuclear DNA, or mitochondrial DNA, or both? Are certain areas of the DNA more prone to these mutations (i.e., are there hot spots?)? Can we categorize the types of mutations (e.g., transitions, transversions, deletions, insertions)? And how efficient are the repair mechanisms? Are these mutagens also known to be carcinogens?

Toxicity-screening tests for both the acute and delayed effects are needed, therefore, to evaluate the combustion products including any irritants that may be present in newly proposed materials and

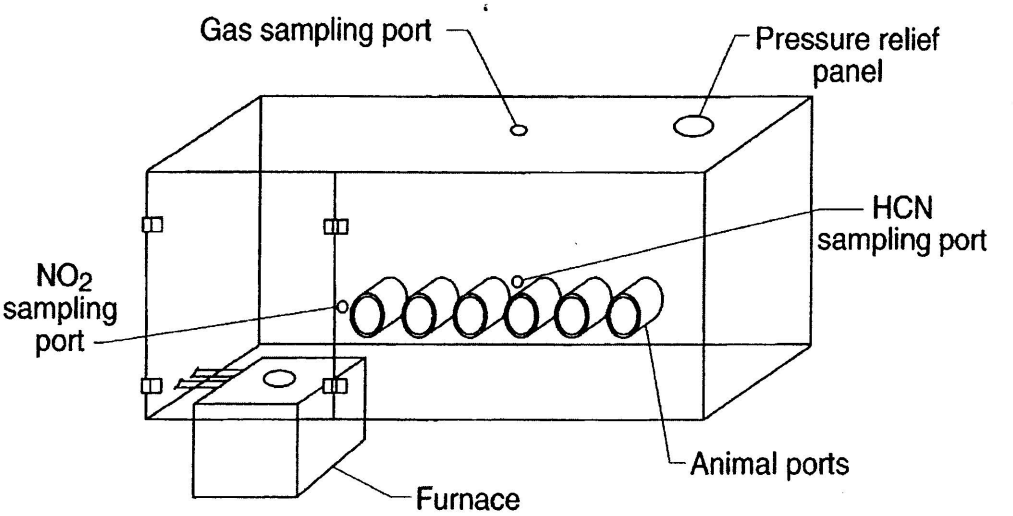

FIGURE 10.1 Schematic illustration of the NBS Cup Furnace Smoke-Toxicity Method.

products. It is imperative that the materials and products be tested under experimental conditions that simulate realistic fire scenarios of concern (e.g., flash-over conditions emanating from smoldering and then flaming of upholstered furniture in homes or smoldering fires in concealed spaces in aircraft). The ideal tests should be simple, rapid, and inexpensive; these tests should use the least amount of sample possible (because, in many cases, only small amounts of a new experimental material may be available), use a minimum number of test animals, and have a definitive toxicological end point for comparison of the multiple candidates.

Although faulty judgment and incapacitation are significant causes of worry because they can prevent escape and cause death, they are extremely difficult and complex end points to define and measure in non-human test subjects. Death of experimental animals (e.g., rats), on the other hand, is a more definitive and easily determined end point and can be used to compare the relative toxicities of alternate materials deemed suitable for the same purpose. The assumption made here is that, if the combustion products of material X are significantly more lethal than those of material Y, the combustion products of X would probably cause more incapacitation and more impairment of judgment than Y as well. The number of experimental animals can be significantly reduced by utilizing one of the predictive mathematical models developed for combustion toxicology such as the N-Gas Models previously discussed in this chapter.

Many test methods for the determination of the acute toxicity of combustion products from materials and products have been developed (Kaplan et al., 1983). In 1983, thirteen of the methods published up to that time were evaluated by Arthur D. Little, Inc. to assess the feasibility of incorporating combustion toxicity requirements for building materials and finishes into the building codes of New York State (Anderson et al., 1983). On the basis of seven different criteria, only two methods were found acceptable. These two methods were the flow-through smoke toxicity method developed at the University of Pittsburgh (U. Pitt.I Method) (Alarie and Anderson, 1979, 1981; Levin et al., 1992a) and the closed-system cup furnace smoke toxicity method (NBS Cup Furnace Method) (Figures 10.1 and 10.2) developed at NIST (known at that time as the National Bureau of Standards) (Levin et al., 1982, 1991b). In 1991 and 1992, standard reference materials were developed at NIST and made available to the users of these two methods to provide assurance that they are performing the methods correctly (Levin et al., 1991b, 1992a). Based on the results of the Arthur D. Little report, the state of New York under Article 15, Part 1120 of the New York State Fire Prevention and Building

