

Fire and Smoke: Understanding the Hazards

Committee on Fire Toxicology, Board on Environmental Studies and Toxicology, National Research Council

ISBN: 0-309-56860-9, 166 pages, 6 x 9, (1986)

This PDF is available from the National Academies Press at:
<http://www.nap.edu/catalog/1916.html>

Visit the [National Academies Press](http://www.nap.edu) online, the authoritative source for all books from the [National Academy of Sciences](http://www.nap.edu), the [National Academy of Engineering](http://www.nap.edu), the [Institute of Medicine](http://www.nap.edu), and the [National Research Council](http://www.nap.edu):

- Download hundreds of free books in PDF
- Read thousands of books online for free
- Explore our innovative research tools – try the “[Research Dashboard](#)” now!
- [Sign up](#) to be notified when new books are published
- Purchase printed books and selected PDF files

Thank you for downloading this PDF. If you have comments, questions or just want more information about the books published by the National Academies Press, you may contact our customer service department toll-free at 888-624-8373, [visit us online](#), or send an email to feedback@nap.edu.

This book plus thousands more are available at <http://www.nap.edu>.

Copyright © National Academy of Sciences. All rights reserved.

Unless otherwise indicated, all materials in this PDF File are copyrighted by the National Academy of Sciences. Distribution, posting, or copying is strictly prohibited without written permission of the National Academies Press. [Request reprint permission for this book](#).

FIRE AND SMOKE: UNDERSTANDING THE HAZARDS

Committee on Fire Toxicology
Board on Environmental Studies and Toxicology
Commission on Life Sciences
National Research Council

NATIONAL ACADEMY PRESS
Washington, D.C. 1986

NOTICE: The project that is the subject of this report was approved by the Governing Board of the National Research Council, whose members are drawn from the councils of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine. The members of the committee responsible for the report were chosen for their special competences and with regard for appropriate balance.

This report has been reviewed by a group other than the authors according to procedures approved by a Report Review Committee consisting of members of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine.

The National Research Council was established by the National Academy of Sciences in 1916 to associate the broad community of science and technology with the Academy's purposes of furthering knowledge and of advising the federal government. The Council operates in accordance with general policies determined by the Academy under the authority of its congressional charter of 1863, which establishes the Academy as a private, nonprofit, self-governing membership corporation. The Council has become the principal operating agency of both the National Academy of Sciences and the National Academy of Engineering in the conduct of their services to the government, the public, and the scientific and engineering communities. It is administered jointly by both Academies and the Institute of Medicine. The National Academy of Engineering and the Institute of Medicine were established in 1964 and 1970, respectively, under the charter of the National Academy of Sciences.

This study was supported by the Consumer Product Safety Commission, the Environmental Protection Agency, the Federal Aviation Administration, and the Department of the Navy under Contract 68-02-4122 between the National Academy of Sciences and the Environmental Protection Agency.

Available from: Committee on Fire Toxicology, National Academy of Sciences, 2101 Constitution Avenue, NW, Washington, D.C. 20418

Printed in the United States of America

COMMITTEE ON FIRE TOXICOLOGY

Arthur B. DuBois (Chairman), John B. Pierce Foundation Laboratory, New Haven, Connecticut

Rosalind C. Anderson, Arthur D. Little, Inc., Cambridge, Massachusetts

Frederick B. Clarke, III, Benjamin/Clarke Associates Inc., Kensington, Maryland

J. Wesley Clayton, University of Arizona, Tucson, Arizona

Donald Dressler, Harvard Medical School, Cambridge, Massachusetts

Raymond Friedman, Factory Mutual Research Corp., Norwood, Massachusetts

William T. Lowry, William T. Lowry, Inc., Arlington, Texas

Gordon Pryor, SRI International, Menlo Park, California

Linda Rosenstock, University of Washington, Seattle, Washington

James Dean Sun, Lovelace Inhalation Toxicology Research Institute, Albuquerque, New Mexico

National Research Council Staff

Karen L. Hulebak, Project Director

Zoltan Annau, Consultant

Vicky Phillips, Staff Assistant

Norman Grossblatt, Editor

BOARD ON ENVIRONMENTAL STUDIES AND TOXICOLOGY

Donald Hornig (Chairman), Harvard University, Boston, Massachusetts
Alvin L. Alm, Thermal Analytical, Inc., Waltham, Massachusetts
Richard N. L. Andrews, University of North Carolina, Chapel Hill, North Carolina
William E. Cooper, Michigan State University, East Lansing, Michigan
John Doull, University of Kansas Medical Center, Kansas City, Kansas
Emmanuel Farber, University of Toronto, Toronto, Ontario, Canada
John W. Farrington, Woods Hole Oceanographic Institution, Woods Hole, Massachusetts
Benjamin G. Ferris, Harvard School of Public Health, Boston, Massachusetts
Philip Landrigan, Mt. Sinai Medical Center, New York, New York
Raymond C. Loehr, University of Texas, Austin, Texas
Roger Minear, University of Illinois, Urbana, Illinois
Philip A. Palmer, E. I. DuPont de Nemours & Co., Wilmington, Delaware
Emil Pfitzer, Hoffmann-La Roche Inc., Nutley, New Jersey
Paul Portney, Resources for the Future, Washington, D.C.
Paul Risser, Illinois Natural History Survey, Champaign, Illinois
William H. Rodgers, University of Washington, Seattle, Washington
F. Sherwood Rowland, University of California, Irvine, California
Liane B. Russell, Oak Ridge National Laboratory, Oak Ridge, Tennessee

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Ellen Silbergeld, Environmental Defense Fund, Washington, D.C.
Peter Spencer, Albert Einstein College of Medicine, Bronx, New York
Gerald Wogan, Massachusetts Institute of Technology, Cambridge,
Massachusetts

Ex Officio

Gary P. Carlson, Purdue University, Lafayette, Indiana
Thomas Chalmers, Mount Sinai Medical Center, New York, New York
Arthur B. DuBois, John B. Pierce Foundation Laboratory, New Haven,
Connecticut
Alan M. Goldberg, Johns Hopkins University, Baltimore, Maryland
Bernard D. Goldstein, Robert Wood Johnson Medical School, Piscataway, New
Jersey
David Jollow, Medical University of South Carolina Charleston, South Carolina
Roger O. McClellan, Lovelace Inhalation Toxicology Research Institute,
Albuquerque, New Mexico
Norton Nelson, New York University Medical Center, New York, New York
Duncan T. Patten, Arizona State University, Tempe, Arizona

National Research Council Staff

Devra Lee Davis, Acting Director, BEST
Jacqueline Prince, Staff Associate

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

ACKNOWLEDGMENTS

Several persons provided the Committee and staff with helpful information, suggestions, and the benefits of their experience during the preparation of this report. We express our special gratitude for the many contributions made by Zoltan Annau, Department of Environmental Health Sciences, The Johns Hopkins University, who was a consultant to the Committee. We thank the funding agency liaison personnel for their sincere interest, flexibility, and encouragement: Susan Womble and Colin Church of the Consumer Product Safety Commission; Frederick Williams of the Department of the Navy; Donald Schroeder of the Federal Aviation Administration; and Edward Massaro of the Environmental Protection Agency.

We are grateful to Edna Paulson and Victor Miller of the Toxicology Information Center, without whose unfailingly well-informed information services no Board on Environmental Studies and Toxicology (BEST) study would be as good as it is. Devra L. Davis, Acting Director of BEST, provided valuable assistance in review throughout this project, and Alvin G. Lazen, Executive Director of the Commission on Life Sciences, provided much helpful guidance and advice. Norman Grossblatt's excellent editorial work transformed the writings of many into the voice of one committee; his contributions were invaluable.

Finally, we thank Victoria Phillips, secretary to the Committee, whose spirit and hard work made so many of our goals achievable, and Karen Hulebak, staff officer for the study, who gave unstintingly of her talents and energy. Dr. Hulebak's guidance and diplomacy in the production of this report are deeply appreciated.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

PREFACE

In 1977, the Committee on Fire Toxicology in the National Research Council's Assembly on Life Sciences (now the Commission on Life Sciences) produced a report in which the state of toxicity testing of combustion products was surveyed. The report noted that current techniques of fire-product research were so deficient that there were “no acceptable screening tests to evaluate relative toxicities of pyrolysis and combustion products of polymeric materials.” That committee made several recommendations regarding the direction of test method development, among them the following:

- Toxicity tests should use both pyrolysis and flaming decomposition conditions.
- Specific test animal species and exposure conditions should be used.
- A measure of incapacitation should be developed.
- Atmospheres to which test animals are exposed should be monitored for gas composition and temperature.
- Data derived from tests should not be used as absolute values in any fashion, but rather should be used only in comparison with data on standard reference materials.

The intervening years have seen continuing research in fire science. For example, the Research Council in 1984 established the Committee on the Toxicity Hazards of Materials Used in Rail Transit Vehicles, in the National Materials Advisory Board of the Commission on Engineering

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

and Technical Systems. Its study is funded by the Department of Transportation; a final report is expected in January 1987. The present Committee on Fire Toxicology was formed in December 1984 in the Board on Toxicology and Environmental Health Hazards (now the Board on Environmental Studies and Toxicology) in the Commission on Life Sciences. It is supported by a consortium of federal agencies (the Consumer Product Safety Commission, the Federal Aviation Administration, the Department of the Navy, and the Environmental Protection Agency) concerned with developing sound regulatory policy. The Committee's general task was to review the state of the art of combustion-product toxicity testing and fire hazard assessment, and its membership reflects the multiple disciplines required for such a task. Information generated by the fire science community was reviewed, especially data produced and analytic developments achieved since the previous Research Council committee report in 1977. In addition, the Committee considered the relationship between the physiologic and behavioral end points currently used in combustion-product toxicity test systems and the performance capabilities of humans exposed to pyrolysis and combustion products. The Committee was also to evaluate fire hazard models (both available and in development), focusing on the use of toxicity as an input, and provide guidelines for their application.

The Committee expects its findings to be of interest not only to its sponsors, but to all public officials with a similar mission and to manufacturers concerned with understanding the performance of their products.

Arthur B. DuBois, Chairman
Committee on Fire Toxicology

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

CONTENTS

EXECUTIVE SUMMARY	1
INTRODUCTION	12
1 FIRE DEATHS IN THE UNITED STATES	15
Scope of the Problem	15
Causes of Fire Death	16
The Contemporary Fire Environment	20
2 A PRIMER ON FIRE AND FIRE HAZARD	23
The Burning Process	23
A Typical Compartment Fire	25
Fire Hazard Assessment	28
Time Needed for Escape	34
Time Available for Escape	36
3 STATUS OF FIRE HAZARD MODELS AND TEST METHODS	45
Introduction	45
Detection Models	46
Models for Time Available for Escape	48
Models for Time Needed for Escape	54
Test Methods for Model Input Data	55
Summary	59
4 HAZARDS ASSOCIATED WITH FIRES	62
Heat	62
Oxygen Depletion	63
Smoke	63
Health Effects of Smoke Inhalation on Humans	73
Exposed to Fires	
Summary	77

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

5	LABORATORY METHODS FOR EVALUATION OF TOXIC POTENCY OF SMOKE	78
	Use of Combustion-Product Toxicity Tests: To Screen or Not to Screen	78
	Chemical Analysis vs. Biologic Assay	79
	Test Methods That Use Death as an End Point	83
	Test Methods That Use Nonlethal End Points	97
	Summary	104
6	GUIDELINES FOR HAZARD ASSESSMENT: CASE STUDIES	105
	Case Study 1: Burning of an Upholstered Chair	106
	Case Study 2: Concealed Combustible Material	118
	Summary	129
	REFERENCES	131

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

EXECUTIVE SUMMARY

The National Research Council's Committee on Fire Toxicology was formed in December 1984 in the Board on Toxicology and Environmental Health Hazards (now the Board on Environmental Studies and Toxicology) in the Commission on Life Sciences. It was supported by a consortium of federal agencies (the Consumer Product Safety Commission, the Federal Aviation Administration, the Department of the Navy, and the Environmental Protection Agency) concerned with developing sound regulatory policy. The Committee's general task was to review the state of the art of combustion-product toxicity testing and fire hazard assessment. In addition, the Committee considered the relationship between the physiologic and behavioral end points currently used in combustion-product toxicity test systems and the performance capabilities of humans exposed to pyrolysis and combustion products. The Committee was also to evaluate fire hazard models (both available and in development), focusing on the use of toxicity as an input, and provide guidelines for their application.

HAZARD ASSESSMENT VS. RISK ASSESSMENT

Risk is the product of an event's severity (i.e., its degree of hazard) and the probability that the event will occur. This report deals only with fire severity (i.e., fire hazard) and its quantification. The degree of fire hazard is a function of a number of factors, such as fuel load, building structure, ignition, and propagation of flames, but also including the amount and toxicity of the smoke, the exposure to the smoke before escape, and the

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

exposure to heat. The assessment of fire hazard concentrates on combining the factors that affect the time available for escape (TAE).

ASSESSMENT OF FIRE HAZARD

Hazard assessment, whether based on full-scale simulations or on mathematical models, requires an array of quantitative information, such as:

- The amount of material present.
- The amount of energy required to ignite the material and spread flame over its surface.
- The mass loss and heat release rate of the material, both when it is burning alone and when it is exposed to known energy fluxes from external sources.
- The toxic potency of its smoke, expressed in terms of concentration, such as lethal concentration, effective concentration, or lethal concentration-time product.
- Similar information on whatever else is burning, in addition to the material of interest.
- Ventilation in the fire environment.
- The geometry and thermal characteristics of the compartment that contains the fire (the fire environment).

Detection models calculate the size of a fire at the time the detector (of smoke or heat) is activated and therefore the extent to which smoke or heat has developed in the compartment that encloses or is adjacent to the detector. Fire growth and smoke transport models can be used to predict the buildup of heat and smoke, thereby permitting calculation of TAE. The most widely known is the Harvard fire model, which can predict the growth of a fire with time and the resulting buildup of smoke and heat in up to five interconnected compartments, all on one level. The model's outputs include the times of ignition of second or third objects, the rate of gas outflow from the compartment, and the concentrations of various species in the outflowing gas. The FAST model

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

developed at the National Bureau of Standards embodies some of the characteristics of the Harvard code, but emphasizes prediction of movement of fire products. It is claimed to provide more “rugged” or “robust” solutions--e.g., those with a smaller tendency to give grossly wrong answers or to fail to run to completion--for a wide variety of input parameters. Such models assume that each compartment divides into a hot upper zone and a cold lower zone, with no mixing--hence their frequent designation as “zone” models. In contrast, field models divide a compartment into hundreds or thousands of zones in a three-dimensional array and can therefore predict fluid motion far more realistically than zone models can. Field models, however, require very powerful computers and are not yet practical for routine hazard analysis.

Computer models now becoming available can, in principle, calculate the development of a fire in a compartment and the buildup of smoke at specific locations in it. Because toxicity data are relevant to the TAE, the fate of occupants of those locations cannot always be predicted unless the smoke toxicity is known: TAE can be compared with the time needed for escape or rescue (TNE) for a selected scenario. TNE in turn also depends on the ages and health of the occupants. Theoretically, non-lethal exposure to toxic fire products can affect the TNE (e.g., by impairing mental acuity and so hindering escape), so for some scenarios data on nonlethal effects would be more relevant than lethality data.

The details of the computations vary with the scenario under consideration, but it should be clear that smoke toxicity data constitute only one ingredient. Toxicity data alone are insufficient for complete and accurate assessment of a fire hazard.

The overall hypothesis of hazard assessment is that survival of any fire is likely if the TAE exceeds the TNE. TAE depends on how quickly the environment becomes untenable; this in turn is controlled by the material's flammability and smoke toxicity. TNE is largely independent of the material burning.

