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Toxic Products from Fires

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The role of toxic combustion products in causing fire deaths has received considerable public attention in recent years. This paper outlines critical aspects of the problem, including the identification of common combustion products, the toxicological effects of these products and the dependence of their formation on the combustion process itself. The significance of escape as the key to survival in a fire is particularly emphasized. This paper then focuses on the development of test methods for assessment of toxic hazards produced by burning materials, with the objective being to enable decisions to be made as to whether the use of one material, as opposed to another, would result in any significant difference in hazard to life safety in a fire. It is argued that smoke toxicity test methods which determine only lethal toxicological potency under "worst case" conditions do not satisfactorily address the critical issue of relative hazard, including time-to-escape and tenability limits resulting from the fire performance of materials under comparable conditions. Since threats to escape from a fire are largely time-dependent, toxic insults produced by burning materials should also be considered as rate processes. Assessment of time thresholds exhibited by burning materials under test conditions to effect performance impairment (incapacitation) of an animal model would appear to be more relevant than lethal toxicological potencies in estimating probability of successful escape from fires. A model is advanced in which intoxication rate thresholds for materials are obtained using a rodent exposure test method. Concentration-time curves, obtained from experimentally derived concentration-time-response surfaces, are the basis for estimating rate thresholds which are distinctively different for each material and which vary as a function of test conditions. It is this performance impairment response surface which is potentially a key to the modeling of toxic hazards of smoke in perspective with other hazards presented by fire. Only when overall hazards are appropriately assessed can smoke toxicity tests be meaningful as tools in choosing materials to reduce risk to life safety in fires.

Introduction

"Every year in the United States about 10 000 people lose their lives because of fires. It has been observed and commented upon that many of these victims are not burned but succumb to the effects of 'smoke' and gases. When deaths from this source are reported it is notable that almost never has it been found, specifically, what poisonous gas or gases caused the fatality." These words were written almost 50 years ago in the Quarterly of the National Fire Protection Association.[1] They could well be written today, except for the citing of a somewhat lower fatality figure of about 8000, due partly to more reliable methods for the gathering of statistical information on fire deaths.

Fire is an exceedingly hostile environment. Human tolerance for the products of combustion, including fire gases, flame, heat and visible smoke, is very low. The role of these combustion products in causing fire deaths has received considerable public attention over the years, from the 1929 Cleveland Hospital fire (125 fatalities) through the more recent disasters at the Beverly Hills Supper Club (164 fatalities), the MGM Grand Hotel (85 fatalities) and the White Plains Stouffers Inn (26 fatalities). Also receiving the attention of the general public have been some 480 fire fatalities resulting from 15 postcrash aircraft fires involving U.S. turbine-powered transport carriers since 1965.[2] Impairment of escape caused by inhalation of toxic fire gases has been implicated in many of these deaths. Although such fires in transportation vehicles, public buildings and institutions receive the major public attention, the fact remains that 80% of all fire fatalities occur in places of residence, with very few fires being responsible for large numbers of deaths.

Escape from a Fire

Escape from a fire is the key to survival. Assuming sufficient time, unimpaired faculties and the absence of physical restraints (physical handicaps, locked doors, unsuitable egress areas, *etc.*), escape should be possible. However, fire presents a combination of time-dependent threats to life safety due to a variety of physiological and behavioral effects resulting from the inhalation of heated air and fire gases.