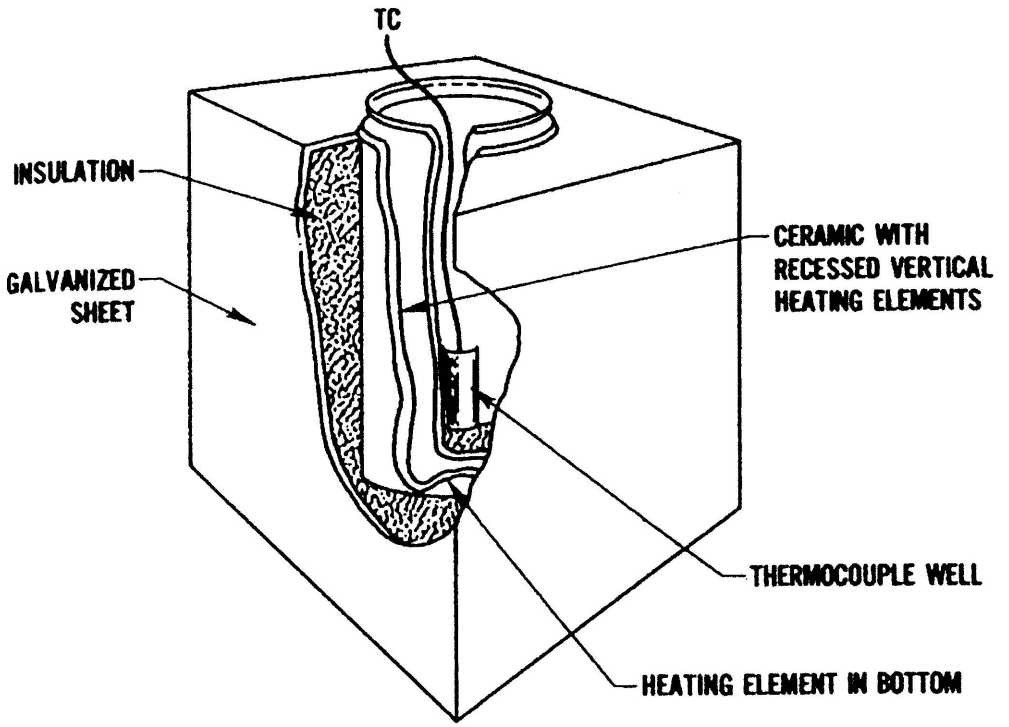

FIGURE 10.2 Pyrolysis/combustion cup furnace used in the NBS Cup Furnace Smoke-Toxicity Method.

Code, decided to require that building materials and finishes be examined by the method developed at the University of Pittsburgh and that the results be filed with the state (New York State Fire Prevention and Building Code, 1986). Note, however, that although the results are filed, the state of New York does not regulate any materials or products based on the results of this or any other toxicity test.

Since 1983, several new approaches to assess acute combustion toxicity have been examined. These approaches include a radiant furnace smoke toxicity protocol developed by NIST and SwRI (Babrauskas et al., 1991, 1998; Levin,1992a, 1992b), the University of Pittsburgh II radiant furnace method (Caldwell and Alarie, 1990a, 1990b, 1991), and the National Institute of Building Sciences (NIBS) toxic hazard test method (Norris, 1988; Roux, 1988). All three use radiant heat to decompose materials.