Besides having some importance in predicting the outcome of a given scenario, TAE serves as a surrogate for a material's relative fire hazard in that scenario. A comparison of the TAEs for a series of materials is

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

therefore considered to give an indication of their relative fire hazards under the selected conditions.

THE TESTING OF COMBUSTION-PRODUCT TOXICITY

At least two combustion-product test methods can be used to provide the toxicity data required for modeling hazard: the National Bureau of Standards (NBS) and University of Pittsburgh methods. These methods are relatively well documented and yield toxicity values in substantial agreement for most materials that have been tested with both methods. The Deutsche Industrie Norm. (DIN) 53 436 method, developed in Germany, probably would also be adequate, but has not been thoroughly evaluated in the United States. And it is to be expected that these bioassays will be improved and that others will be developed for specific uses (e.g., to measure effects on mental acuity).

In general, the primary unit of toxicity is the LC_{50} , which is the concentration of a toxicant that causes death in 50% of the exposed animals in a specified period. The $L(Ct)_{50}$, a unit that combines concentration (of fire products) and time, where appropriate for integration into a numerical fire hazard model, would theoretically make more refined results possible. The few pathologic measures that have been used (e.g., lung weights and corneal opacity) have yielded only limited information on the biologic effects of exposure to fire products.

No test providing data on incapacitation has yet been developed that is demonstrably more sensitive than the use of death as the end point, although for some fire scenarios accurate measurement of incapacitation or performance decrements could be important. If such a test is developed, one would wish to demonstrate that incorporation of an end point other than death into a fire hazard model improved the ability to assess hazard.

In the NBS method, a quartz beaker is heated to above or below the autoignition temperature of a sample to be burned; the sample is then placed in the beaker. Gases are collected in an airtight chamber, where rats are exposed for 30 min. The test results (referred to as LC_{50s}) are expressed as sample weight charged per chamber volume (mg/200 L). The animals are observed for

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

14 days after exposure, and the “LC₅₀” is based on the number of animals killed at each “concentration” (although these are not actually exposure concentrations or dosages, because the atmosphere in the exposure chamber is not characterized). The method has proved reliable and reproducible in both intralaboratory and interlaboratory tests with Douglas fir and various other materials.

In the Pittsburgh method, the sample is placed in a furnace that is then heated at 20°C/min. Mice are exposed to the continuous smoke stream, which is diluted with chilled air, for 30 min. This method also generates reproducible results. Although it has not been subjected to as many interlaboratory tests as the NBS method, the data suggest good agreement among results from three laboratories for Douglas fir and two other materials.

As is true of any small-scale test, these tests do not model a “real fire” accurately. If only the data pertinent to mortality (LC₅₀) are used in the estimation of hazard, both methods might be equally applicable. However, if mass loss rate and time to death were to be used, only the Pittsburgh method could provide this information. Although there might be exceptions to this generality, it appears on the basis of the limited comparative data available that the choice of one or the other method would not alter substantially the outcome of a fire hazard assessment.

For purposes of predicting the fire hazard of different materials, the Committee believes that the required smoke toxicity data are currently best obtained with animal-exposure methods. However, chemical analysis of smoke might be useful in the process of measuring smoke toxicity. The advantages of chemical tests are that many are quicker to perform than bioassays and that they avoid the use of test animals. The main advantage of biologic tests is that they produce data of high validity. The major potential danger of a chemical test is that it could “miss” unanticipated, and perhaps unusually toxic, combustion products (although unusually toxic combustion products whose formation was not predicted by chemistry have rarely been encountered); there is little danger of missing biologically relevant response in a bioassay. In addition, most current fire hazard models are designed to accommodate toxicity data in the form of LC₅₀ or L(Ct)₅₀ values; the use of chemical data alone in such a model

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

would require development and verification of an appropriate scheme to summarize and add the various measured concentrations.

A toxicity-testing strategy that avoids the uncertainties of a chemical analysis while exploiting its advantages could have the following steps:

- Chemically analyze the test material's smoke for expected major toxicants, such as carbon monoxide, hydrogen cyanide, and hydrogen chloride.
- Calculate an "expected" LC_{50} for the smoke, on the basis of the response of test animals to the toxicants identified in the chemical analysis.
- Perform a bioassay of the material's smoke at, slightly above, and slightly below the expected LC_{50} . If all the important toxicants have been identified in the chemical analysis, this test should be sufficient to confirm that identification and to yield an approximate LC_{50} . If the observed LC_{50} is very different from the expected LC_{50} , the difference will be apparent, and more extensive bioassays must be carried out.

Beyond LC_{50} data, the routine measurement of carbon monoxide in smoke or of carboxyhemoglobin in the blood of exposed animals, however useful such measures are for research purposes, provides no information of utility to hazard assessment efforts that is not provided with more certainty by the LC_{50} itself.

CONCLUSIONS AND RECOMMENDATIONS

There is a strong need for additional research in combustion-product toxicity testing and fire hazard assessment. Indeed, knowledge in these fields is still quite incomplete. Thus, the approaches embodied in the following conclusions and recommendations should be viewed as being of an interim nature. The issues should be reviewed again in 5 years and the recommendations revised in the light of new knowledge.

No model or test method comes close to reproducing the peril in which fire places human life. The results of mathematical models are only as good as the data used in

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

them, and those data, whether they are abstractions of smoke movement and ventilation or toxicity values determined in rodents, are only representations of the reality that takes the lives of some 5,000 people a year in this country. Although efforts to reduce the likelihood of fire must continue, we must assume that there will always be fires. We must therefore work to improve our ability to cope with fire by improving the fire performance of the materials that furnish the places in which we live and work, while continuing to improve building designs, fire codes, and fire detection and suppression techniques.

- **Although among the highest in the world, the number of fire fatalities in the United States has been declining for the last 30 years.**

The concern that the introduction of synthetic materials into general use in homes has increased the risk associated with fire is not supported by data on recent fire-death trends. New materials are being used in homes and commercial buildings, and these materials have different combustion characteristics, but the number of fire fatalities per 100,000 people has declined. This decline cannot be fully explained by improvements in firefighting equipment, sprinkler systems, or detectors, although they are relevant.

- **The best-characterized threats associated with fire are the acute results of exposure to heat, the toxic agents and irritants that make up smoke, and perhaps oxygen depletion. Long-term effects of repeated exposure, such as would be encountered by firefighters, have not been conclusively characterized.**

Smoke inhalation is the cause of death in the majority of fire fatalities. The toxic components of smoke are largely carbon monoxide and other gases, such as hydrogen cyanide. Carbon monoxide is well accepted as a factor in 50-80% of all fire fatalities; the role of hydrogen cyanide is still under investigation. Other components, such as respiratory and sensory irritants, might contribute to the inability of people to escape from fire, as well as to long-term pulmonary complications in survivors. The cause of death of many fire victims is not fully understood. Even less is understood about the potential long-term health consequences in survivors of single exposures to fire. The influence of the type of

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

materials involved in a fire on mortality has not been established. To determine cause of death, postmortem study of fire victims would include both autopsy and blood-gas investigations (for carbon monoxide, hydrogen cyanide, etc.). Prospective studies of pulmonary and neurologic function in both single-exposure fire victims and occupationally exposed firefighters are needed to evaluate long-term health consequences. There seems to be no conclusive evidence that the long-term effects of repeated exposure to fire include an increased risk of developing cancer, although some groups of firefighters have been found to have excesses of some cancers.

Finally, research on chemical and cellular biologic markers of combustion-product toxicity (e.g., with bronchoalveolar lavage) should continue, inasmuch as such markers might provide early indicators of pathologic effects of smoke toxicity.

- **The dynamics of a fire in generating heat and toxic products will determine the ability of people to escape; the use of fire hazard assessment to estimate ability to escape a given fire is currently the best approach to measuring the hazard associated with materials.**

Determination of the likelihood of escape from a burning building requires evaluation of the time available for escape and the time needed for escape. TAE can be calculated from the time at which the fire is detected, the temperature and the quantity of smoke, and the growth curve of the fire or smoke. TAE is not to be used at face value as a measurement of real time, but as a tool for comparing materials under some set of conditions. In a rapidly growing fire, temperature can increase so rapidly that the toxicity of smoke is irrelevant. In a fire that is growing slowly or if the potential victim is not in the same room as the origin of the fire, the toxicity of smoke might be the prime source of danger.

TNE is calculated from factors associated with the potential victim's ability to find a safe escape route, the nature of the building, the victim's age and related characteristics, etc.

- **If a combustion-product toxicity test is to be useful in a hazard model, it should have good inter-**

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

laboratory reproducibility, should differentiate between materials on the basis of relative toxicities, should be able to determine dose-response relationships for a given material, and should yield data in units that are compatible with hazard models.

Just as there is no single set of fire conditions, there is no “correct” set of toxicity-test parameters. Different tests can be expected to yield different rankings of the same group of materials. This failure of agreement is unlikely to be resolved by additional research and test development; and total agreement might not even be desirable, in that use of a single “standard” test could lead to unnecessarily restricted sampling of combustion conditions.

- **Laboratory methods for measuring the toxicity of combustion products have been developed to the point where relative toxicities of materials can be reliably measured.**

Both the NBS and the Pittsburgh test methods provide a comparison of relative toxicities; when used by different laboratories, each has reasonable reproducibility. Neither method provides a complete model of a real fire, but each provides data on some aspect of combustion. Data from these methods can be used in hazard models.

- **Currently used toxicity test methods use lethality as the end point; other end points remain to be developed.**

The use of death as the end point provides a reliable index of smoke toxicity, but fails to provide information on the inability of people to escape fires. This inability could be due to sublethal exposure to toxic gases, such as carbon monoxide, or to the effects of irritants in impeding escape. No animal model of sublethal effects has been found more useful than measures of lethality in providing the desired information. A sensitive measure of sublethal effects would theoretically improve the ability to assess hazard; at the least, it would allow for a more logically consistent representation of ability to escape.

In order to understand the effects of combustion products on mental acuity, specific tests of impairment

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

of judgment or performance in animals and in humans should be developed. The results of such tests could be used in a fire hazard evaluation in various ways, e.g., with respect to toxicity or human performance.

- **As a basis for judging or regulating materials' performance in a fire, combustion-product toxicity data must be used only within the context of fire hazard assessment.**

In determining overall hazard to people, toxicity data obtained from animal experiments should be incorporated into a fire hazard model. Given all the other factors that are relevant to fire hazard, such as rate of burning and heat generation, toxicity cannot be the sole criterion for defining the hazard. If products for some intended use have been shown to be very similar in composition and other fire properties, a pass/fail decision that depends on a toxicity test could be justified. Although this appears to be a screening test, it is in fact simply the final point of discrimination in a less formal hazard analysis. For uses with no regulatory component (e.g., a manufacturer's surveillance of products under development), any chosen test can be used for screening, with specific performance criteria set by the user.

- **Because of the possibility that new toxic chemicals will not be detected in chemical tests of combustion products, biologic tests must remain the ultimate toxicity assays.**

Chemical tests can be extremely useful in measuring concentrations of known chemicals in combustion products and thus might become a first screen for testing toxicity. An animal biologic model acts as the ultimate integrator of the combined toxicities of combustion products, whereas a chemical assay is a selective measure of specific chemicals and might or might not detect all toxic agents. Therefore, animal tests must remain as the final determinants of human hazard. This necessity could become even more evident as nonlethal measures of toxicity become available.

- **Although techniques will continue to improve, fire hazard assessment can already be used to answer fundamental questions about the suitability of materials.**

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Computational models require a knowledge of the burning rate of materials and critical concentrations of toxic combustion products, if TAE is to be calculated. TAE is used as a surrogate of fire hazard and makes it possible to compare relative fire performance of materials in a given application.

Powerful computers and increasingly detailed analysis of more complex spaces will lead to better understanding of the dynamics of fires and to more realistic approximation of TAE. In the meantime, however, many regulatory questions can already be answered.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

INTRODUCTION

This report deals with current understanding of combustion-product toxicity and fire hazard assessment. In studying these issues, the Committee on Fire Toxicology examined the number of fire-related deaths in the United States and possible causes of these fatalities. A short primer on fire and fire hazard was developed, to provide a better understanding of the status of fire hazard models and test methods. The Committee has studied the hazards associated with fires and reviewed the test methods now used to evaluate the toxicity of combustion products. Finally, the Committee developed guidelines for hazard assessment and prepared specific case studies based on them.

About 5,000 people die every year in the United States as a result of fire. Technical improvements have occurred--e.g., in building fire codes, firefighting techniques, flammability standards for mattresses, the use of fire detection devices in homes, the use of sprinkler systems in public buildings, and public fire-safety awareness--and the annual number of fire deaths has been decreasing for 20 years. But it is commonly believed that the fire death rate might have decreased even more if new materials of synthetic origin had not come into use.

Most fire-related deaths are due to inhalation of toxic gases in smoke, not to fire or heat itself. Carbon monoxide (CO) is thought to be the most common cause of fire-related death. Because of its high affinity for hemoglobin, relatively small concentrations of CO can saturate the blood, form carboxyhemoglobin (COHb), and deprive tissues of oxygenation. In general, COHb

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

concentrations above about 30-40% seriously impair the ability of humans or animals to perform a task, and concentrations above 50-60% can be fatal. Some people can function even at COHb concentrations up to 50%, because a hypoxic challenge, whether caused by oxygen (O₂) depletion or CO accumulation, increases cerebral blood flow. In the face of decreasing O₂ availability, however, this compensatory mechanism eventually fails to deliver enough O₂ to the brain, and the victim loses consciousness. If the victim survives, severe neurologic disorders can sometimes be seen after a period of apparent recovery. It was recently suggested that hydrogen cyanide (HCN) can also contribute importantly to the overall toxic hazard of fire. The neuropathologic patterns after HCN and CO exposure appear to be similar; thus, despite the difference in mechanisms of inducing hypoxia, the two gases might damage neural tissue in an identical manner.

In addition to toxic gases, fire generates many respiratory irritants--such as hydrogen chloride, acrolein, and sulfur dioxide--that can cause necrosis and pulmonary edema. Delayed deaths have reportedly occurred after what appeared to be mild exposures to these gases. A review of the major combustion products and their individual contributions to the toxic hazard affords only partial insight into the total toxic hazard in fire. The overall hazard is associated with exposure to mixtures of the individual combustion products and with their effects in preventing escape from the fire environment. The irritant effects of fires on survivors can be classified as early and late. The early effects are usually associated with damage to the upper airways and the respiratory tract in general. The most common late sequela of a single exposure is some degree of pulmonary obstruction. Studies of firefighters have shown, with some variability, that a long-term consequence of repeated fire exposure can be the development of an obstructive, restrictive, or mixed ventilatory defect. With regard to the possible increase in cancer incidence in firefighters, the results of several studies have suggested higher than normal incidences of a variety of cancers, but there seems to be no conclusive relationship between the type of cancer and exposure history.

The roles of the technical improvements in fire prevention and detection mentioned above in fire safety and fire loss are not well understood. It is therefore

not possible to determine whether the use of “new materials” is associated with different or increased hazard. However, even in the absence of a demonstrable increase in the role of toxicity in fires, the concern over new materials warrants attention. It is appropriate to continue the deliberate process of developing methods for analyzing and predicting product response to fire under expected conditions of use. Such methods will be useful both for selecting materials for specific uses and for substantiating regulatory positions.