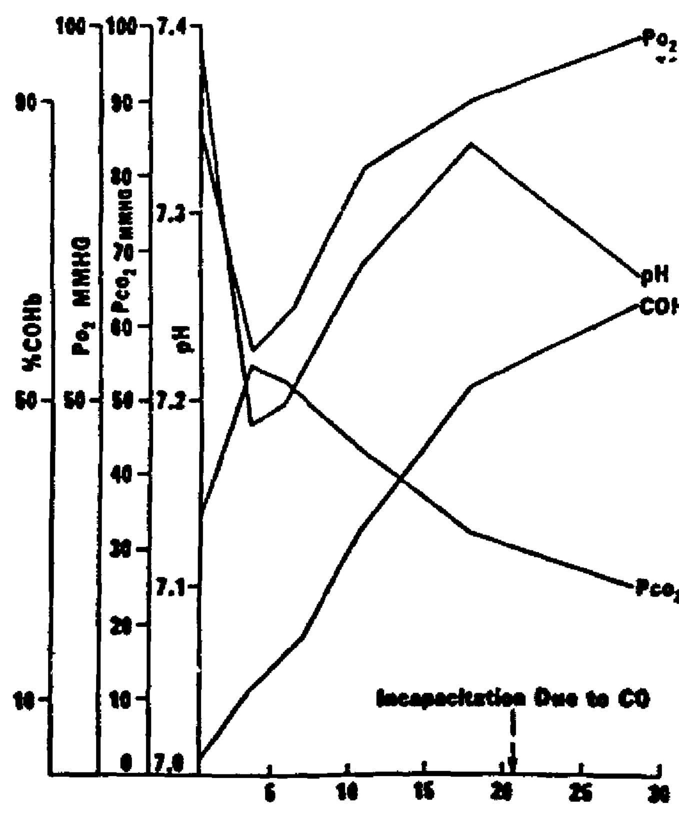

Figure 1 — Exposure of rats to irritating smoke: time course of blood analysis values.

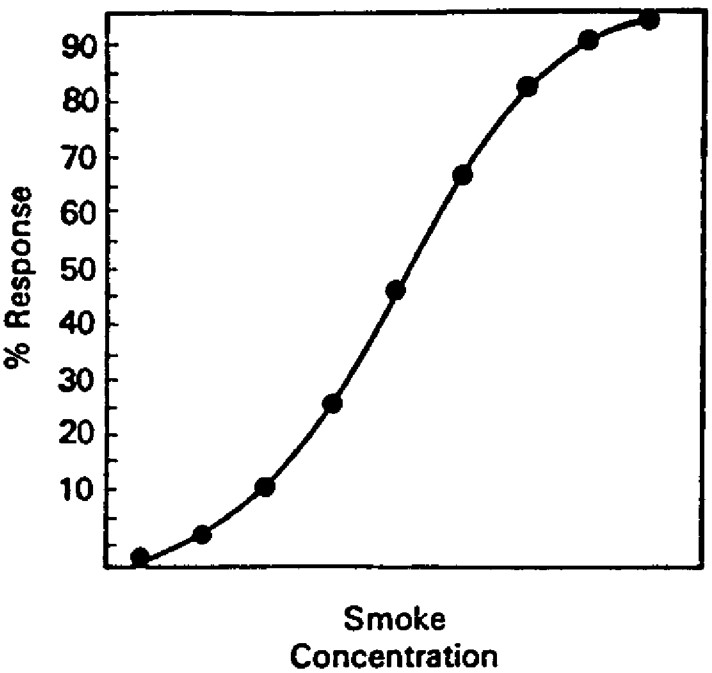

Figure 2 — Concentration-response relationship.

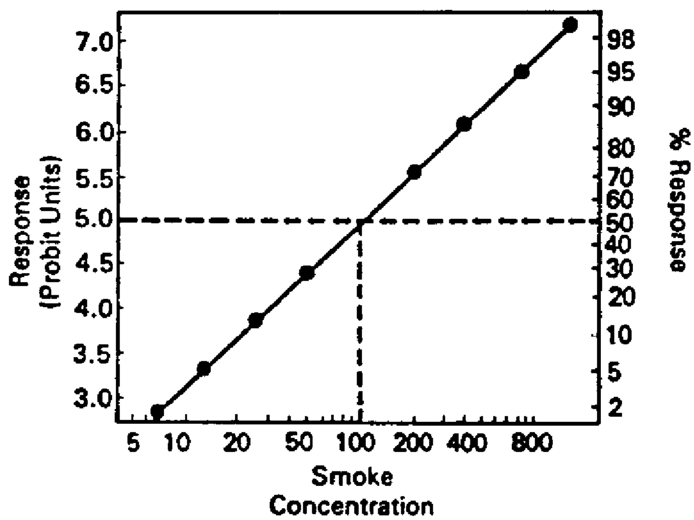

Figure 3 — Concentration-response relationship (probit plot).

Common combustible materials, *e.g.*, cellulosics, contain carbon which, in the presence of sufficient oxygen, burns to form carbon dioxide. Under conditions of limited ventilation or inadequate oxygen in the combustion zone, incomplete oxidation occurs with the resulting formation of carbon monoxide as well as carbon dioxide. This is usually the case, except for premixed fuel and air. Therefore, carbon monoxide is a product of essentially all fires.

Many of the materials in our environment, both natural and synthetic, contain nitrogen, sulfur and halogens, which, when burned along with carbon and hydrogen, may form hydrogen cyanide (HCN), nitrogen oxides (NO_x), sulfur dioxide (SO_2), ammonia (NH_3) and halogen acids (HCl, HBr and HF). Incomplete combustion often leads to the formation of hydrocarbons, aldehydes (*e.g.*, acrolein), isocyanates, nitriles and many other species. Well over 100 chemical species have been identified from the burning of some materials. Thus, it is evident that the toxicity of smoke is a far more complex situation than that involving a single chemical exposure. The complexity is further magnified when one considers the possibility of the interactive effects of toxicants including additivity, synergism and antagonism.