The NIST radiant test and the NIBS toxic hazard test use the same apparatus consisting of three components: a radiant furnace, a chemical analysis system, and an animal exposure chamber (Figure 10.3). The chemical analysis system (Figure 10.4) and animal exposure system are identical with that developed for the NBS cup furnace smoke-toxicity method. Although the apparatuses of both methods are essentially the same, they have different toxicological end points. In the NIST method, an approximate LC_{50}, based on the *mass* of material needed to cause lethality in 50% of the test animals during a 30-min exposure or a 14-day postexposure period, is the determinant of toxicity. The number of animals needed to run the test is substantially reduced by first estimating the LC_{50} by the N-Gas model and analytical tests without animals. This estimate is then verified with one or two animal tests to ensure that no unforeseen gas was generated. The toxicological end point of the NIBS toxic hazard test is the IT_{50}, the irradiation *time* (the time that the material is exposed to the radiant heat) that is required to kill 50% of the animals during a 30-min exposure or 14-day postexposure time. The actual results of the NIBS test with 20 materials indicated that the test animals died in very short periods (personal communication) and the test was unable to discriminate very well

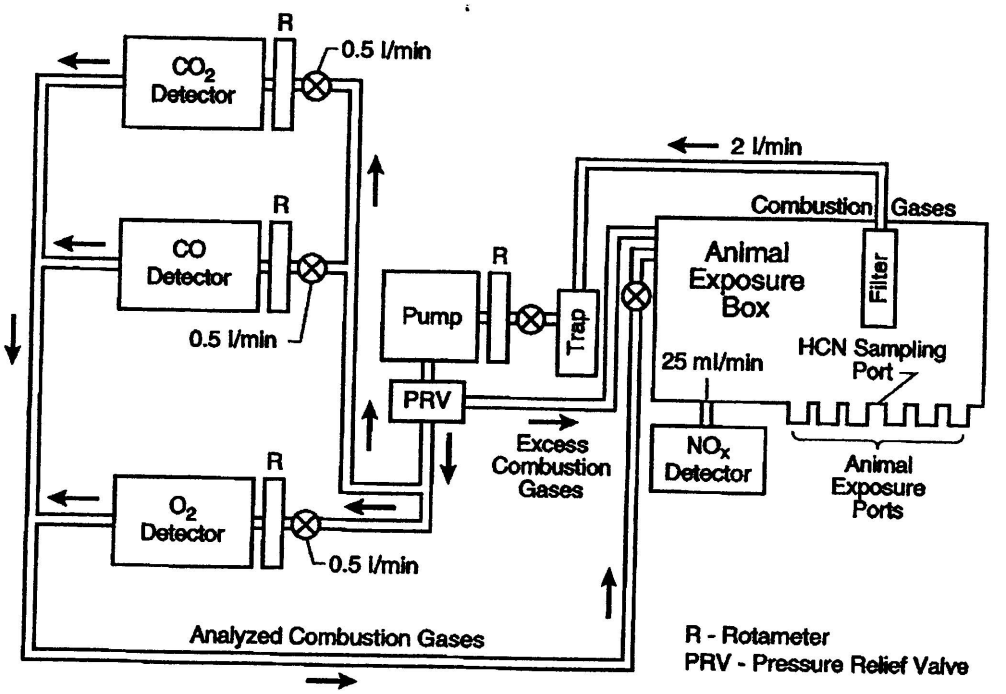

FIGURE 10.3 Schematic illustration of the NBS/NIST smoke-toxicity chemical analytical equipment in relation to the animal exposure chamber.

between materials. These results substantiate the thesis that mass (the smaller the mass necessary for an LC_{50}, the more toxic the material) is a better indicator of acute toxicity than time.

Both the NIST and NIBS test procedures are designed to simulate a postflashover scenario. The premise for simulating a postflashover fire is that most people that die of inhalation of toxic gases in *residential* fires in the United States are affected in areas away from the room of fire origin. Smoke and toxic gases are more likely to reach these distant areas following flashover. This scenario may not be relevant in certain circumstances (e.g., aircraft interior fires, where a smoldering fire in a concealed space may cause significant problems if the plane is over a large body of water and unable to land for a considerable period). In the United Kingdom, more fire fatalities are found in the room of fire origin (Hall, 2003). The reason for this difference between the two countries is not clear.

The NIST radiant test has been accepted by the ASTM as a national standard designated ASTM E1678–02 and entitled "Standard Test Method for Measuring Smoke Toxicity for Use in Fire Hazard Analysis"(ASTM, 2002). The NFPA has also adopted the NIST Radiant Test Method as a national standard called "Standard Test Method for Developing Toxic Potency Data for Use in Fire Hazard Modeling," NFPA 269 (NFPA, 2003). In 1995, the International Organization for Standardization, Technical Committee 92, Subcommittee 3 (ISO/TC92/SC3) on Toxic Hazards in Fire published an international standard for combustion toxicity after approval by 16 countries (ISO, 1996). This standard, ISO/IS 13344 entitled "Determination of the Lethal Toxic Potency of Fire Effluents," describes the mathematical models (including the N-Gas Model) available for predicting the toxic potency of fire atmospheres based on the toxicological interactions of the main combustion gases present. In the international standard, investigators have the flexibility of designing or choosing a system that will simulate conditions relevant to their fire scenario, rather than having to accept a designated combustion system.