The hazards presented by fire are best assessed through consideration of all the characteristics of fire. The hazard associated with smoke depends both on how rapidly a material produces smoke and on the toxic and irritant potency of the smoke once produced. In general, smoke hazard cannot be characterized unless both kinds of information--production rate and potency--are taken into account. Smoke production rate is a function of a material's fire properties and of the environment in which the fire takes place. Therefore, hazard depends on the situation: laboratory measurements of materials themselves do not predict hazard until the measured properties are evaluated in the context of how the material is to be used and how it might burn--i.e., in a given fire scenario. The two means of providing information on the fire scenario are full-scale simulations and mathematical fire models. The expense and cumbersomeness of full-scale fire simulation make reliance on mathematical models desirable, and the Committee believes that models have been developed to the point where they can be used--cautiously--as a basis for fire hazard assessment.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

1

FIRE DEATHS IN THE UNITED STATES

SCOPE OF THE PROBLEM

As a cause of accidental death in the United States, fire is exceeded only by automobile collisions, falls, and drowning.²⁶ The United States and Canada have the highest absolute numbers of fire-related deaths in the world and fire-death rates generally 2-4 times those in Europe.¹⁹⁸ The United States also has one of the highest per capita fire rates.²²²

U.S. fire deaths have been decreasing for the last 20 years, with an overall decrease of about 35% in that period (Figure 1-1). When adjusted for population, the decrease is even more marked--approximately 42% since the early 1950s (Figure 1-2). Throughout the latter period, fires in the home accounted for an average of more than 75% of all fire deaths (Figure 1-1 and Figure 1-2).

Most fire deaths occur in one- or two-family dwellings and apartments (Table 1-1). People are most at risk of dying in a fire when they are sleeping⁶² or when their ability to escape is otherwise impaired. In a common type of residential fire, death occurs at night,²⁶ results from the ignition by cigarette of upholstered furniture or bedding (Table 1-2), and involves intoxicating amounts of alcoholic beverages.³⁵

Although most fire deaths occur in residences (one or two per fire), the fires that seem to attract public attention are the dramatic and catastrophic ones that result in the loss of many lives. According to the National Fire Protection Association (NFPA), reported

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

multiple-death fires (those which resulted in three or more deaths per fire) caused 16.4% of the fire deaths in 1984. On the basis of NFPA data for 1980-1984, loss of life in multiple-death fires has decreased, owing to a reduction in the number of these incidents, rather than in their severity.⁶²



FIGURE 1-1 Fire deaths in the United States, 1950-1980, total and home. (Data do not include transport-related fire deaths.) Data from National Center for Health Statistics.¹⁶⁰ Census data for 1968 are missing.

CAUSES OF FIRE DEATH

Accurate data on the causes of deaths associated with fire are difficult to obtain. Autopsy is the only means available to determine the cause of death conclusively (i.e., smoke inhalation versus burns), but it does not always provide more definitive information--e.g., was death due to carbon monoxide (CO), to some other toxicant, or to a combination of toxicants? Finally, autopsy is not usually ordered in cases of fire death.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.



FIGURE 1-2 Fire deaths per 100,000 population in the United States, 1950-1980, total and home. (Data do not include transport-related fire deaths.) Data from National Center for Health Statistics.¹⁶⁰ Census data for 1968 are missing.

It is generally accepted that 70-80% of fire deaths result from smoke inhalation.^{35 135} Smoke, as defined by the American Society for Testing and Materials,¹³ is “the airborne solid and liquid particulates and gases evolved when a material undergoes pyrolysis or combustion.” Indeed, a comprehensive study of fire deaths, performed by the Applied Physics Laboratory of The Johns Hopkins University on the basis of data from Maryland, found that CO, a toxic gaseous component of smoke, was the cause or a contributing cause of 80% of fire deaths (Table 1-3). Alcohol was involved in 40% of deaths.

CO, produced by all fires as a component of smoke, is often considered to be the major toxicant produced by fires; it acts by binding to red blood cells and forming carboxyhemoglobin (COHb), which interferes with oxygen transport. In studies of fire death, COHb concentrations of 50-60% are generally accepted as fatal.^{35 194} (For a complete discussion of CO toxicity, see Chapter 4.)

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Besides CO, smoke contains carbon dioxide and can contain oxides of nitrogen, hydrogen cyanide (HCN), hydrogen chloride, sulfur dioxide, acrolein, benzene, phenol, and other compounds.¹⁹² These substances, individually or in combination with each other or with CO, can cause immediate or delayed death. They can also impede escape from fire, and thereby increase risk of death, by obscuring vision as a result of eye irritation and lacrimation, by impairing mobility, or by impairing mental acuity.

TABLE 1-1

Property Use	Estimated Number of Civilian Deaths	Fraction of Civilian Deaths, %
Residential (total):	4,240	80.9
One- and two-family dwellings	3,290	62.8
Apartments	785	15.0
Hotels and motels	120	2.3
Other residential	45	0.9
Nonresidential structures	285	5.4
Highway vehicles	530	10.1
Other vehicles	100	1.9
Other	85	1.6
Total	5,240	99.9

^aData from Karter.¹¹⁵

The possibility that toxic gases other than CO cause fire-related deaths has been investigated in several studies. Analysis of samples from 80 victims of the MGM Grand Hotel fire³⁶ revealed that approximately half the victims had COHb concentrations less than 50%; that raises the question of which other toxic factors might have contributed to these deaths. Investigations of a jail fire in Johnson City, Tennessee,³⁷ and of Maryland fire deaths over a 42-month period³⁵ discovered potentially toxic concentrations of HCN in the blood of a number of victims.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

High HCN concentrations, however, were always associated with high (not necessarily lethal) CO concentrations. Surveys of fire victims in Glasgow have confirmed this, in that both survivors and nonsurvivors had substantial quantities of cyanide in their blood after the fire.^{17 49}

TABLE 1-2

Item Ignited	Ignition Source	Fraction of U.S. Fire Deaths, %
Furnishings	Smoking	27
Trash, apparel	Smoking	4
Furnishings, flammable liquids, apparel	Open flame	11
Furnishings, flammable liquids, apparel, interior finish	Heating and cooking equipment	13
Structural materials, interior finish	Electric equipment	4
Flammable liquids, apparel	Other	7
Other scenarios, each less than 2% of total	Variable	34
		100

^aData from Benjamin/Clarke Associates, Inc.³²

Eighteen of the 23 victims of the Air Canada cabin fire in 1983 had sublethal COHb concentrations (less than 50%). Blood concentrations of HCN, however, were lethal in 14 or 19 of the victims, depending on whether one assumes a fatal concentration of HCN to be 1.0 or 2.0 µg/ml.³⁸ That a number of survivors breathed through wet towels supports the inference that HCN, a hydrophilic agent, was a major factor in causing death. Breathing through wet fabric can in principle reduce the concentration of hydrophilic compounds, but not of CO. However, it is not known how many of those who died also breathed through wet towels. (High fluoride concentrations were also found in the victims' blood. The toxicologic significance of the observed concentrations, however, was not established. Exposure to hydrogen fluoride, a hydrophilic acid gas, could also have been reduced by breathing through wet fabric.)

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TABLE 1-3

Cause of Death	Fraction of Deaths, %
CO alone ^b	60
CO plus cardiovascular disease	20
Burns	11
Unexplained	9
Total	100

^aData from Birky et al.³⁵

^bCarboxyhemoglobin content over 50%.

THE CONTEMPORARY FIRE ENVIRONMENT

Findings like those just described, combined with a growing public awareness of the toxic hazards associated with fire, have led to the belief that today's fires produce combustion products that are more toxic than the fires of 30 or 40 years ago. Some assume that the increased presence of synthetic materials in the built environment causes fires to burn hotter and faster and to produce more toxic smoke than ever before. Although synthetic materials are more prevalent in our work and residential environments than they were 40 or even 20 years ago (Figure 1-3), the national fire-death rate has decreased over the last 30 years. No single factor can explain this trend. For example, the decrease might reflect recent decreases in fire incidence, improvements in firefighting techniques, changes in building fire codes, and the use of home smoke detectors.

Although it is possible to document the cause of death in fire victims and potentially possible to identify through pyrolysis/mass spectrometry³⁸ the sources of the combustion products inhaled by victims, such studies are infrequent. And the existing data cannot be used to

determine trends, because comparative data from the presynthetic era (before 1950) are not available. Post-mortem examination of fire victims for pathologic evidence of exposure to such irritants as HCl is a fairly new practice; and some techniques for measurement of combustion products, such as atomic-absorption spectroscopy for detection of heavy metals and gas chromatography for measurement of blood cyanide, have become widely available only recently.

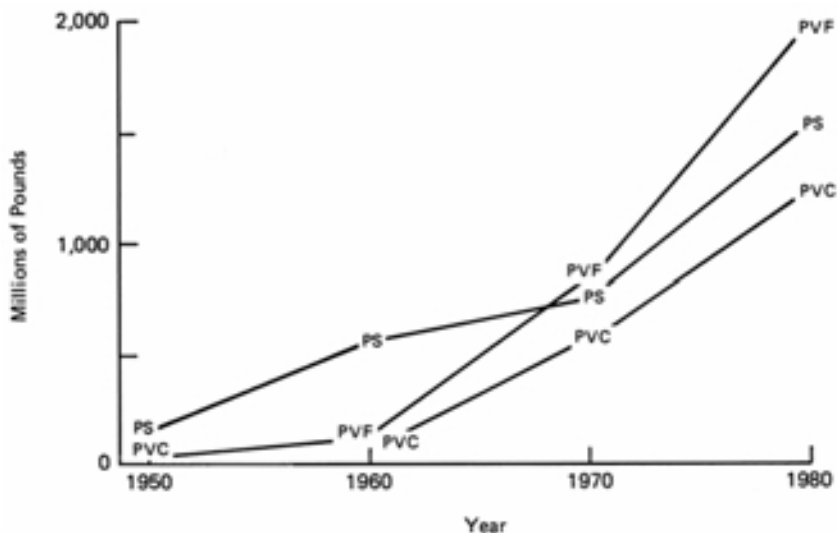


FIGURE 1-3 Production of poly(vinyl chloride), polystyrene, and polyurethane. PUF = total polyurethane production; data from Society of the Plastics Industry.²⁰⁷ PS = molded polystyrene production for selected consumer markets; data abstracted from *Modern Plastics*.^{149 150 151 152} PVC = poly(vinyl chloride) film production for selected consumer markets; data abstracted from *Modern Plastics*.^{149 150 151 152}

In view of the lack of comparative-pathology studies and of death-rate trends, there is little evidence that modern fires present a greater risk of death than fires of 30 or 40 years ago--either residential fires or large multiple-death fires, such as the Cocoanut Grove fire of 1943.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

The hypothesis of a greater toxic hazard in contemporary fires might be tested by collecting prospective epidemiologic evidence and exploring potentially variable postexposure health effects in survivors of fires of different kinds. For example, evidence of a change in smoke toxicity could appear as an increased incidence of some pulmonary complications in those exposed to fires that involved greater amounts of synthetic materials. Data for such a study could be drawn from hospital records, insurance-company records, firefighter-association statistics, and so forth.

Many factors impinge on the fire problem in the United States; the change in the fuel load of the built environment is only one of them. However, whatever the cause of death, the United States has the highest fire-death rate in the world. An improved understanding of the hazards associated with fires, including toxic hazards, will certainly assist all who must deal with fire and its consequences, be they fire-safety engineers, firefighters, medical personnel, or those who find themselves threatened by fire.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

2

A PRIMER ON FIRE AND FIRE HAZARD*

Combustion-product toxicology is becoming a technical specialty in its own right, but its regulatory utility is limited to the degree to which it aids the regulator in measuring and controlling the overall fire hazard of a material. Thus, it is important to develop understanding of the dynamics of fire to the point where the role of smoke and its toxic effects can be placed in perspective. That is the purpose of this chapter. (Two other sources that offer a more thorough treatment are the report of the Products Research Committee¹⁸² and Drysdale.⁶⁹)

THE BURNING PROCESS

The fuel for most unwanted fires is organic material, e.g., the wooden frame of a house, an item of furniture, or gas leaking from a heater. Except in smoldering fires, the combustion reaction itself occurs in the vapor phase, where fuel vapor and oxygen (O₂) in the air can mix. The reaction is rapid, usually taking a few hundredths of a second. The speed of burning and hence the intensity of the fire are usually governed by the rate at which fuel vapor and air enter the flame, where temperatures are high enough to initiate their reaction. Most accidental fires involve such “diffusion” flames, as opposed to “premixed” flames.

*Portions of this chapter have appeared in modified form in Clarke (copyright, 1986)⁵⁰ and are published here with permission.

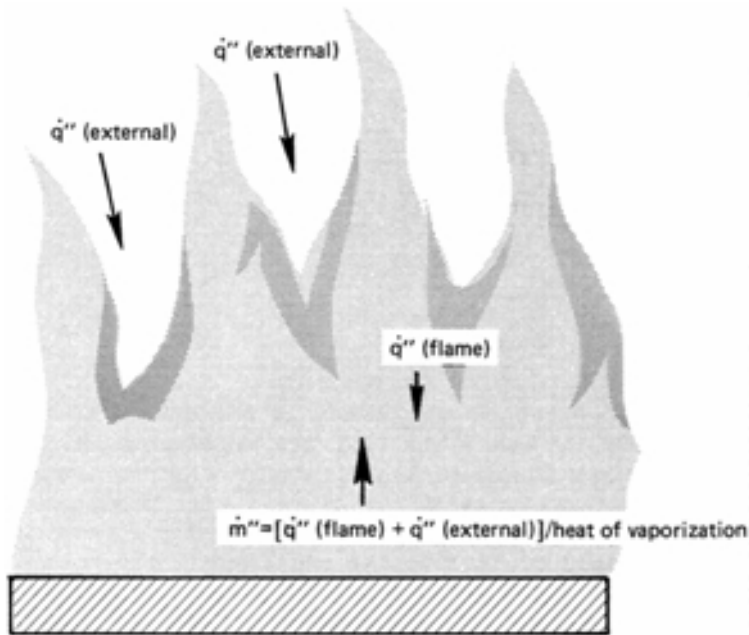


FIGURE 2-1 Schematic of burning fuel surface.

The important transport processes in a diffusion flame are shown in [Figure 2-1](#). Fuel vapor is produced when heat from the flame radiates back to the fuel surface. The hot vapor rises, mixes with air entering near the base of the flame, and ignites. This buoyant expansion in turn creates turbulence, which causes more air to be entrained. A sizable fraction of the heat produced by combustion appears as radiant energy, some of which is absorbed by the fuel surface beneath, so the evolution of fuel vapors continues. Adjacent surfaces are also heated until they are hot enough to evolve combustible amounts of vapors; this is how the flame spreads. Diffusion flames rarely produce totally oxidized products, such as carbon dioxide (CO_2) and water in the case of hydrocarbons; fuel that is burned incompletely gives rise to visible smoke, as well as carbon monoxide (CO). The amount of visible smoke produced varies somewhat with the availability of O_2 ; but the tendency to produce smoke also varies widely among materials.

If the fuel is a volatile liquid, very little energy is required to vaporize it. But the fuels in most accidental fires are polymeric, solid materials, and these generally must be thermally decomposed (pyrolyzed) to yield combustible vapors; because chemical bonds must be broken, this takes much more energy than does volatilization of flammable liquids. The surface of a burning solid usually either melts or chars. If it melts, the surface will remain well below the flame temperature, because much of the heat that the pool absorbs from the flame is carried off in the volatilization process. If the fuel bed chars, heat energy might have to penetrate into the interior of the sample to generate fuel vapors, and the surface will be correspondingly hotter, but still below flame temperature. Char-forming fuels often pyrolyze in two stages: most of the readily volatile material is driven off, and then the char left behind decomposes.

A TYPICAL COMPARTMENT FIRE

The following discussion is restricted to fires in compartments, e.g., an enclosed space. Indoor fires are by far the most important of these, with respect to safety. In addition, compartments in buildings, ships, and planes catch and hold heat and combustion products; this increases a fire's severity, both physically and in its impact on those exposed.