Toxicity of Fire Gases

Carbon Monoxide

Extensive investigations examining human fire fatalities have shown carbon monoxide to be the primary toxicant.[3,4] The extremely dangerous carbon monoxide levels in the range of several thousand parts per million are quite commonly encountered in fire atmospheres. Even substantially lower concentrations of carbon monoxide may be detrimental to some individuals' ability to escape from a fire. For example, exposure to carbon monoxide sufficient to produce carboxyhemoglobin saturations in the 3% to 5% range has been reported to impair cardiovascular function in patients with cardiovascular disease as well as in normal subjects.[5-8] Carboxyhemoglobin saturations in the 4% to 6% range have been shown to significantly reduce the threshold for ventricular fibrillation in normal anesthetized dogs and those with acute myocardial injury.[9] These levels also increase myocardial ischemia associated with acute myocardial infarction in dogs.[10]

Thus, it is not surprising that in only about half of the fire fatality cases studied, blood carboxyhemoglobin levels resulting from inhalation of carbon monoxide were found sufficiently high as to be lethal.[3,4] In an additional 30% of the victims, combinations of carbon monoxide with pre-existing heart disease and/o alcohol intoxication were considered to be the cause of death. Data showed that in fire fatalities in which alcohol was involved, 88% of the victims were legally intoxicated. The seriousness of inhalation of even relatively low levels of carbon monoxide over short periods of time should not be underestimated in the case of individuals with pre-existing physiological impairment.

There exists further evidence that relatively low levels of carboxyhemoglobin saturation may have adverse effects on vigilance ability, reaction time, time discrimination and perhaps mental functions, all of which may be important to escape from a fire.[11]

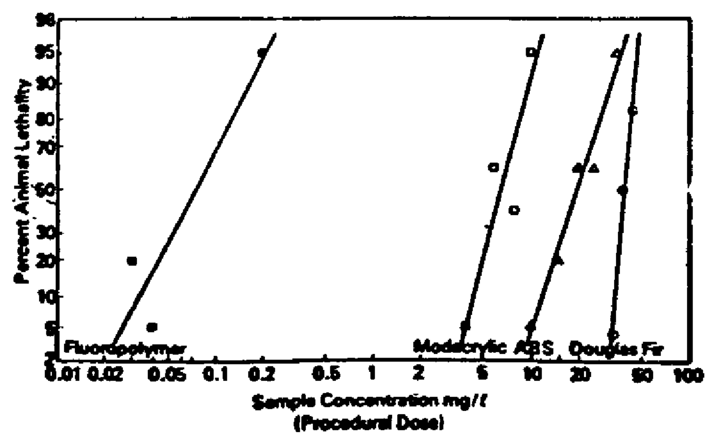

Figure 4 — Spectrum of concentration-response relationships for burning of materials (flaming mode).

Hydrogen Cyanide

The role of hydrogen cyanide as a causative agent in human fire fatalities is somewhat less clear than that of carbon monoxide. Analysis of blood for cyanide must be interpreted with caution, since cyanide is continually metabolized by living organisms. Furthermore, cyanide is normally present in blood as a result of the breakdown of hemoglobin and body tissue.

Hydrogen cyanide, possibly arising from nitrogen-containing polymeric materials, was found at elevated levels in the blood of 70% of the fire victims in one study, with possible toxic levels of blood cyanide being found in 35% of the victims.[4] However, significant levels of blood cyanide in this study were generally found associated with high levels of blood carboxyhemoglobin saturation. Thus, the contribution of each to death could not be determined with confidence. Documented cases in which hydrogen cyanide alone can be considered to be the primary toxicant are rare, although its role in causing incapacitation, particularly in the presence of carbon monoxide, is unclear.

Irritant Gases

The potential roles of sensory and pulmonary irritants (*e.g.*, halogen acids, aldehydes, *etc.*) contained in fire atmospheres have been studied using laboratory animals.[12,13] Respiration rates are reduced, often leading to severe hypoxia. In Figure 1, it is demonstrated that exposure of rats to an irritating smoke atmosphere quickly results in decreased blood oxygen content, retention of blood carbon dioxide and blood acidosis long before carboxyhemoglobin saturation reaches a level normally associated with incapacitation. Relevance of this form of asphyxia exhibited by rats to escape of human subjects in fire atmospheres has not been demonstrated.