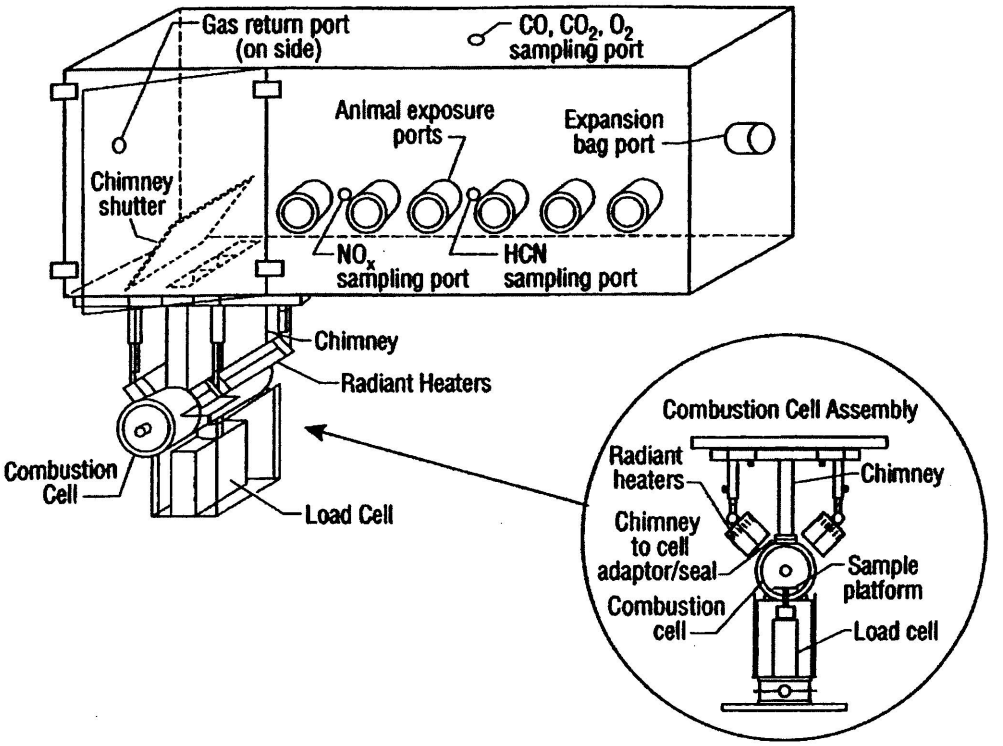

FIGURE 10.4 Schematic illustration of the NIST Radiant Smoke-Toxicity Method and the combustion cell assembly.

10.8 TOXICANT SUPPRESSANTS

Fire scientists are very familiar with fire-retardant chemicals, which are defined by ASTM as "Chemicals, which when added to a combustible material, delay ignition and combustion of the resulting material when exposed to fire" (ASTM, 2004). The discussion adds "A fire-retardant chemical can be a part of the molecular structure, an admixture or an impregnant." The term "toxicant suppressant," however, is a new expression arising from research at NIST which demonstrated that the addition of copper compounds to flexible polyurethane foam (FPU) significantly reduced the generation of hydrogen cyanide (HCN) as well as the toxicity of the combustion products when the foam was thermally decomposed (Levin et al., 1988, 1989a, 1989b, 1990b, 1992b). These experiments were designed to simulate the nonflaming and then flaming stages of a chair ignited by a cigarette (a two-phase heating system that simulates the fire scenario that results in the most fire deaths in the United States) (Levin et al., 1985b). The term "toxicant suppressant" may be defined as a chemical that, when added to a combustible material, significantly reduces or prevents one or more toxic gases from being generated when that material undergoes thermal decomposition. The resultant gas effluent should be less toxic than that from the untreated material, i.e., the toxic gas, whose concentration is being reduced, should not be converted to an equally or more toxic product.