To be life-threatening, a compartment fire must be of at least some minimal size. It usually will have begun small (e.g., with a dropped cigarette, a match in a wastebasket, or a frayed electric connection), but later spread to involve a major fuel source, such as an item of furniture. Such a fire will quickly exhaust the available O₂ in a normal room, and air for further burning will have to be supplied through a doorway or window. The hot combustion products rise from the fire, entraining additional air and forming a distinct, hot, smoky upper layer just below the ceiling, which will deepen as the fire continues to burn. When the hot layer extends down to the top of a doorway, open window, or other vent, smoke will begin to spill out of the room, some of it into the rest of the building. Doorways and windows provide both the air needed for continued combustion and a path for combustion products. Relatively cold air

flows in through the bottom part of the vent and hot fire gases flow out through the top part. Assuming that available fuel is sufficient to consume all the available air, a steady state will eventually be reached in which the burning rate is limited by the rate at which new air is supplied. For a fire whose air is supplied by a normal doorway, 80 in. (203 cm) high by 36 in. (91 cm) wide, the maximal fire intensity is about 2-3 MW.¹¹⁶

The availability of air influences the products of combustion, as well as the intensity of a fire. When a fire is relatively small, and excess air is available, relatively little CO is formed. As the fire grows, it becomes more difficult for air to reach all parts of the flame while the vaporizing fuel and partially oxidized products are still hot enough for further reaction. As the fire approaches its maximal size, O₂ depletion becomes pronounced, the fraction of CO in the smoke increases appreciably, and complex pyrolysis products are likely to appear (in particular, products that would be oxidized further if more O₂ were available). For this reason, the toxicity of smoke from a fire usually depends on the intensity of the fire and certainly on the availability of air. A small fire might produce mostly CO₂ and water vapor and little else; smoke from the same material burning near flashover conditions (see below) can contain large quantities of CO and unoxidized pyrolysis products. The amount of ventilation, not the size of the compartment, controls the fire's eventual rate of energy output. Compartment size does, however, influence the rate at which the fire grows and the likelihood that it will spread beyond the compartment. As the upper part of a room becomes filled with very hot combustion products, this hot layer, like the flame itself, radiates energy to the fuel bed. The extra radiant heat makes the fuel burn faster than it would otherwise. Combustible items some distance from the original fire are also exposed to the radiation from the hot layer, so they will be ignited sooner than they would otherwise. This phenomenon constitutes a major threat to anyone still in the room; one need not be close to the original fire source to be severely burned by radiation from the hot layer.

Figure 2-2 is a schematic of a room fire, showing the development of the hot upper layer and the flow of hot and cold gases through a vent. Much of the heat

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

generated is carried out the doorway by the hot smoke. The rest goes either into the ceiling and the portions of the walls in contact with the hot layer or into the room and its contents by radiation (from both the flame and the hot layer). As the room's surfaces become hotter, they themselves begin to radiate heat back into the room. The net result is that all combustible materials in the room are heated. If their ignition temperatures are reached before the initial fuel supply is exhausted or the fire is extinguished, burning will no longer be confined to one item; the whole room will become involved in flames. This phenomenon, called flashover, is the typical result of an unchecked fire in a residence or a commercial occupancy that contains an abundance of combustible materials. At flashover, more combustible fuel vapor is being produced than can be consumed by the air coming in, so hot vapors are carried out the doorway, where they burn as they encounter more air. Combustible materials in adjacent spaces can then be ignited by flames emerging from the original fire compartment. Even where such additional combustible material is not available, the production of heat will increase dramatically, because additional air is available. Obviously, a flashed-over

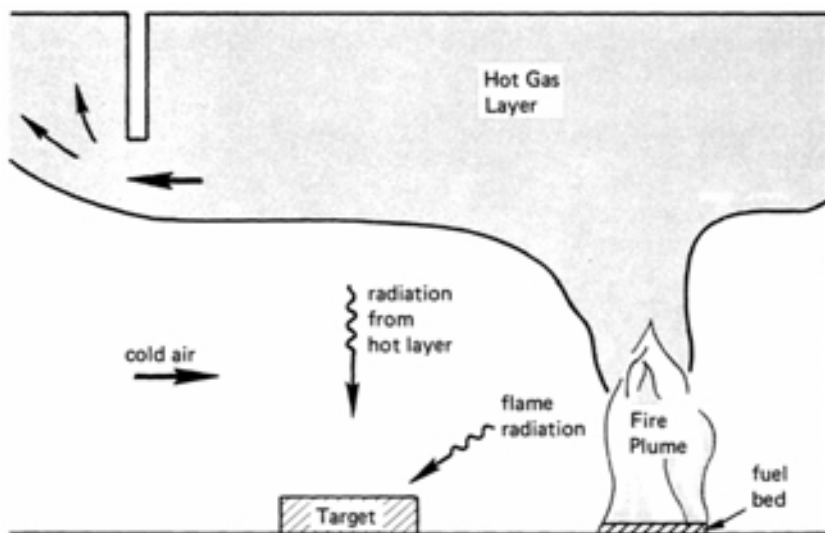


FIGURE 2-2 Two-zone schematic of fire burning in enclosure; doorway at left.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

room is difficult for firefighters to approach, so there is often little opportunity to apply water to the burning fuel in the room of origin.

The consequences of any large room fire are potentially serious. The temperature of the hot gases coming out of the room as the fire approaches flashover typically exceeds 700°C; fuel is consumed at rates of around 0.5 kg/s; CO content of the smoke might be 5%—high enough for a few breaths to be disabling or lethal. Such a fire produces hot gases at several cubic meters per second, so an entire floor of a building can be filled with smoke within a few minutes. In such a situation, the magnitude of the hazard is dominated by the size of the fire. No matter what materials are burning, the threat is acute; no big fires are safe.

FIRE HAZARD ASSESSMENT

DEFINITIONS: RISK AND HAZARD

Risk and hazard are defined in various ways. As used in this document, fire hazard is the potential for exposure to a fire or its products. Thus, the relative hazard posed by two materials is the relative potential for exposure they offer. Fire risk is the probability that a given fire outcome will occur. A discussion of fire risk, like that of any class of risk, needs to include both the likelihood and the severity of the event.^{57 195} The Committee does not concur with the American Society for Testing and Materials, which blurs the terms “hazard” and “risk” and defines “fire hazard” as a fire risk greater than acceptable.¹³

It is impossible to discuss the fire hazard associated with a product without knowing the circumstances in which it is used and the fire conditions to which it will be exposed. These circumstances together constitute the fire “scenario”⁵² for which hazard is to be assessed.

The simplest scenario would be a fire involving a small quantity of combustible material in an essentially closed compartment and no fire spread to neighboring objects. Suppose an occupant is sleeping soundly in the compartment. In a fire, toxic smoke accumulates and mixes roughly uniformly with the air in the compartment.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

The toxic smoke approaches a maximal concentration as the combustible material is consumed. The occupant, who is presumed to be unable to escape, might or might not be able to survive the exposure. Assessing smoke hazard in this scenario involves, for most purposes, only elementary calculations; required inputs are the mass of combustible material involved, the volume of the room, and the lethal concentration of the combustion products, assuming a substantial exposure time. The simple refinement of considering the ventilation rate through the compartment would provide a dilution factor, as well as a limit to the duration of exposure. Given valid smoke toxicity data, such calculations are simple--no computer is needed.

This simple fire scenario represents an extremely common type of fatal fire. In fact, most residential-sized rooms contain many times the amount of combustible material that, if burned, would produce a lethal amount of smoke. In other words, to avoid death in most potentially lethal fires, it is necessary either to suppress the fire or to escape from it. The remaining categories of fire scenarios deal with the possibility of escape. (The detailed ramifications of fire suppression are not considered in this report.)

QUANTIFYING HAZARD

Fire behavior is a time-dependent process, but, even after a fire itself has reached a steady state, the concentrations of smoke in most of the building will continue to change. Hence, it is natural to use time as a basis for evaluating the relative hazard of different fires and of a given fire in different locations. This is generally done by identifying some temperature or smoke concentration that is unacceptable for safety and determining how long the fire in question takes to reach those points.²⁰⁶

Figure 2-3 shows a generalized fire growth curve, where the ordinate is a measure of the intensity, or size, of the fire. The figure could represent the upper room temperature in a room in which an item of fuel was ignited with a match. Little energy is generated at the outset, but eventually the fire becomes large enough to begin heating the room. The average room temperature, which reflects the size of the fire, begins to increase

rapidly. This corresponds to the steep middle portion of the curve. Finally, the fire will reach its maximal size. This limit will be reached either because the entire surface of the item is involved or because air cannot enter the doorway any faster. In either case, the temperature will approach a limiting value, governed by the relative size of the fire and the rate at which hot gases escape from the doorway. If Figure 2-3 were drawn for a longer period, the temperature would eventually decrease as the item of fuel burned itself out.

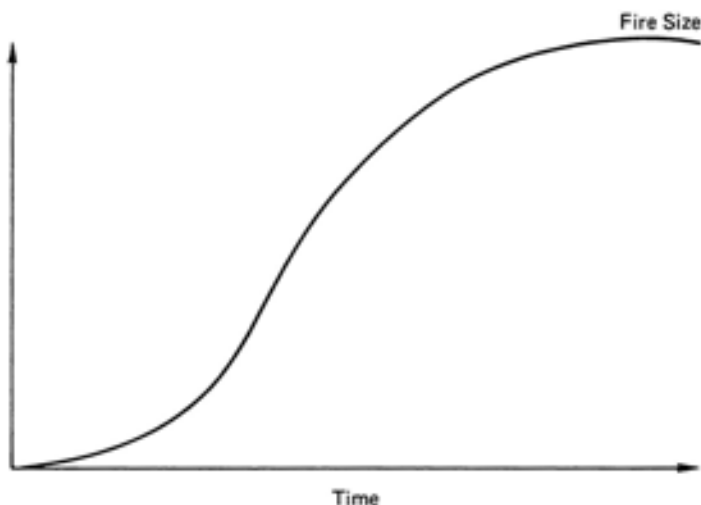


FIGURE 2-3 Typical growth of room fire.

Figure 2-4 shows fire growth curves from different fuel packages--say, two sofas. As the temperature increases, it reaches the upper limit of possible escape, shown here arbitrarily as 100°C. The sofa represented by curve 2 produces this temperature at time t_2 , and the slower-burning sofa represented by curve 1 somewhat later, at t_1 . The difference between these two times is a measure of the relative hazard posed by these two sofas in this particular room environment and for this ignition scenario. Choosing a critical temperature much higher than 100°C (or much lower) would change the difference between t_1 and t_2 . This illustrates that the perceived performance of materials can depend heavily on the chosen criterion of hazard.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

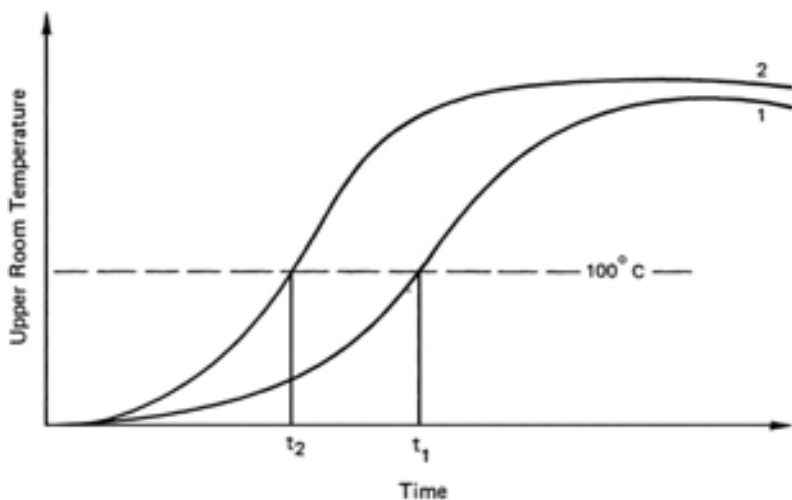


FIGURE 2-4 Comparative growth of two fires burning in room, showing time taken in each case to reach 100°C.

The situation is different when one wishes to evaluate hazards associated with the smoke. Figure 2-5 shows the same fire growth curve as Figure 2-3, with a dotted curve added to show smoke production. Smoke production continues to increase very steeply even after fire temperature has reached a constant value. In effect, the amount of smoke produced is proportional to the integral of the fire-size curve with respect to time.

Unlike thermal hazard, the amount of smoke that represents untenable conditions is different for each material. The toxicity of the smoke must be measured by some appropriate method, and the “toxic” concentration for a given material must be determined. Dose can generally be related to concentration and time, so it is possible in principle to identify the point on the smoke concentration-time curve that corresponds to the arrival of unacceptably toxic conditions. Then the two materials can be compared as they were above (see Figure 2-6).

Smoke also interferes with visibility. It is possible to estimate relative smokiness of materials by measuring the light attenuation produced by the smoke from a known mass of sample--the so-called mass optical density. In a manner analogous to determining the onset

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

of toxic conditions, one can then estimate when, in a developing fire, the smoke will be dense enough to block sight-directed escape.¹⁸⁹ In reality, however, smoke might effectively impede vision at concentrations below those at which it blocks light transmission, if it irritates the eyes; no adequate biologic model is available for assessing such properties.

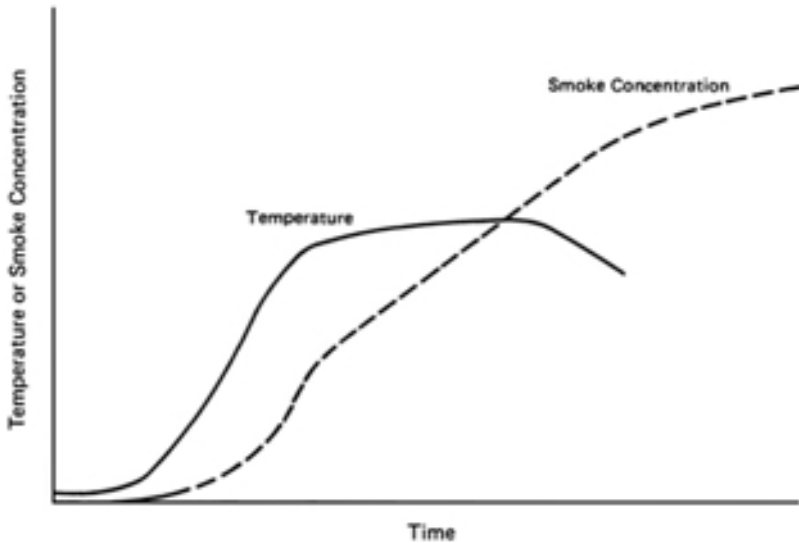


FIGURE 2-5 Growth of temperature and smoke concentration as functions of time.

The time available for escape or rescue begins when the fire is detected. For those in the room of origin of the fire, this detection might occur as soon as the fire starts. In many cases, however, exposure does not begin until later, e.g., when the hot upper layer descends far enough from the ceiling to be breathed. Smoke is usually the first sign of fire detected, either by those exposed or by a smoke detector. The properties of the smoke, and hence of whatever material is producing it, influence how readily it can be detected. Obviously, the sooner the fire is detected, the greater the fraction of the available time that can be used for escape.

Three kinds of information must be available for time available for escape (TAE) to be determined:

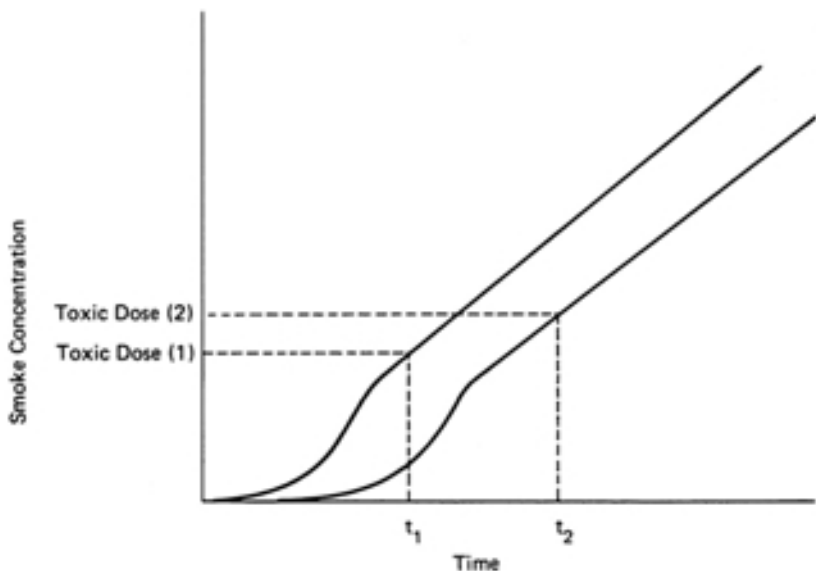


FIGURE 2-6 Comparison of smoke production and development of toxic smoke dose for two different materials.