Carbon Dioxide

While carbon dioxide is not particularly toxic at levels normally observed in fires, moderate concentrations do stimulate the rate of breathing. This condition contributes to the overall hazard of a fire gas environment by causing accelerated uptake of toxicants and irritants.

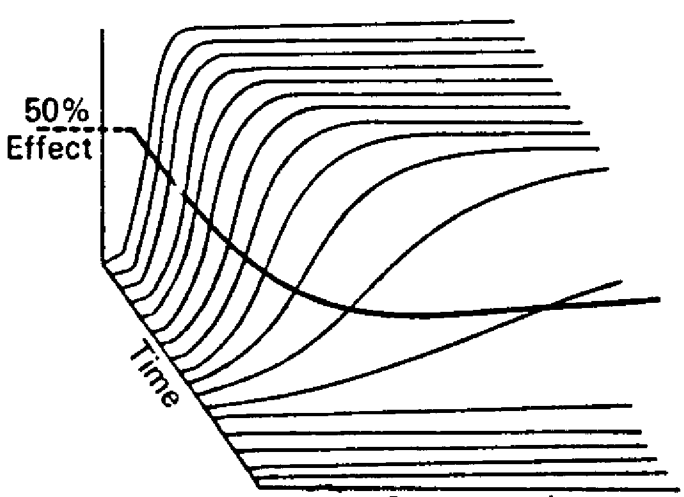

Figure 5 — Family of concentration-response curves.

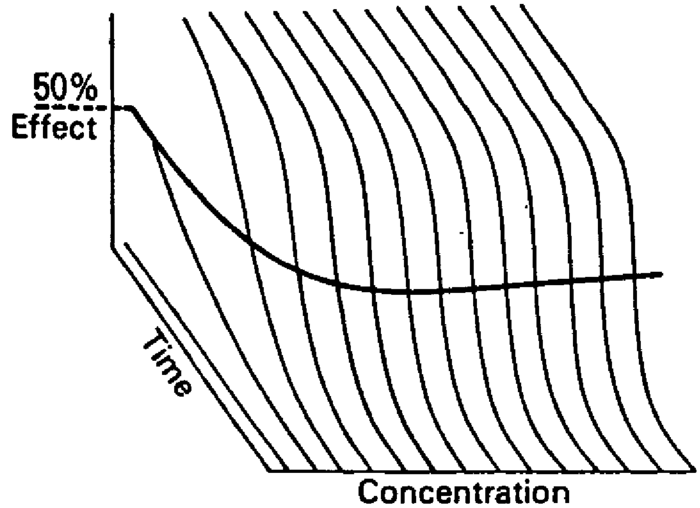

Figure 6 — Family of time-response curves.

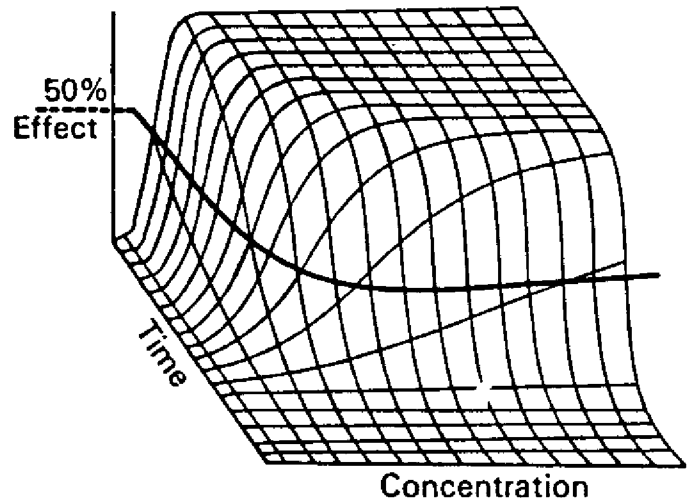

Figure 7 — Concentration-time-response surface.

Insufficient Oxygen

Oxygen is consumed from the atmosphere during combustion. When oxygen drops from its usual level of 21% in air to about 17%, motor coordination is impaired, when it drops into the range of 14% to 10%, a person is still conscious but may exercise faulty judgment and will be quickly fatigued, in the range of 10% to 6%, a person loses consciousness and must be revived with fresh air or oxygen within a few minutes to prevent death.[14] During periods of exertion, increased oxygen demands may result in symptoms of oxygen deficiency at higher percentages.