The results of these studies at NIST indicated that:

1. HCN concentrations in the thermal decomposition products from a flexible polyurethane foam were reduced approximately 85% when the foam was treated with 0.1% or 1.0% Cu_2O and thermally decomposed via a two-phase heating system in the NBS cup furnace smoke-toxicity apparatus.

2. The copper or copper compounds could be added to the foams during or after formulation and still be operative in reducing the toxicity and HCN yield. (The BASF Corporation prepared the foams that had the Cu powder and Cu_2O added during formulation.) The addition of the copper or copper compounds during formulation did not affect the foaming process or the physical appearance of the foams except for a slight change of color.
3. Low levels of the copper compounds were effective. In particular, when cupric oxide (CuO) was used, the concentration of copper needed was only 0.08% by weight and when cuprous oxide (Cu_2O) was used, only 0.07% by weight was needed to significantly reduce the generation of HCN.
4. Full-scale room burns indicated that the presence of Cu_2O in the FPU reduced the HCN generation by approximately 50 to 70% when the experimental plan was designed to simulate a realistic scenario (the foams contained 1.0% Cu_2O, were covered with a cotton upholstery fabric, and arranged to simulate a chair; smoldering was initiated with cigarettes and flaming occurred spontaneously).
5. Under small-scale conditions, less than 3 ppm of NO_x was generated from the untreated foams, whereas a range of 3 to 33 ppm of NO_x was measured from the 0.1 to 1.0% Cu_2O-treated foams. About 6% of the HCN appeared to be converted to NO_x. In the full-scale room tests, approximately 23% of the HCN appeared to be converted to NO_x. Because we have shown in our laboratory that NO_2 acts as an antagonist to HCN, this amount of NO_x may also act to counteract the immediate toxic effects of any residual HCN.
6. Because the atmospheric oxygen (O_2) concentrations can reach very low levels in real fires, it was important to know if the reduction of HCN by copper would occur under low O_2 conditions. Small-scale tests with the ambient O_2 concentrations as low as 6% indicated that the HCN levels were reduced by as much as 82% when the FPU was treated with 0.1% Cu_2O.
7. The toxicity of the gas effluent was also reduced (an indication that the HCN was not being converted into some compound that was even more toxic). Fewer animal (Fischer 344 rats) deaths occurred during the 30-min exposures to the FPU treated with the copper and copper compounds than the untreated FPU. Toxicity based on LC_{50} values was reduced 40 to 70% in the small-scale tests with 0.1% Cu_2O-treated foams. The blood cyanide levels in the animals exposed to combustion products from the CuO-treated foams for 30 min were one-half to one-fourth those measured in the animals exposed to the smoke from the same amount of untreated foam.
8. Postexposure deaths were also reduced in the animals exposed to the combustion products from the Cu- and Cu_2O-treated FPU foams in the small-scale tests. These delayed postexposure deaths have *not* been observed in animals exposed to combustion products from flexible polyurethane foams decomposed in large-scale room fire tests. The specific cause of these postexposure deaths is not known.
9. No differences in flammability characteristics between the 0.1% Cu_2O-treated and untreated FPU foam were observed. These characteristics were examined to ensure that the positive effect on toxicity was not contradicted by negative effects on the flammability properties. The flammability characteristics examined were (1) ignitability in three systems (the cup furnace smoke-toxicity method, the Cone Calorimeter, and the Lateral Ignition and Flame Spread Test [LIFT]); (2) heat release rates under small-scale (Cone Calorimeter) and medium-scale (furniture calorimeter) conditions; (3) heats of combustion under small-scale (Cone Calorimeter) and medium-scale (furniture calorimeter) conditions; (4) CO/CO_2 ratios under small-scale (Cone Calorimeter) and medium-scale (furniture calorimeter) conditions; (5) smoke obscuration (Cone Calorimeter); and (6) rate of flame spread (LIFT).
10. Research conducted at the BASF Corporation indicated that the physical properties of the 1.0% Cu_2O-treated FPU were not significantly different from the comparable untreated

FPU. The physical properties examined were tensile strength, elongation, tear strength, resilience, indentation force deflection, support factor, compression sets, and airflow.