- The unacceptable temperature or amount of smoke toxicity.
- The temperature, amount of smoke, or time at which the fire is detected.
- The fire or smoke growth curve.

The first is derived (directly or indirectly) from exposure of a test animal to hostile conditions of heat or smoke. The second depends on the scenario. The third can be obtained either from full-scale burn experiments or, in many cases, analytically from small-scale data, laboratory data, and knowledge of the fire scenario. Calculations of fire growth (fire modeling) are discussed in more detail in [Chapter 3](#).

Computation of TAE permits one to compare the relative hazard posed by products in the same application; a ranking of TAEs is a ranking of hazard for the scenario under consideration. TAE alone, however, does not

determine whether a difference in hazard is significant and does not determine the degree of “safety” of a given product. For example, if product A offers 2 min of available escape time more than product B, but product B provides 50 min of escape time when 10 are required, then the difference will be relatively unimportant. If product A offers 5 min and product B 3 min, but escape requires 10 min, then neither product is acceptable.

TIME NEEDED FOR ESCAPE

Determining whether a fire situation is survivable requires knowing the time needed for escape (TNE), as well as TAE. The escape margin, TAE - TNE, is a measure of safety.¹⁶⁷ A negative escape margin is inherently bad; the larger the margin, the better, although, as discussed above, it is possible to reach a point of diminished significance.

Although the effect of smoke on visibility (discussed in the next section) plays a role, TNE depends almost entirely on the nature of the structure, the capabilities of those exposed to the fire, and human behavior. In short, it is not a function of a material's fire or smoke properties, so it is of only indirect interest to this study, although it is critical to the proper use of the results of fire hazard assessment.

Figure 2-7 illustrates the conceptual framework of fire hazard assessment modeling. The five components labeled N1-N4 and A5' permit computation of the time needed (N) for escape; the seven labeled A1-A7 permit computation of the time available (A). N1, occupant location and condition, includes such information as how far occupants are from exits or refuge areas, whether they can escape unaided, and their expected ages. N2, the decision/behavior model, permits prediction of how the occupants will behave in a fire emergency once alerted to the fire; how and when they are alerted depends on the protection system, N3. The evacuation model, N4, predicts how long the building population will take to reach safety in a given layout (A5'). The output of N4 is thus TNE.

The “exposure response evaluation” compares TNE with TAE, which is computed from the “A” components (discussed in detail in the next section) by determining when fire

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

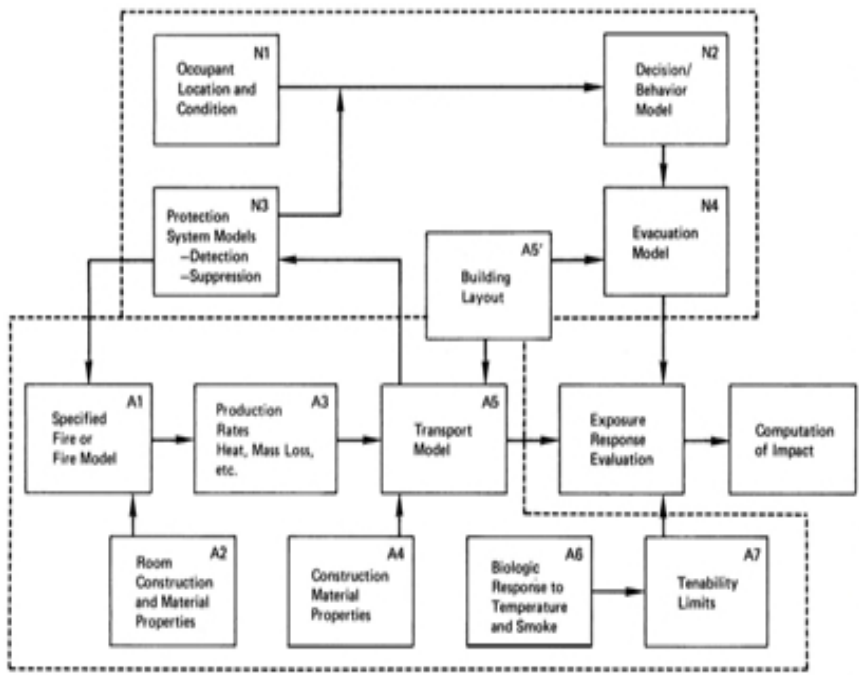


FIGURE 2-7 Major components of fire hazard model.

conditions become untenable (A7). The escape margin might differ for occupants in different locations, and the overall effect of a given fire is determined by computing its impact on occupants of specific locations.

It seems likely that the behavior of those exposed to a fire, and hence the time required to vacate a fire zone, would be influenced by the presence of smoke. Even relatively little smoke is enough to impair visibility, as discussed below, so one can usually expect that visibility will be partly or fully blocked before lethal temperatures or toxic conditions are reached. However, information on the sublethal effects of smoke is not generally available in a form that permits it to be used in detailed predictions of occupant behavior (N2) and the corresponding evacuation times (N4). It seems reasonable to assume that evacuation will be slowed if visibility is impaired or effectively blocked and that the TNE should be increased over what it would be in clear air. The simplest way to do this is to multiply the estimated TNE by some appropriate “safety factor.” Visibility is usually blocked early in a fire, so the most conservative assumption is that the safety factor must always be applied; hence, the detailed smoke properties of a given set of burning fuels do not have a strong influence on TNE. In other words, it is assumed that evacuation will always be accompanied by poor visibility.

TIME AVAILABLE FOR ESCAPE

As discussed above, the sublethal effects of smoke from various materials are not understood in sufficient detail for their influence on TNE to be predicted with confidence, and it has been suggested that those effects and reduced visibility be approximated by applying an appropriate multiplier, a “safety factor,” to a computed TNE.

If it is assumed that TNE is a constant for a given scenario, usually independent of the burning material, and that, in contrast, the fire and smoke properties of a given material influence TAE, then, of a series of materials postulated to be burning in a given scenario, that with the largest TAE offers the greatest opportunity of escape. Hence, TAE becomes a surrogate for the relative degree of hazard that a material offers in the scenario under study.

Without a knowledge of TNE, it is not possible to say whether a material provides acceptable fire performance; therefore, in using TAE as a measure of relative hazard, one should not infer that it represents a degree of safety. However, computing TAE does constitute a method for assessing the overall fire hazard of a material or product in a given scenario and comparing it with that of others intended for the same use.

The details of fire hazard depend on the scenario. For example, a rapidly developing flaming fire whose products accumulate in a relatively confined space, such as a small apartment, produces a well-defined hot gas layer that descends rapidly. There is little opportunity for the smoke to lose energy to the walls and ceiling, so the layer is very hot. Once the hot layer has descended to a few feet above the floor, it is difficult to escape without coming into direct contact with it. Regardless of its chemical composition, this layer poses an immediate threat because of its temperature. In a larger space, or if the fire is burning more slowly or perhaps smoldering, the layer is cooler. In fact, a distinct upper layer might not be apparent, because high temperature is what gives the layer its buoyancy and results in stratification. In such a case, the toxic properties of the smoke become important, because they, not the temperature, determine the tenability of the compartment.

In larger structures, it is common practice to provide barriers to the free passage of smoke or fire between floors, into exitways, and often between groups of rooms. (Modern apartment buildings, for example, have fire walls between apartments, but not as interior partitions.) The smoke from a fire might leak into spaces far from the fire, but rarely as a hot buoyant stream, the form in which it exists when it is nearer the fire. Therefore, the primary threat away from the fire is usually smoke toxicity, not heat.

One uses scenarios involving multiple burning items when one wishes to examine the hazard attributable to an item that can burn only after exposure to a fairly large ignition source. Examples of single-item and multiple-item scenarios are described below.

BURNING OF A SINGLE ITEM

Consider a fire ignited in a compartment of 250 m^3 (which corresponds roughly to $1,000 \text{ ft}^2$ of floor area and a normal 8-ft ceiling) and restricted to one relatively large item, such as a heavy upholstered chair or loveseat. Because the fire is restricted to an item of furniture, the fire properties of the rest of the room are unimportant, although the thickness and thermal properties of the walls and ceiling should be known, if one is to determine how much of the heat energy of the fire is lost to these surfaces. As for the furniture itself, its burning rate (heat release and mass loss rate) must be measured or calculated from small-scale test results. These data constitute the input to A3 in Figure 2-7. Because the scenario envisions monitoring conditions in the same room as the fire, the layout of the building (A5) is not very important in this calculation. Neither are construction material properties (A4), other than those already mentioned for A2. Assume that an air temperature of 100°C is the upper limit for human escape from the compartment. Finally, suppose that smoke toxicity data (A6) available on the furniture material fairly reflect the toxicity under actual burning conditions. The most useful measurement is the $L(\text{Ct})_{50}$, the concentration-time product required for death to occur in 50% of animals exposed to the smoke. In a smoke toxicity test, this product is obtained by continuously monitoring the smoke concentration to which the animals are exposed and reporting the time integral of this quantity when the animals die. (This takes no cognizance of the possibility that animals die after exposure.) The tenability limits for temperature and toxicity are determined in A6 and constitute A7.

The simplest burning scenario is one in which a moderate fire begins on the furniture and does not spread appreciably. If the fire size is 100 kW --i.e., about 0.6 m in diameter--it can be shown that the hot smoke will have filled the room to a depth of 1 m from the floor in about 6 min; the temperature of the hot layer will have reached 100°C after 11 min.⁵⁵ Hence, by the temperature criterion mentioned above, the environment will have become lethal in 11 min.

Whether smoke toxicity becomes a problem sooner depends on whether the occupants have been exposed to the smoke throughout the course of the fire and on how toxic the smoke is. For smoke toxicity to be the immediate threat in this scenario, the atmosphere must become lethally toxic before it becomes lethally hot.

When the burning rate of the fire and the associated mass loss rate are known, it is simple to compute the average smoke concentration in the hot layer. Assuming that the occupants have been exposed to the smoke from the time when the hot layer was at the 1-m level, the time to receive a lethal dose of smoke, TAE, is given by the integral over time, dt:

$$L(Ct)_{50} = \int_{t_1}^{TAE} C_s(t)dt,$$

(1)

where

$L(Ct)_{50}$ is the lethal dose determined from a laboratory toxicity measurement,

t_1 is the time at which the smoke reaches the 1-m predetermined level, and $C_s(t)$ is the smoke concentration, expressed as a function of time.

The smoke concentration, $C_s(t)$, is a function of the mass loss rate, $\dot{m}(t)$:

$$C_s(t) = \frac{1}{V} \int_0^t \dot{m}(t)dt.$$

(2)

TAE is plotted as a function of $L(Ct)_{50}$ for this scenario in [Figure 2-8](#). If the furnishing material has a smoke $L(Ct)_{50}$ below about 200 g·min/m³, death from smoke toxicity could be expected to occur before conditions were thermally untenable; otherwise, in this scenario, the thermal hazard is more immediate.

It is also instructive to compute the smoke density, and hence the visibility, in the upper layer when it has descended to 1 m above the floor. Depending on the mass optical density of the fuel, visibility in the upper layer after 6 min of burning will be no more than about 2 m, and more typically about 0.8m. If visibility is

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

restricted to 0.8 m, a person cannot see any farther than an outstretched arm, and even 2 m of visibility is likely to be of little real help, in that typical room dimensions are at least twice as large.

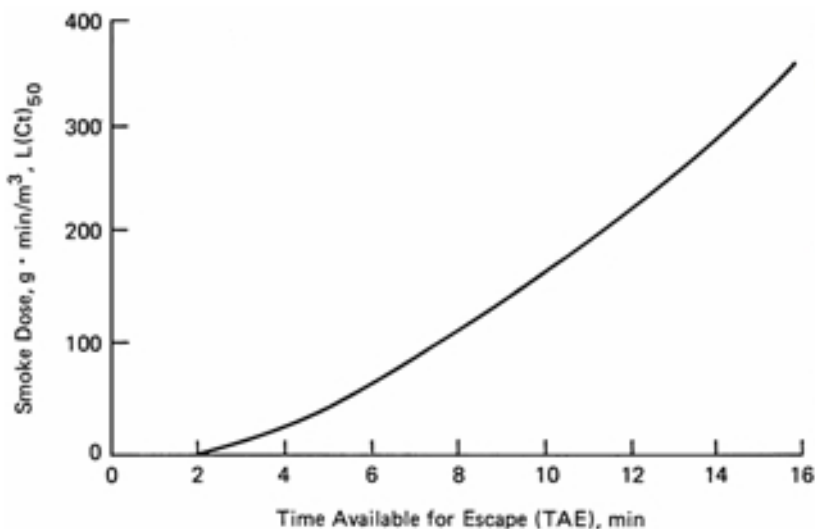


FIGURE 2-8 Buildup of lethal smoke dose with time, single-item scenario.

Studies by Jin¹⁰⁹ in Japan have shown that, when smoke optical densities are above 0.25 m^{-1} (i.e., when visibility is less than about 5 m), movement by those exposed is slowed appreciably. For a fire to produce so little smoke in the foregoing scenario, it would have to have a mass optical density of $20 \text{ m}^2/\text{kg}$, which is about one-tenth the smoke-producing potential of a typical furnishing material, such as polyurethane.

If, instead of a flaming fire, the fire on the furniture is smoldering, too little heat will probably be generated to maintain a stable upper layer, and the smoke will disperse generally uniformly through the compartment volume. TAE (including time needed to detect the fire) is given by:

$$L(Ct)_{50} = \int_0^{\text{TAE}} C_s(t) dt.$$

(3)

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

For a smoldering fire that does not change in size (\dot{V} is constant), Equation 2 and Equation 3 can be solved to yield:

$$\text{TAE} = [2L(\text{Ct})_{50}(\dot{V}/\dot{V}_0)]^{1/2}.$$

(4)

For one that grows linearly with time ($\dot{V} = kt$), the same equations yield:

$$\text{TAE} = [6L(\text{Ct})_{50}(\dot{V}/k)]^{1/3}.$$

(5)

The higher order of time dependence on mass loss rate, the less sensitive TAE is to changes in $L(\text{Ct})_{50}$. This is the case for flaming fires, as well as smoldering fires, although the former are complicated by the hot-layer descent discussed above.

Most fire scenarios are too complex for calculations of TAE to be expressed in analytic form; computer-based numerical solutions are usually required. The logic of the procedure, however, is the same. The material properties that control burning rate and fire growth are at least as important as are smoke toxicity characteristics in determining TAE--the fire properties alone control the hazards associated with thermal exposure and the rate at which smoke is produced.

BURNING OF MULTIPLE ITEMS

Most real fires involve several items. In some cases, the sequence of ignitions seems idiosyncratic--it is as easy to envision drapes igniting from a burning chair as the reverse. In other cases, however, the sequence is likely to be predictable--combustible materials, such as plastic pipe or wiring, behind a wall are much more likely to be exposed to heat from a fire in the room than to be ignited directly by a small ignition source.