Other Fire Gases

The toxicological effects of other constituents of fire gases and their sources are summarized in Table 1.[15]

Laboratory Assessment of Toxicity of Smoke from Materials

Laboratory studies using rodents have indicated that the overall toxicity of smoke and fire gases do not vary greatly among most common materials in a conventional toxicological

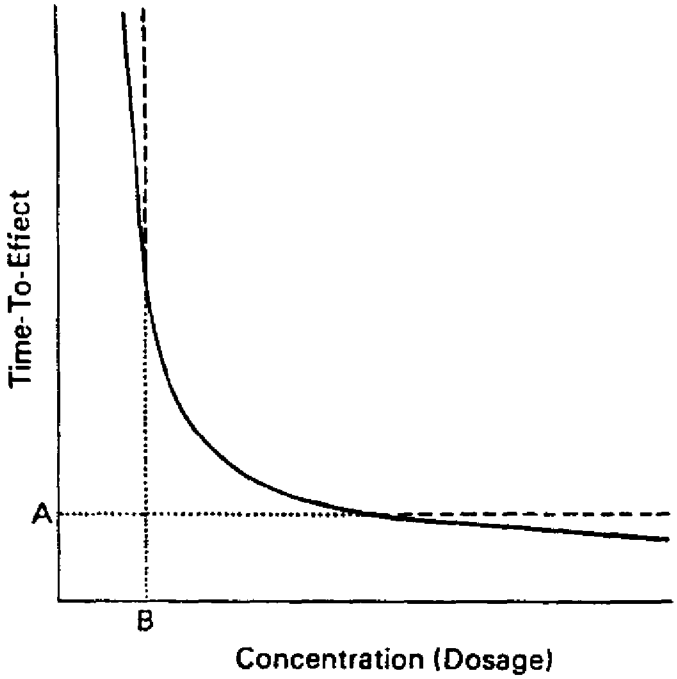

Figure 8 — Schematic of theoretical hyperbola describing Haber's Law (solid line), and the deviation usually observed in actual Ct-curves (broken line).

sense[16] However, such studies have been limited in scope, and criteria for defining significant differences in fire gas toxicity have not been established. Furthermore, the application of toxicity data in fire hazard assessment has not been sufficiently developed as to be generally useful in choosing between alternate materials with confidence. The finding of an unusually potent neurotoxin being produced from a specific material in a laboratory fire environment has raised the issue of possible hazards from unique toxic insults.[17] Therefore, considerable effort is being spent on the development of test methods which will identify materials of unusually high toxicological potency and unusual rapidity of toxic action.[13,16,18-20]

Most test methods follow the same general plan. Using a variety of combustion methods, selected for relevancy to certain real-fire scenarios, smoke is produced by burning or thermally decomposing a material. The smoke is confined in a chamber containing animal subjects, usually rodents, which are exposed for a specific period of time with certain biological response endpoints being observed. Responses may be either behavioral or physiological. Behavioral responses refer to an animal's ability to perform a learned task when appropriately stimulated. Physiological responses may include a variety of endpoints, with death being the one most frequently used. Data from behavioral and physiological responses are reconciled with combustion product analytical data in order to properly assess the nature of the toxic insult. Reduced data may be treated in a variety of ways to reach some assessment of potential toxic hazard. Toxic potency, as conventionally determined from dose-response relationships and expressed as the LC_{50} (that concentration of material effecting 50% lethality) has been the most commonly employed assessment tool in the U.S.

In a test method under development at the U.S. National Bureau of Standards, six rats restrained for exposure in the head-only mode are presented simultaneously to smoke for a period of 30 minutes at each of several concentrations of smoke or quantities of material burned under "worst case" conditions.[20] The percent animal-response is determined at each of these concentrations and a plot can be constructed of percent-response as a function of the quantity of material burned. Such a "dose-response" plot is illustrated in Figure 2. More conventionally, the probability of response or probit is plotted as a function of the logarithm of the concentration of material, as shown in Figure 3. If this procedure is carried out on a number of materials, a spectrum is obtained of toxicological potencies of materials covering as much as four orders of magnitude of toxicity. Such a spectrum is shown in Figure 4. The toxic potency spectrum is frequently separated into zones relating to some reference material, *e.g.*, "as toxic as wood," "more toxic than wood," and "much more toxic than wood." This representation of smoke toxicity test data serves to broadly categorize materials and to identify those which, under the test conditions, produce smoke exhibiting unusual lethal toxicological potency.