11. The use of melamine-treated FPU is becoming more common (Weil and Choudhary, 1995; Weil and Zhu, 1995); it is one of two FPU foams currently allowed in Great Britain. Small-scale tests indicated that a melamine-treated FPU generated six times more HCN than an equal amount of a non-melamine-treated foam. The presence of Cu_2O reduced the HCN from the melamine foam by 90%.

Jellinek and coworkers in the late 1970s also showed that the concentrations of HCN generated from the thermal decomposition of a polyurethane at 300 and 400°C decreased when flowed through copper compounds (Jellinek and Takada, 1977; Jellinek et al., 1978). In their studies, the polyurethane films were very thin (15 μm thick and 50 mg in weight). In some experiments, the metal powder was mixed with the polymer and, in others, copper metal films of 400 to 1000A were deposited on top of the polymer films. In most cases, the percent of copper was 10% or greater. The lowest concentration that they tested was a 2.6% copper film that inhibited the evolution of HCN by 66%. Their experiments indicated that the copper is probably acting as an oxidative catalyst that would decompose gaseous HCN into N_2, CO_2, H_2O, and small amounts of nitrogen oxides. Further research is needed to determine whether this is the actual molecular mechanism that allows copper to act as a HCN toxicant suppressant.

The research of Levin and her coworkers differed from that of Jellinek in that much larger samples of FPU (including full-scale room burns of cushions and simulated chairs), much smaller concentrations of copper were used, and the toxicity of the combustion products from the copper-treated FPU was also examined.

Unpublished data of Levin also indicated that the combustion products from a wool fabric treated with copper would generate 50% less HCN than the untreated fabric. These results demonstrate a potentially more universal effect, namely that treating nitrogen-containing materials with copper compounds will reduce the HCN generated when that material is exposed to fire conditions. Taking these results one step further, one could develop other toxicant suppressants that, when added to materials and products, would now prevent or significantly reduce the toxic effluents that are generated when they are thermally decomposed.

10.9 CONCLUSIONS

The field of toxicology of combustion products has come a long way since the early 1970s when the National Commission on Fire Prevention and Control submitted their report entitled "America Burning" to President Nixon (National Commission on Fire Prevention and Control, 1973). That report, which included the statement that "Appallingly, the richest and most technologically advanced nation in the world leads all the major industrialized countries in per capita deaths and property loss from fire," provided the impetus to determine the cause of and to try to solve this major problem in the United States. It was shortly thereafter that Petajan et al. (1975) published their paper in *Science* entitled "Extreme Toxicity from Combustion Products of a Fire-Retarded Polyurethane Foam" and raised the specter of "supertoxicants" emanating from combustion atmospheres. In the following years, the field of combustion toxicology expanded greatly and many materials and products were tested by various methods. With the exception of the material described in the Petajan et al. article and one other product described in section 10.3, the concern about supertoxicants has proven to be largely unfounded. However, the potential that the combustion products from new formulations or combinations of compounds may prove to be extremely or unusually toxic has encouraged manufacturers to continue to test their new products. The state requirements of New York have also been a motivating force for manufacturers to test the building products they wish to sell in New York (New York State Uniform Fire Prevention and Building Code, 1986). This testing may have prevented dangerous products from reaching the marketplace.

After reaching a peak in the 1980s, the amount of research in the area of combustion toxicology has declined significantly. With the exception of some military laboratories, very few laboratories are still doing this type of research. The Building and Fire Research Laboratory at NIST, where much of the research quoted in this chapter was conducted, abolished its combustion toxicology research program in 1992. This does not mean, however, that additional research is not needed. Some of the areas that should be examined include: the effects of the addition of more gases and heat to the N-Gas Model; the effects of particulates alone and in combination with the N-gases; the effects of chronic exposures to fire atmospheres (e.g., those experienced by fire fighters); sublethal effects on the neurological and other organ systems; mutagenic, teratogenic, and carcinogenic effects of acute and chronic exposures; the development of improved therapeutic methods for the treatment of victims of smoke (including particulates) inhalation; and additional research on toxicant suppressants with the ultimate aim of developing fire-safe materials (i.e., materials that do not produce toxic gases even when they are thermally decomposed). Because approximately 80% of fire deaths are the result of smoke inhalation, a less toxic smoke could significantly increase the time available for escape and reduce the number of injuries and deaths from fire.

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