Figure 2-9 shows the buildup of temperature in a room as the result of a known fire, the standard time-temperature curve for the ASTM E119-83 fire endurance test.¹²

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

It also shows the buildup of temperature behind a wall of 5/8-in. gypsum wallboard. These two curves are inputs A1 and A2, respectively, for determining the smoke production rate of the room fire and the concealed combustible materials.

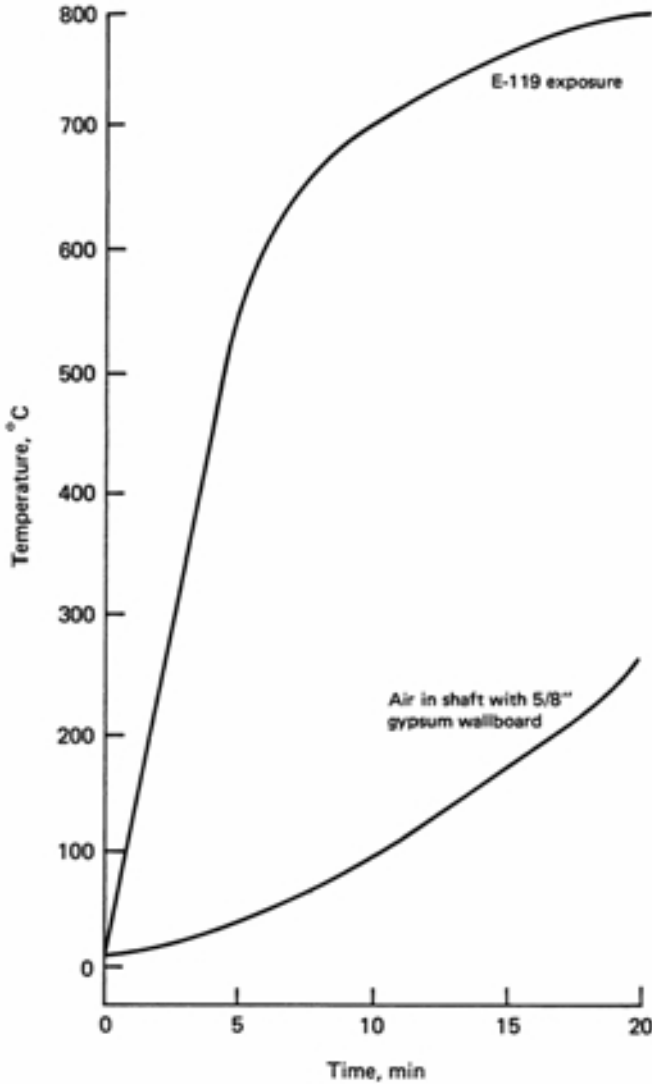


FIGURE 2-9 Time-temperature profile of fire simulating ASTM E119 fire endurance test and of cavity behind gypsum wallboard.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

It should be clear that the temperature in the room becomes untenable long before the region behind the wall warms appreciably. At some distance from the fire, the sole hazard of concern is the toxicity of the smoke. For the first 15 min or so, smoke issues only from the room fire. When the temperature behind the wall is high enough, the combustible materials will begin to decompose, then to ignite and burn. The data necessary to characterize this process include ignition temperature, mass loss vs. temperature, heat release rate, and amount of hidden fuel. These data constitute part of A2.

The curves in [Figure 2-10](#) show the contribution of the in-room and behind-wall fuel packages to the total smoke produced. One must know the toxicity of the smoke from both fuel sources to predict the effect of the behind-wall material on TAE. Formal expressions have been proposed by Bukowski⁴² and others to compute TAE associated with fires involving multiple components. TAE can be computed from the equation:

$$\frac{1}{L(Ct)_{50}(1)} \int_0^{TAE} C(1)dt + \frac{1}{L(Ct)_{50}(2)} \int_0^{TAE} C(2)dt = 1$$

(6)

where (1) and (2) refer to the smoke generated from the in-room and behind-wall fuels, respectively. In practice, the smoke production curves are followed, integrated, and normalized with respect to the toxic dose (determined in advance by small-scale tests). When the normalized contributors sum to unity, TAE is deemed to have been reached. The smoke concentration depends both on the mass loss rate of the fuel and on the point in the building at which the hazard is being assessed. Hence, knowledge of the building layout (A5') and the construction material properties (A4) might also be needed for this computation.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

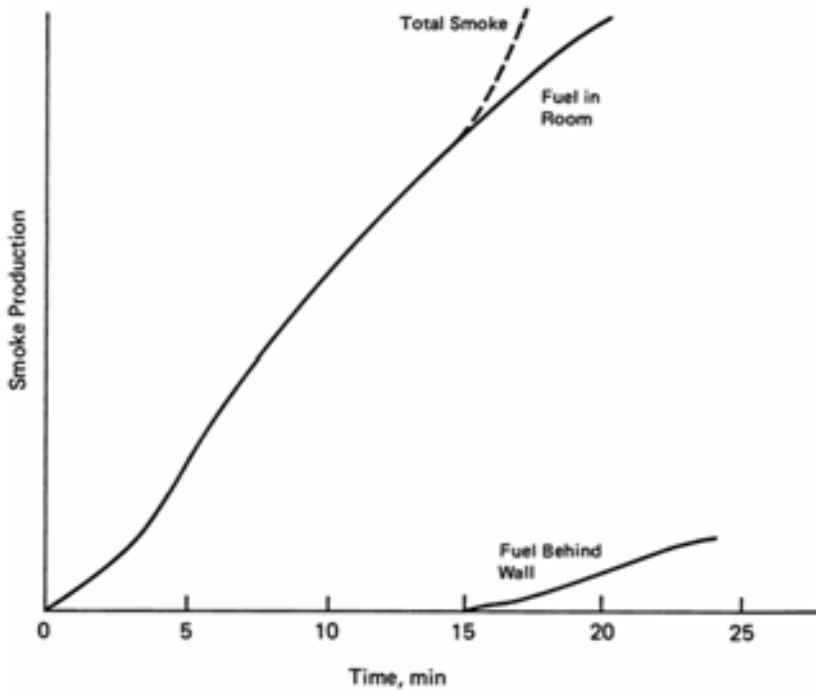


FIGURE 2-10 Smoke production for fire burning in room and igniting combustible materials behind wall.

In comparing two alternative materials for the same use behind the wall, it is possible to compute a difference in TAE associated with the change from one material to another. How this is accomplished is the subject of [Chapter 6](#).

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

3

STATUS OF FIRE HAZARD MODELS AND TEST METHODS

INTRODUCTION

A fire hazard model permits calculation of the time available for escape for a given occupant under a given set of conditions. The first step is to specify a detailed fire scenario, which can be selected as a “worst case,” a “most probable case,” or a combination of the two.

A major class of fire scenarios consists of fires that continue to burn, with or without growth, beyond the point where lethal concentrations of fire products (the products of both combustion and pyrolysis) reach occupant locations and escape paths. For escape to be possible, a fire must be detected. Eventually, the escape path will be blocked (by obscuring of vision, by irritants, by toxicants, or by heat). The interval between detection and blockage of escape is the time available for escape (TAE). If TAE is greater than the time needed for escape (TNE), the occupant can escape. A hazard model of such a fire must calculate several entities: the time at which the fire is detected, the fire size and distribution of fire products at that time, the postdetection TAE, and the postdetection TNE. The toxicity of the fire products influences mainly the TAE. TAE cannot be calculated until the fire condition at detection is established.

For this category of scenarios, TAE might or might not be strongly influenced by the toxicity of the fire products. For example, if the fire is growing and the escape path is effectively blocked because vision is obscured well before lethal conditions are reached at the occupant location or in the escape path, the occupant

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

might live only an additional minute or two if the LC_{50} is increased by substitution of materials. In this example, TAE depends on the obscuring of vision by fire products, but not on their degree of toxicity.

If loss of visibility (or the presence of irritants) in the escape path is not crucial, the LC_{50} becomes important. For example, the occupant is trapped, and toxic products are building up around him or her; after 10 min, the rescuing firefighter arrives through the window. Or the occupant is willing to grope his or her way through the escape path, in spite of lack of visibility. In such cases, the greater the LC_{50} , the larger the TAE. However, the dependence of TAE on LC_{50} is less than linear, even for a steady fire, as shown in [Chapter 2](#). For a rapidly growing fire, a doubling of LC_{50} might increase TAE by only 10 or 20%. For each specific case, a mathematical model must be run to determine the degree of benefit.

Of course, some fire scenarios include an extremely rapidly growing fire, perhaps involving flammable liquids, in which TAE is much less than TNE. In such a case, the LC_{50} is irrelevant to the result. The modeling of such fires will not be considered further here.

DETECTION MODELS

Assume that a ceiling-mounted detector is a known distance from an initially small, growing fire. In principle, a model can calculate the size of the fire at the time the detector is activated.^{9 72}

The effects of walls, doorways, corridors, etc., on the detector response introduce complications. For the simplest case, one may assume a very large, flat-ceilinged room that contains both the fire and the detector. An algorithm has been worked out for this case and presented as a family of curves;³¹ it could easily be computerized. The critical fire size (the size when the detector goes off) depends on the following variables:

- Ceiling height above fire.
- Distance from fire to detector.
- Rate of fire growth.
- Characteristics of detector.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

- Characteristics of smoke (if a smoke detector is assumed).

Either a smoke detector (more sensitive) or a heat detector (less prone to false alarm) might be used. To illustrate the magnitudes involved, assume that the ceiling is 10 ft (3 m) above the fire and that a smoke-detector is 20 ft (6.1 m) from the fire axis. Assume that the fire is following a parabolic (t^2) growth law, reaching 1 MW in 10 min, that the combustible material produces a type of smoke easily detectable by the detector used, and that the detector is designed to have very little resistance to smoke entry. The design curves then indicate that the detector should respond when the fire reaches about 100 kW. If, instead, the fire is assumed to grow 4 times as rapidly (still following a t^2 law), the detector will not respond until the fire is twice as large (200 kW). If it is assumed that the fire grows at the original rate, but that the combustible material produces a less easily detectable type of smoke and that the detector is designed with substantial resistance to smoke entry, then the detector might not respond until the fire is, say, 5 times as large (500 kW). This would occur 3.9 min after it reaches 100 kW. In the original case, if the detector were 40 ft (12.2 m) away from the fire axis, instead of 20 ft (6.1 m), the fire size at detection would be nearly 3 times as great, and detection would occur 2.2 min later.

If one assumes that a heat detector of known characteristics is used, instead of a smoke detector, similar calculations could be performed; the fire size at detection would be substantially greater, and detection would occur several minutes later.

The foregoing method of calculation is based on a very large compartment. If the fire compartment is small and the detector is in the fire compartment (rather than in an adjacent compartment), response will occur much sooner and while the fire is much smaller. However, the calculation is complicated, because the prefire condition of the compartment will generally involve a vertical temperature gradient, i.e., the temperature will be higher near the ceiling than near the floor. Accordingly, if the fire is small and produces only a weak plume with insufficient buoyancy, the smoke will tend to stratify somewhere below the ceiling (and below the detector). Of the published

modeling approaches, only the very complex "field model" can deal with this. However, for a small room with an 8-ft (2.4-m) ceiling, a rule of thumb is that, once the fire is larger than a few kilowatts, the plume will be tall enough to reach the ceiling, spread out under it, reach the detector, and activate it within a minute or so.

If the detector is in an adjacent compartment, a computerized fire model (see the next section) is used. It should calculate the accumulation of hot fire products under the ceiling, the loss of heat from these products to the ceiling, the deepening of the hot layer to below the top of the doorway that leads to the adjacent compartment, the mixing and dilution of the hot gases as they flow through the doorway, and the eventual filling of the adjacent compartment.

Of course, detection might be by a person, rather than by a device. Any of the five senses can be involved. The location of the person relative to the fire is crucial. For safety purposes, it is common to assume a "credible worst case"--that the people are at a remote part of the structure, relative to the fire. The key element would be the time it takes smoke to move through the building. The modeling of this case is discussed later.

MODELS FOR TIME AVAILABLE FOR ESCAPE

THE HARVARD MODELS

The Harvard fire computer code has several variants, two of which, Mark 5.3 and Mark 6, are discussed here. Mark 5.3 treats one compartment with openings, and Mark 6 treats up to five interconnected compartments, all on one level. Even the simplest version involves some 50 variables and requires a computer larger than the largest microcomputers available in 1984. A standard run on a VAX 11/780 computer takes about 1.5 min of CPU time with the Mark 5.3 version and perhaps 15 min with the Mark 6 version.

The necessary inputs consist of routine and nonroutine items. The routine items include dimensions of compartments and their openings, whatever forced-ventilation flow is present, thermophysical properties of ceilings

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

and walls, locations and characteristics of detectors, and locations and ignition characteristics of combustible items present but not originally ignited. The nonroutine items are related to the assumed fire. The model can accommodate either a steady or a growing fire. The modeler must specify the size or growth rate of the fire, in the absence of radiative feedback from the compartment to the burning object. The modeler must also specify the sensitivity of the burning rate to radiative feedback. (The model computes the resulting burning rate). Other needed items are burning efficiency, fraction of energy that leaves the flame as radiation, fuel-air stoichiometry, and mass fraction of carbon monoxide (CO), smoke, or other important species in the fire products. These nonroutine items, often difficult to obtain, are discussed later.

The model combines the inputs with a number of built-in simplifying assumptions and then calculates the history of the fire. One assumption is that each compartment divides into a hot upper zone and a cold lower zone, each uniform (but changing with time) in temperature and composition. Mixing across the horizontal interface between zones is assumed not to take place, except by fire plumes. A second assumption is that each fire plume entrains air in a standard fashion according to a formula that takes no cognizance of small fluid-mechanical disturbances (e.g., eddies and turbulence), which can have substantial effects on entrainment rate. In summary, the fluid-mechanical aspects of the fire are approximated rather roughly.

The outputs of the model consist of a number of time-dependent quantities, including the rate of deepening of the hot layer in the compartment, the ignition time of objects after the first object, the rate of gas flow out of the compartment, and the concentration of species in the outflowing gas.

Mark 5.3 has been compared with data from several full-scale fire tests, with fairly good agreement for hot-layer temperature, layer height, and gas outflow rate.¹⁴⁷ However, ability to predict CO and optical density is often limited by inadequacies in input data. As for Mark 6 (multiple compartments), no comparison with fire test data has yet been published.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Attempts are now being made to improve the Harvard 5.3 model to consider additional effects: radiant heating of the floor, ceiling venting, ignition of the hot layer, mixing between layers, wall burning, ceiling burning, and horizontal spread of a hot gas front in a corridor.

OTHER TWO-LAYER MODELS

The FAST model¹¹⁰ considers essentially the same physical elements as the Harvard model, but formulates equations with a mathematical technique that is claimed to give considerably faster and more “rugged” or “robust” solutions--e.g., those with a lower tendency to give grossly wrong answers or to fail to run to completion--for a wide range of input parameters. Some successful comparisons with data on upper-layer temperatures and interface heights have been made.

The ASET model⁵⁶ is a considerably simplified version of a computer code with many of the same basic features as the codes mentioned above, but requiring less user skill and computer capacity. It is usable only for a single closed compartment with leakage near the floor. It ignores radiative feedback to the burning object and does not accommodate the ignition of more than one object. It allows for energy loss from the hot layer to the compartment in an ad hoc manner. With these restrictions, it computes the time available before the smoke layer deepens to reach the occupant, if burning rate is known.

Zukoski and Kubota²³³ have developed a model limited to two interconnected compartments, of possibly different ceiling heights and exterior openings, with a specified fire in one compartment. It emphasizes the fluid-mechanical aspects of gas motions.

Tanaka,²¹⁵ of the Japanese Building Research Institute, has developed a two-layer computer model similar in many ways to those noted above, but differing in that it can be applied to tall buildings with many compartments. An example involving 50 rooms in a 10-story building has been worked out. There is no forced ventilation, and windows are assumed to be open throughout, which is unrealistic. Wind velocity can be an input. Tanaka stated that the weakest element of his model is the method of calculating gas transport in vertical shafts.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

The model gives results for tall buildings that seem reasonable, but has never been compared with multistory-building fire test data.