The test method described, which determines only lethal toxicological potency under "worst case" conditions, does not satisfactorily address the critical issue of relative hazard, including time-to-escape and tenability limits resulting from the fire performance of materials under comparable conditions. Tenability limits are functions of the nature of toxicants produced, their rapidity of action and their rates of production, *i.e.*, the thermal decomposition rates of materials under test conditions. Since threats to escape from a fire are largely time-dependent, *e.g.*, rate of flame spread, rate of mass burning, rate of smoke development, rate of heat

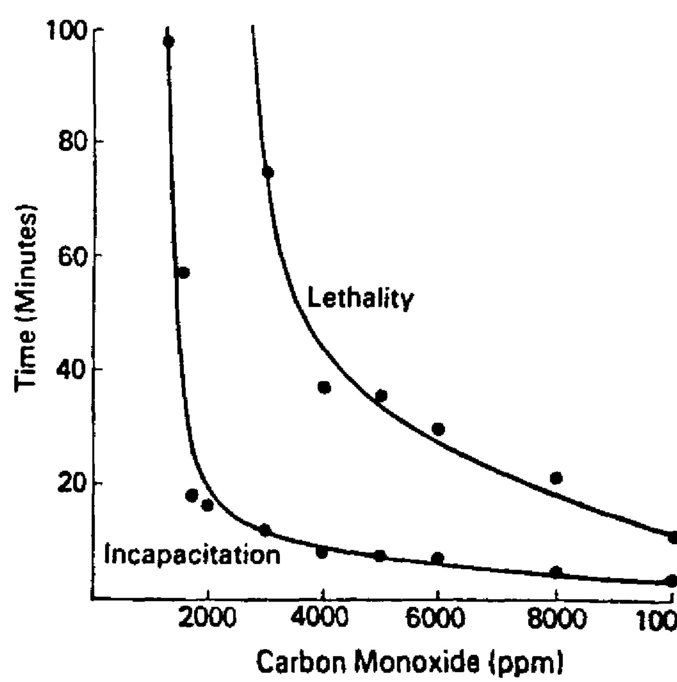

Figure 9 — Lethality and incapacitation Ct-curves for carbon monoxide.

release, *etc.*, toxic insults produced by burning materials should also be considered as rate processes. These considerations are all necessary, since the ultimate objective is to determine whether the use of one material, as opposed to another, would result in any significant difference in hazard to life safety in a fire.[21]

Assessment of time thresholds exhibited by burning materials to effect sublethal performance impairment (incapacitation) of an animal model would appear to be more relevant than lethal toxicological potencies to the escape of living subjects from a fire.[21] The hind-leg flexion avoidance response is one of a number of such techniques for assessment of rodent incapacitation.[22] This paradigm requires that the subject maintain avoidance of contact between a hind leg and a shock-producing metal plate. The response is said to be lost when the animal is no longer able to perform the task. A smoke toxicity experiment utilizing the leg flexion avoidance response is conducted essentially the same as for lethality except that restrained animals are fitted with appropriate instrumentation for delivering the shock and monitoring avoidance. Data obtained include percent of subjects incapacitated, as well as mean time-to-incapacitation of subjects in a particular test. As with lethality, percent response can be plotted as a function of smoke concentration, as shown previously in Figure 2, with an EC_{50} being determined. (The general term EC_{50} refers to that concentration effecting 50% response and may be either lethal or sublethal.) Concentration-response curves may be constructed representing a variety of test time frames. The result is a family of concentration-response curves, shown in Figure 5, whose 50% response points may be connected, yielding a plot of EC_{50} as a function of time.

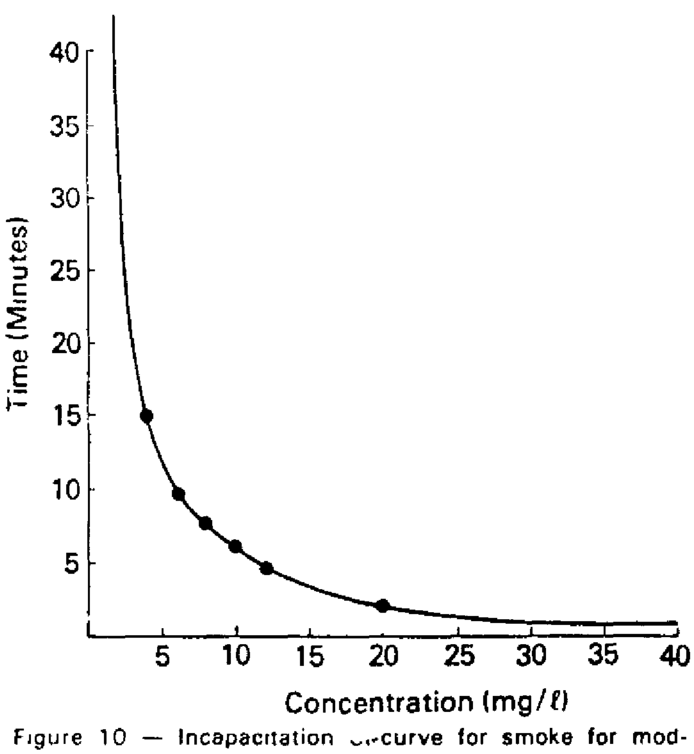

Figure 10 — Incapacitation Ct-curve for smoke for modacrylic material.