The Dayton Aircraft Cabin Fire model (DACFIR) was developed by MacArthur and Myers¹⁴⁰ specifically for fires in wide-body and standard-width aircraft cabins. This two-layer model accommodates cabin openings and forced ventilation. The flame-spread feature is more detailed than in the other models; the horizontal and vertical surfaces of each seat are divided into 6-in. (15-cm) squares. Each square can undergo transitions from virgin to smoldering to flaming to charred, depending on the instantaneous value of the imposed heat flux. The model handles creeping flame spread if the creeping rate is an input. It may be instructed to produce different toxic gas or smoke concentrations from the smoldering elements and the flaming elements. The model has been compared with a series of seven full-size aircraft cabin test fires. It did not do well at predicting areas of fire spread. It predicted toxic gas concentrations in the hot layer (CO, hydrogen cyanide, hydrogen chloride, and hydrogen fluoride) that were generally accurate to within an order of magnitude, but the prediction of time of first appearance of these gases at potentially toxic concentrations was not accurate. The model was also deficient in predicting hot-layer temperatures at the end of the cabin away from the fire.

FIELD MODELS

The models discussed above are all two-layer zone models. Research is also being done on field models, which, instead of dividing a compartment into two uniform zones, divide it into hundreds or even thousands of zones in a three-dimensional array. Such models can predict fluid motions far more realistically, but, needless to say, require extremely powerful computers and do not yet appear to be practical for routine hazard analysis. Two recent examples of field-model studies are those of Cox et al.⁵⁹ and Baum and Rehm.²⁹

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

EXAMPLE OF CALCULATIONS FOR MODEL FOR TAE

Fowell⁸² has used the FAST model to calculate fire development in an apartment in which a loveseat in the living room is assumed to be burning. The burning rate of the loveseat was determined from a measurement in the NBS Furniture Calorimeter.¹²⁶ The living room is open to a hall 9 m (29.5 ft) long, which in turn is open to an occupied bedroom. Assume that the fire is detected by a smoke detector in the hall and that the occupants must then escape through the hall or be overcome.

The model inputs were the following: dimensions of rooms and openings, thermal properties of ceiling and walls, burning rate of the loveseat (grams per second), heat of combustion (18.1 kJ/g), smoke yield (0.03 g/g), and LC₅₀ (32 mg/L). The sequence of calculated events was as follows:

Time	Event
•40 s	Calculation is started (small fire)
0 s	Fire actuates smoke detector in hall
+73 s	Upper temperature in living room is untenable (183°C)
+91 s	Visibility is lost in hall (smoke 1 m--3.3 ft--above floor)
+100 s	Upper temperature in hall is untenable (183°C)
+133 s	Lethal concentration is reached in hall
+153 s	Lethal concentration is reached in bedroom
+166 s	Upper temperature in bedroom is untenable (183°C); fire still developing rapidly

For this scenario, the model predicts that the bedroom occupants had 91 s to escape before visibility was lost in the hall and an additional 9 s during which they could grope their way through the hall before the temperature became intolerable. If they failed to escape in this period, but remained in the bedroom, they would be overcome by toxicants 53 s later or by heat 66 s later. (This assumes an instantaneous effect on the victim when the LC₅₀ concentration is reached, whereas a more sophisticated treatment would use the integrated product of concentration and time and would require information on time to loss of consciousness, which, in the case of humans, is not documented.)

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Consider the sensitivity of TAE to LC_{50} , assumed to be 32 mg/L. If LC_{50} were reduced to 16 mg/L (doubled toxicity), the loss of visibility and excessive temperature in the hall would occur at the same times as before, and lethal concentration in the hall would still not be reached at these times, so escape would be unaffected. However, if the occupants had remained in the bedroom, they would now be exposed to lethal concentrations or higher for perhaps 40 s (instead of 13 s) before being overcome by heat. This conceivably would be important only if the occupants were assumed to be rescued--say, by a firefighter who enters through the bedroom window--between 126 and 153 s after detector actuation. However, if the postulated rescuer arrived sooner or later than that narrow interval, the results would be essentially unaffected by the reduction in LC_{50} . If the LC_{50} were reduced by a factor of 10, instead of by a factor of 2, lethal conditions would be predicted to occur in the hallway before loss of visibility, and TAE would be reduced from 91 s (based on visibility) to only 20 s (based on instantaneous incapacitation when a concentration of 3.2 mg/L is reached).

This modeled scenario could also be explored for sensitivity to other properties of the combustible material, namely, burning rate, heat of combustion, and smoke yield. Clearly, each can affect TAE. In particular, the relative order in which smoke, heat, and toxicants reach critical points will change.

This example illustrates the possibilities of using a model to explore the sensitivity of TAE to all the relevant properties of the combustible material, not only the LC_{50} . Obviously, it depends heavily on the scenario; under some realistic conditions, it might be very insensitive to LC_{50} . In general, smoke toxicity has a relatively small impact on TAE when the fire (and hence the rate of smoke production) is growing rapidly. In such scenarios, the biggest incremental changes in TAE are caused by manipulating the flame spread rate and the heat of vaporization. When the burning rate (and hence the rate of smoke production) is constant, the impact of toxicity and the impact of burning rate on TAE are the same; e.g., doubling one has the same effect as doubling the other. When TAE is controlled by the buildup of smoke, however, the effect of either is less than one might intuitively expect; e.g., doubling the smoke

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

toxicity does not reduce TAE by 50%, but only by about 30%.

Most real fires are characterized by a period of growth followed by a period of relatively constant burning, once the fuel or air supply reaches its limiting value; so calculating TAE in practice must take account of both phases.

MODELS FOR TIME NEEDED FOR ESCAPE

Once a fire is detected, the time needed for escape is controlled by a combination of psychologic, physiologic, and physical factors. Some models, such as EVACNET+,¹²¹ deal with physical factors--specifically, the escape paths available, the time needed to traverse each path, the flow capacity of each path, and the initial locations of the occupants. If these inputs are available for a large building with many occupants and multiple escape paths, the computer program can calculate the evacuation status as a function of time.

However, it is widely recognized that a variety of psychologic and physiologic factors are at least as important as the physical factors represented in models like EVACNET+. For example, response to alarm signals, decision-making, behavioral patterns, male-female differences, physical capability, knowledge of escape routes, experience with fire, effects of reduced visibility, and panic behavior are important. Stahl²¹¹ has developed BFIRE-*II*, a behavior-based computer simulation of emergency egress during fires. Human behavior during fires has been reviewed by Paulsen¹⁷⁴ and by Paul.¹⁷³

Although chemical components of fire products might influence decision-making or physical ability to escape, no escape models deal with this possibility, presumably because the available data are inadequate. Available TNE models do not use LC₅₀ or other toxicity-related data, so they are not discussed in detail here. However, even a crude estimate of TNE, which might not require a computer model, could be sufficient to show whether, in a given scenario, TAE is of the same magnitude as TNE or one is much larger than the other. This comparison will often be enough to show how sensitive the hazard potential of the scenario is to TAE.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TEST METHODS FOR MODEL INPUT DATA

BURNING RATE

Fire-product calorimeters built at the Factory Mutual Research Corporation,⁹⁵ at the National Bureau of Standards,²⁵ at the University of California, Berkeley,⁷⁸ and elsewhere can measure the instantaneous rate of heat release of a fire up to 10 MW in intensity. The principle is to collect the product gases with excess air, to mix them, and to measure their flow rate and instantaneous composition. From the degree of oxygen deficiency or from the total composition, one can calculate the rate of energy release. Simultaneously, the mass loss is measured. When the rate of energy release is divided by the rate of mass loss, the quotient is the heat of combustion per unit mass. (If the combustible material were a pure material that burned completely, the heat of combustion could be obtained from a handbook, but most real combustible materials are composites, burn incompletely, or both.)

One way of using such a calorimeter (essentially an instrumented fume hood) is to place it above an item to be “realistically” ignited and burned, such as a bed or a sofa. Another way is to start a test fire in a suitably furnished “burn room” with an opening and to collect the fire products as they emerge from the opening.

Much useful information for fire models has been obtained with such large calorimeters. But it is a rather expensive way to obtain data, so small-scale tests are highly desirable. Small-scale fire tests are numerous and have been described frequently.^{98 163 190} Each gives some information on the flammability of the item tested, as each blind man who touches a portion of an elephant obtains some idea of what an elephant is like. However, no standard small-scale tests or any known combination of them is adequate for predicting the full-scale burning rate of an item made from the tested material.

One exception to this generalization would be a noncharring combustible material uniformly ignited over a single horizontal surface, for example, a dish of heptane 1 m across or a horizontal slab of polymethyl methacrylate 1.5 m square. In such a case, if one measures the burning

per unit area of a small-scale version--say, 10 cm across--one finds that the burning rate is only 55% (heptane) or 40% (polymethyl methacrylate) of the full-size burning rate.²¹⁹ However, by applying an empirically arrived at radiant heat flux of around 50 kW/m² to the small-scale sample or by burning the small-scale sample in an atmosphere artificially enriched in oxygen, one can make the small-scale sample burn at about the same rate as the full-scale sample. Then, by using the same small-scale test conditions for other noncharring horizontal materials, one can predict full-scale burning rates. This empirical procedure seems to work reasonably well for a number of materials. Research is in progress to develop such a procedure involving flame radiation characteristics of the combustible material. And means of treating char-forming combustible materials are being studied.

Turning from a horizontal to a vertical orientation of the combustible material (for example, a fire-retarded plywood wall), prediction of burning rate, or indeed of whether a fire will propagate or die out, is not yet possible with small-scale tests. The small-scale test that provides the best hope of being useful, when combined with other information, is a rate-of-heat-release test. In this test, a small sample is allowed to burn while being irradiated by an external heater at a specified flux, and burning rate vs. time is measured. Many commonly used household materials char. The burning rate rises rapidly to a maximum and then gradually decreases as the char builds up. Toward the end of the test, the burning rate might increase again, because by that time the sample has heated through. One difficulty is in translating a complex curve like this into a single useful number. A second problem is that, in a real fire spreading upward, the largely radiative flux intensity from the burning plume is the input to the not-yet-ignited material just above the burning region. The rate-of-heat-release test gives no measure of the radiative output of the flame, which varies from material to material. No standard test method measures this radiative flux. Finally, no established theory can combine such data into a prediction of whether and how fast the flame will spread.

Nonetheless, the rate-of-heat-release data give the best available indication of flammability. ASTM Test

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

E906¹⁴ has recently been established as a method to measure rate of heat release. Other, more sophisticated small-scale test methods developed by Tewarson et al.²¹⁸ and by Babrauskas²⁵ permit more accurate measurements of rate of heat release, as well as rate of production of smoke and other species of interest. The same methods can be used to measure ignitability under radiative exposure.

If a building fire is confined to a single compartment, the fire can be controlled through ventilation, and the burning rate can be estimated from a knowledge of the size of the ventilation opening and the fuel-air stoichiometry. A model can then be used to predict fire-product movement through the building without further concern with rate of heat release.

RATE OF PRODUCTION OF SMOKE AND TOXICANTS

As a minimum, the various models require as inputs the burning rate (grams per second) and the heat of combustion, which are obtained or estimated as discussed in the previous section. The gross toxic effect of the mixture of products from a gram of burned material, diluted to a given volume, can be determined in animal exposure tests. However, the fire models can predict local concentrations of any species of interest, such as CO or hydrogen chloride (HCl), if test methods can provide the needed inputs, specifically grams of the species of interest yielded per gram of burned material.

Some species initially formed in the fire undergo change as the fire products move through the building. For example, soot particles can agglomerate via Brownian motion over time. HCl can be adsorbed on the walls of ducts or corridors, and acid mist can settle to the floor. Such processes could in principle be included in the computer model, but initial yields must be known.

The fire-product calorimeters previously mentioned for measuring burning rate^{25 78 95} are easily adaptable for handling any measurable constituent of the fire products. The techniques for these measurements are well known and will not be reviewed here. For each species to be measured, an additional element of cost and complexity is introduced into the fire-product calorimetry procedure;

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

therefore, unless there is a specific need, the data taken are often limited to an indication of the smoke density by optical transmission through the products and CO and carbon dioxide (CO₂) measurements by infrared absorption.

The small-scale rate-of-heat-release apparatuses^{25 218} are also easily adaptable to such measurements. However, the results are very sensitive to ventilation conditions. For example, the molar CO:CO₂ ratio in the products of well-ventilated flaming combustion is around 0.002:1 for wood, 0.04:1 for rigid polyurethane foam, and 0.18:1 for benzene.⁸³ If the same materials are burned with restricted air flow around the samples, the CO:CO₂ ratio can increase progressively toward unity in each case. At the same time, smoke production increases. However, no standard procedure for reducing air supply to the sample has yet been developed that gives results matching those from a realistic underventilated fire in a room.

TOXICITY DATA

This subject is reviewed in [Chapter 5](#) and will not be discussed here. One should note, however, that it is widely believed that the lethal condition is expressed more realistically as an integral of concentration and time of exposure than simply as concentration. Models can be modified to accept such inputs. Lethality data in concentration-time units are available on CO, as well as on the combined effects of CO and other pure gases, but no standard test method is available for obtaining such data on the fire products of a given composite substance.

IGNITABILITY

In many fire scenarios, the original ignition is a “given,” and the task of a model is to describe the history of the fire after ignition. For example, smoking materials are improperly discarded in a wastebasket, or there is a stove-top accident in the kitchen, or lightning strikes. In each case, a model can assume that a small localized fire appears at time zero and then calculate the development (if any) of the fire. Ignitability of a combustible material exposed to the existing fire can be crucial. This exposure is usually either by direct flame

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

gas impingement or by radiation. In some scenarios, whether ignition occurs can be crucial. For example, a burning cigarette abandoned on upholstered furniture might or might not ignite the furniture. Inasmuch as materials can be chemically modified to make them more resistant to ignition (e.g., by introducing halogens) while increasing the toxicity of the smoke produced when they do burn, a model can in principle provide quantitative information on the tradeoff involved.

Numerous methods can determine ignitability as a function of heat flux, time of exposure, and sample size and orientation, for both radiative and flame-impingement exposures. The small-scale rate-of-heat-release methods previously mentioned^{25 218} have also been used to measure ignitability in cases of radiative exposure. Ignitability of flammable fabric is measured by a standard test involving a 3-s exposure to a small flame.⁹⁸ The Setchkin furnace⁹⁸ is standardized as ASTM Test D-1929, which measures the furnace temperature at which a small sample will just ignite in an airstream. Either spontaneous ignition or “piloted” ignition from a small pilot flame can be studied.

Test methods have been standardized for specific situations, for example, to determine the resistance of upholstered furniture to ignition by cigarettes.¹³³ Although great masses of data exist on ignitability of various materials by various methods, there is no central source of this information. Furthermore, because material thickness and orientation affect ignitability and an enormous variety of materials and combinations of materials are in use, a modeler cannot expect always to be able to consult a reference source for the needed properties. Rather, specific ignition tests might have to be made, if ignitability must be known.

SUMMARY

A number of available two-layer models represent at least crudely all the physical processes that occur in a fire in a structure. They require such data as burning rate, ventilation, thermophysical properties of ceilings and chairs, and critical concentrations of fire products that will prevent escape or be lethal. TAE can be calculated, and the effect on TAE of LC₅₀, burning rates,

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

compartment sizes, ventilation opening sizes, etc., can be readily explored. The calculated TAE cannot be expected to predict results in an actual fire accurately, for the various reasons cited above, but the relative influences of the various parameters should be more or less correct.