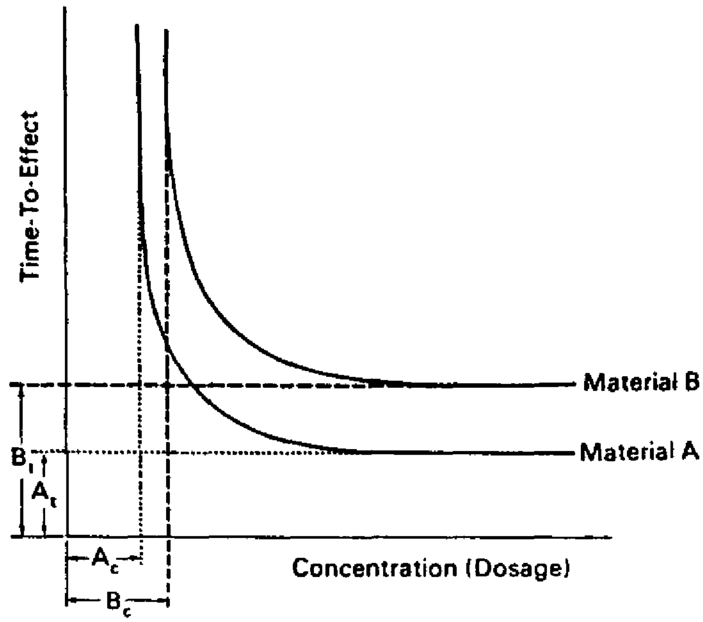

Figure 11 — Comparative concentration-time curves

By analogy, a family of time-response curves for a variety of fixed concentrations can also be obtained, as shown in Figure 6. Connecting the 50% response points yields a plot of ET_{50} as a function of concentration (The general term ET_{50} refers to that time effecting 50% response for a specified concentration.)

If the two families of curves shown in Figures 5 and 6 are superimposed, a concentration-time-response, three-dimensional surface is constructed as illustrated in Figure 7.[21] Curves representing EC_{50} as a function of time and ET_{50} as a function of concentration appear as one and the same curve. This critical curve, shown in Figure 8, is also obtained by plotting mean time-to-effect as a function of concentration and is a representation of Haber's Law, *i.e.*, $C \times t$ = constant. The actual experimental Ct-curve deviates somewhat from a theoretical hyperbola in that true asymptotes are not approached. The deviation is shown in Figure 8 as a broken line. The line "A" is the time-to-effect threshold which parallels the concentration (dosage) axis. The line "B" represents the concentration threshold which parallels the time axis. Examples of experimental Ct-curves for both lethality and incapacitation produced upon exposure of rats to carbon monoxide are shown in Figure 9. Smoke atmospheres generated from the thermal degradation of materials also exhibit characteristic intoxication rates (time thresholds) and toxic potencies (concentration thresholds). An experimental Ct-curve for incapacitation of rats upon exposure to smoke from the nonflaming combustion of a modacrylic material is shown in Figure 10.

It should be realized that the Ct-curve obtained by burning a material differs from the Ct-curve of the toxicant it produces. The toxic insult produced from the burning of a modacrylic material results largely from the cytotoxic action of hydrogen cyanide. The Ct-curve for the modacrylic material is, however, not superimposable (with respect to the coordinate axes) on that for hydrogen cyanide. The concentration threshold for the modacrylic material is displaced toward higher concentrations from that produced by hydrogen cyanide. This is because the modacrylic material does not decompose totally to hydrogen cyanide. The time threshold is displaced toward longer times from that produced from hydrogen cyanide. This displacement represents the thermal stability of the material and is manifest in the time required it to burn.

A given toxicant has only one Ct-curve corresponding to a particular response. However, materials which produce the toxicant may have different Ct-curves, displaced both horizontally and vertically from each other due to differences in quantitative production of the toxicant, to possible interactive effects with other toxicants, and to differences in the combustion performance of the materials. Thus, the Ct-curve is a characteristic of a material and reflects its toxicological performance under specified test conditions. These relationships are illustrated in Figure 11 for two hypothetical materials. In Figure 11, A_c and B_c represent the concentration thresholds for materials A and B, respectively. A_t and B_t represent the time-to-effect thresholds for materials A and B, respectively. In this manner, materials may be compared on the basis of both concentration thresholds and time-to-effect thresholds. It is the time-to-effect thresholds which are of particular interest in this paper.

Figure 12 shows Ct-curves for a number of materials as modeled from experimental data. Comparison of time-to-effect thresholds for the materials at an appropriate material concentration, *e.g.*, 30 mg/L, indicates significant differences between the materials, whereas comparison of potencies may reveal little of toxicological significance. (These materials were combusted under "worst case" thermal conditions and would be expected to result in maximum toxicity.[18] If the materials had been compared under equivalent thermal exposure conditions, considerably greater differences in time-to-effect thresholds would likely have been observed.)

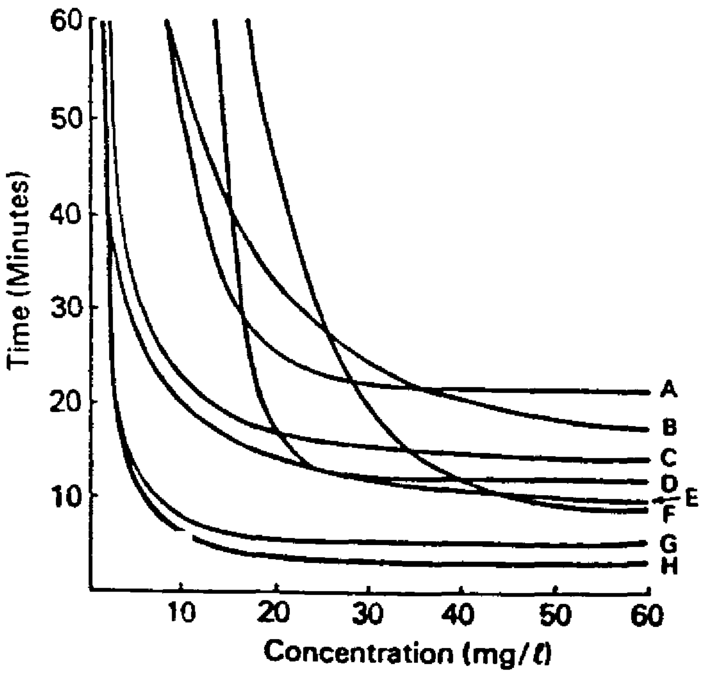

Figure 12 — Concentration-time curves for materials. A) Red oak — nonflaming; B) Red oak — flaming; C) Polyphenylsulfone — flaming, D) ABS — flaming; E) Polyphenylsulfone — nonflaming; F) ABS — nonflaming, G) Modacrylic — flaming; H) Modacrylic — nonflaming.

For present purposes of hazard assessment, it is desirable hat materials be assessed utilizing both lethal potencies and time-to-effect thresholds. A method for accomplishing this would involve plotting material performance on the basis of lethal potency, *i.e.*, LC_{50}'s, simultaneously with time-to-effect (performance impairment) thresholds, analogous to proposed presentations involving time-to-death.[23] This method of analyzing toxicological performance would appear to address smoke toxicity hazard in a more meaningful manner than simple lethality measurement.

The simple intoxication rate threshold represents only one point on the concentration-time-response surface characteristic of a burning material, as illustrated previously in Figure 7. The concept does, however, open the way toward the modeling of toxicological effects from laboratory-derived data. If the concentration in Figure 6 is conceived as a "procedural dose," rather than a toxicological dose, it can represent a function of common fire parameters, *e.g.*, surface area exposed, fire intensity (heat flux) and time of irradiation.[21] These parameters are all controllable in laboratory tests, thus enabling time-related toxicological effects to be expressed as functions of the common fire variables used in the mathematical modeling of fire and its hazards. In this manner, the modeling of overall fire hazard, and ultimately risk, can be made to include the important consideration of smoke toxicity in proper perspective with the other elements of hazard.

Only when overall hazard is appropriately assessed can smoke toxicity tests be useful, with confidence, in choosing materials to reduce risk to life safety in fires. Until such time as this can be done, toxicity tests should be used only for research information and product development guidance, rather than for regulatory purposes. In the meantime, regulations relative to flammability properties, such as ignition, flame spread and heat release, along with advances in the use of fire detection and suppression technology, would appear to be the most effective routes to reduction of fire deaths.

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