None of the available two-layer models can treat the filling of a long corridor with combustion products realistically. Research is being done on this problem. Clearly, the time required to reach a given condition at a given location remote from the fire varies inversely with the burning rate, but, if the flow is buoyancydriven, this dependence can be expected to be nonlinear. Heskestad⁹⁶ has suggested that the time required to reach a given condition varied inversely with the cube root of the burning rate.

No model has attempted to combine the buoyant flow of fire gases in a tall building with all the other factors known to influence air circulation--forced ventilation, the effect of external wind, and the chimney effect that occurs in winter because the air inside the building is warmer and less dense than the outside air.

The breaking of windows by heat from a fire has major effects on burning and on hot-gas movement. Models would be able to treat this if the time of window-breaking were known, but no one knows how to handle this.

Jones¹¹¹ has made a detailed comparison of most of the two-layer models mentioned above, and Friedman⁸⁴ has discussed the components of these models and their interactions, with emphasis on feedback loops and on thermal inertia that causes delays.

Computer models now available can, for some cases, calculate the development of a fire within an enclosure, as well as the buildup of smoke at a selected location in the fire environment. If the toxicity of the smoke is known, the fate of an occupant at this location can be predicted. More specifically, TAE can be compared with TNE for a selected scenario. Toxicity data, as expressed by the LC₅₀, are relevant only to TAE. Theoretically, sublethal effects of toxic fire products can affect TNE (e.g., the possible deleterious effect of CO on judgment or ambulation in hindering escape). The calculated

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

answers depend on material properties in addition to LC_{50} , especially burning rate and its variation with time, and in some cases ignitability. The physical arrangement and ventilation conditions of the enclosure and the means by which the fire is detected are also crucial.

The accuracy of the model predictions is limited by the accuracy of the input data. Even if the inputs are all perfectly correct, the models treat the fluid motions and mixing, as well as the energy feedback from the environment to the fire, by a series of approximations, so the model outputs will still be only approximate. However, comparisons with realistic fire tests have shown order-of-magnitude agreement with model predictions in a number of cases. Furthermore, the relative influence of the various parameters should be generally correct.

More research is needed for further refinement of models. And improvement is needed in methods of predicting burning rate from small-scale tests. For some scenarios, data on sublethal effects (not generally available) would be more relevant than lethality data.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

4

HAZARDS ASSOCIATED WITH FIRES

Fires generate three main sources of hazard--heat, smoke, and depletion of oxygen--all of which can interact in exerting their effects. The relative contribution of each to the overall hazard depends on the physical characteristics of the fire, namely, heat release rate, fuel source, and oxygen supply. These characteristics combine with others, such as structural configuration and distance from the heat source, to constitute the hazard at any moment. In a real fire, many of these characteristics are changing continuously. This chapter reviews briefly some of the information that is available on the potential hazards associated with the components of fires.

HEAT

The most obvious hazard associated with fires is heat. Although most fire deaths are due to smoke inhalation, many are caused by burns from the heat of the flame itself.^{63 64 203} A skin temperature of about 45°C is associated with pain.²¹⁴

Burn injury caused by the inhalation of air heated to 150°C or higher is ordinarily confined to the oropharynx and upper airway (above the vocal cords).^{44 154} Even very hot air is rapidly cooled before it reaches the lower respiratory tract, because of the tremendous heat-exchanging efficiency of the oropharynx and mesopharynx. The inhalation of hot steam, however, can cause a burn as deep as the major bronchioles.⁴⁴

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

OXYGEN DEPLETION

A decrease in arterial pO_2^* stimulates the peripheral chemoreceptors in the aortic and carotid bodies and causes hyperventilation.⁹⁰ The brain suffers irreversible damage if the oxygen (O_2) supply is interrupted for more than about 3 min.⁹⁰

The extent to which O_2 depletion and the resulting hypoxia are important in fires depends on various physical characteristics of the fire and its environment e.g., the size of the fire and the available air supply. O_2 depletion is not generally thought to be a major problem. However, when flashover occurs (see Chapter 2), O_2 can be depleted over a large area, even if the fire is contained in one room. A 3-MW fire in an average-sized house will consume all the O_2 in the house within about 30 s.

SMOKE

Smoke is defined here to include all the airborne products of the pyrolysis and combustion of materials. Smoke consists of particles (soot), gases (e.g., carbon monoxide), volatilized organic molecules of varied complexity, aerosols, and free radicals. The extent to which these components contribute to the overall hazard associated with smoke is discussed briefly below. Recent reviews contain more detailed and comprehensive treatments of the subject.¹¹³

WATER

Water is a frequent product of combustion, although the amount varies greatly. Unlike steam, water is not an important factor in smoke-inhalation injury, except that

*“p” denotes partial pressure (also called tension) of any gas in a mixture. It is the pressure that that gas would exert if it alone were present. The partial pressure of any gas is the product of the total pressure of the gas mixture and the fractional concentration of that gas.

water droplets can serve as a vehicle for the transport of absorbed acids, such as hydrochloric acid.^{39 54 93 97 168}

PARTICLES (SOOT AND AEROSOLS)

Soot and aerosols are the visible components of smoke. A wide range of particle sizes, from 0.1 μm to above 10 μm (mass median diameter), can be found in fire smokes. Those larger than 10 μm in diameter are too large to reach the alveoli, so their role in causing parenchymal lung injury is debatable.^{33 34} Particles can contribute to hazard by reducing visibility and otherwise impeding escape. The extent to which the decrease in visibility caused by particles and soot is a hazard in real fires is, like incapacitation, strongly suspected from anecdotal evidence, but its exact role is largely undetermined. However, it is clear a priori that any impediment to escape will increase the hazard associated with a fire. If the particles are also highly irritating to the eyes and cause lacrimation, vision will be impaired, even if the density of the particles is not great. But the presence of particles can also speed fire detection and thus aid early escape by serving as a visible warning or by triggering smoke detectors.

Some investigators believe that such hydrophilic pyrolysates as hydrogen chloride (HCl) can adhere to smoke particles and thus be transported into the tracheorespiratory tree, where they can directly damage membranes and cause edema.⁵⁸ However, estimates from laboratory modeling suggest that less than 2% of the predicted amount of HCl produced is adsorbed on soot.¹²⁷ How much of this reaches the lower airways is uncertain.

GASES

Carbon Dioxide

Carbon dioxide (CO_2) is a major combustion product. Its concentration in air can reach 15% in some fires.²⁰⁴

The most important physiologic effect of CO_2 is to stimulate the respiratory center. The normal pulmonary ventilation rate is 5-7 L/min, at a pCO_2 of 35-45 mm Hg.

An increase of 2 mm Hg in blood pCO₂ doubles the ventilation rate,⁹⁰ which increases the rate of exposure of the lungs to smoke. CO₂ also contributes to an abnormal acid-base balance when inhaled at the concentrations and for the durations common in fires.

In addition to its effects on the respiratory center, CO₂ at sufficient concentrations can cause headache, somnolence, mental confusion, hyporeflexia, lassitude, and eventually more severe neurologic disturbances, such as tremors, flaccid paralysis, unconsciousness, and eventually, death.¹⁰⁶

Carbon Monoxide

Carbon monoxide (CO) is an odorless and colorless gas. It is the major product of combustion that has been clearly established as contributing to death in fires.¹⁹¹ CO is toxic because of its high affinity for hemoglobin.⁸¹ It forms carboxyhemoglobin (COHb) by binding to hemoglobin, for which its affinity is 250 times greater than the affinity of O₂ for hemoglobin, and thus reduces the O₂-carrying capacity of the blood and causes hypoxia. The formation of COHb also increases the affinity of O₂ for the remaining hemoglobin. That shifts the oxyhemoglobin dissociation curve to the left; as a result, tissue O₂ tensions must fall to lower than normal for the O₂ to be released from hemoglobin. This effect causes greater hypoxia than would be expected only from the COHb-related decrease in the O₂-carrying capacity of the blood.⁸¹ The concentration of COHb achieved in blood depends on both the concentration of CO in inhaled air and the duration of exposure.⁶⁸ CO also binds to myoglobin in muscles.

COHb concentrations as low as 5% in the blood have been associated with angina and dysrhythmias in persons with ischemic heart disease.^{16 23} These effects might explain some fire fatalities, such as sudden death in susceptible persons at what would normally be considered sublethal concentrations of CO. A person can manifest psychomotor and judgment inefficiencies at a COHb concentration of about 10%. At about 10-20%, exertional dyspnea is present. Headaches are common at 20-30%, and nausea, dizziness, and muscular weakness can occur at 30-40%. At 40-50%, there is syncope, and at 50-60%, convulsions.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Concentrations of 60-70% lead to coma and, with long exposure, death. COHb at 80% is rapidly fatal.²²⁷

The central nervous system and myocardium are most sensitive to the O₂ deprivation caused by CO poisoning.²¹²

Because they cause hypoxia, sublethal exposures to CO can impair performance and thus impede escape from a fire. In some animal studies (mostly with rats), performance changes in conditioned behaviors were observed at CO concentrations of 200-400 ppm, corresponding to COHb of 13-15%.¹²⁴ In other studies, higher CO concentrations (600-800 ppm) were required to disrupt behavior during 60-min exposures.²¹ The effect of these exposures tended to be increased pausing in whatever behavior was taking place, not necessarily with any increase in errors; at higher CO concentrations, all behavior would cease. Using the more stressful task of avoidance of un signaled shock, Sette and Annau²⁰⁰ reported that behavioral disruption in rats occurred at 60% COHb (CO at 1,000 ppm) during a 30-min exposure. More recently, monkeys trained in a lever-pressing task that required crossing a cage to obtain positive reinforcement after a correct response suffered performance decrements with CO at 900 ppm in about 20 min.¹⁸⁵ After 30 min, COHb ranged from 25 to 30%; performance disruption was complete in some monkeys (total collapse), and the others completed only about 50% of the trials.

These data suggest that a wide range of CO concentrations can disrupt behavior in rodents and that the monkey responds similarly, at least at high concentrations. With human subjects in a simulated task of driving an automobile, the task became impossible at 45% COHb, and the subjects were near collapse.⁸⁰ Laties and Merigan¹²⁴ concluded that, although there was a lack of well-controlled human studies with clear-cut effects, the COHb threshold for detectable, if not necessarily reliable, changes in human performance was around 10%.

A partial explanation for the wide disparity in the behaviorally disruptive CO threshold is that organisms respond to hypoxic challenge by increasing cerebral blood flow²⁰² and that this compensatory mechanism can sustain function only up to a point, after which a precipitous decline might occur.¹⁸⁵

Because CO-induced hypoxia decreases the total amount of O₂ delivered to the brain, all regions of the brain might be expected to be equally affected. But neuropathologic examination of human fatalities and the results of animal studies suggest that some brain regions are more vulnerable than others to CO-induced hypoxic insult. Vogel²²³ described a case in which a man survived a fire and lived on a respirator for 5 months before dying of pneumonia. Neuropathologic examination of his brain showed marked destruction of several cortical layers and damage to the hippocampus, basal ganglia, and cerebellum. In a more extensive examination of human brain injury after various hypoxic insults, Ginsberg⁸⁷ concluded that lesions of the white matter were prominent. Monkeys exposed to severe CO intoxication exhibited essentially the same neuropathologic pattern. Ginsberg⁸⁷ also described a delayed onset of symptoms sometimes seen in human cases of severe CO intoxication. Such patients recovered from the acute intoxication rapidly and were discharged from the hospital, only to undergo progressive deterioration that began 2-6 weeks later. This deterioration was characterized by disorientation, confusion, excitement, restlessness, defective motor control, and even frank psychosis. In some cases, a vegetative neurologic state eventually led to death.

Hydrogen Cyanide

Cyanide inactivates heavy-metal enzymes by forming stable complexes with them. Of these enzymes, cytochrome oxidase is the most sensitive to cyanide. Formation of the complexes compromises oxidative metabolism and phosphorylation and blocks electron transfer to molecular O₂. The peripheral tissue O₂ tensions increase, and the unloading gradient for oxyhemoglobin decreases.²⁰⁵

Although hydrogen cyanide (HCN) can be formed in many fires, its contribution to toxic hazard is uncertain. It can be produced at appreciable concentrations only if the fuel contains both carbon and nitrogen. Inhalation of HCN can be rapidly fatal. Toxic symptoms occur at blood cyanide concentrations greater than 0.2 mg/L, and 10 mg/L is lethal.²⁰⁵ Symptoms of cyanide poisoning include salivation, nausea without vomiting, anxiety, confusion, vertigo, giddiness, lower jaw stiffness, convulsion, paralysis, coma, cardiac arrhythmia, and transient

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

respiratory stimulation followed by respiratory failure.²⁰⁵

Moss et al.¹⁵⁵ compared the effects of CO and HCN--alone and together--in rats. Whereas rats exposed to CO were calm and remained quiet until they collapsed from anoxia, HCN-exposed rats displayed a brief period of violent escape behavior followed by unconsciousness and death. Rats exposed to CO and HCN together exhibited the typical response pattern seen after exposure to HCN alone. The authors reported that CO at 5,000 ppm was lethal in 30 min, as was HCN at 50 ppm. The combination of CO at only 2,000 ppm and HCN at 16 ppm was sufficient to kill animals in 30 min.

Purser et al.¹⁸⁷ studied the effects of HCN in monkeys sitting in chairs, with gas administered by mask. Incapacitation was defined as a semiconscious state with loss of motor tone. With HCN at about 150 ppm, incapacitation was seen after 8 min. At 100 ppm, the lowest concentration tested, the monkeys became incapacitated in 19 min.

Ginsberg⁸⁷ reviewed the neuropathologic consequences of cyanide intoxication. The lesions produced in the brain closely resemble those seen after CO exposure. In acute, high-dose cyanide intoxication, the victim goes into respiratory arrest. Although the mechanism of cyanide toxicity is completely different from that of CO toxicity, the resulting neuropathology resembles that caused by other hypoxia-inducing agents.

IRRITANTS

Many respiratory irritants are generated in fires, including ammonia, oxides of nitrogen, hydrogen chloride, sulfur dioxide, isocyanates, and acrolein.^{120 217} The site of injury after exposure to these substances is determined largely by their solubility. Highly water-soluble gases, if they are also highly reactive with surface components (e.g., ammonia) are readily absorbed and cause injury to proximal mucosal surfaces and the upper respiratory tract. Deposition of insoluble gases (e.g., some oxides of nitrogen) in the lower respiratory tract causes injury that might be delayed by 24-48 h. Other factors that affect the site--and extent--of injury include chemical form, dose, and duration of exposure.

Survivors of acute injury have a highly variable prognosis. Some recover fully within weeks with no permanent sequelae, whereas others have a spontaneous, usually mild, recurrence of pneumonitis several weeks after the exposure.²⁷ Although most survivors gradually recover, others are left with productive cough and residual obstructive deficits with or without bronchial hyperreactivity. Restriction impairment sometimes remains. A serious but rare complication is bronchiolitis obliterans, in which spontaneous deterioration begins about 4–6 weeks after injury and pulmonary function shows a restrictive or mixed obstructive-restrictive process. Respiratory failure and death can ensue; there are usually permanent residua among those who recover.⁸⁹

Hydrogen Chloride

Airborne HCl exists in the anhydrous state and as an aerosol. Because anhydrous HCl is very hygroscopic, exposure to it is potentially more dangerous to biologic systems than exposure to HCl aerosols. Anhydrous HCl injures not only by corrosion, as does the acid, but also by desiccation. However, its very affinity for water makes exposure to anhydrous HCl extremely unlikely.

The main nonlethal effects of HCl are irritation of the mucous membranes that results in breathing difficulty and lacrimation that obstructs vision, both of which can cause panic. Air concentrations of HCl below 100 ppm are considered tolerable, whereas concentrations near 1,000 ppm are rapidly fatal in rats.⁹²

Hydrogen Fluoride

The physiologic effects of hydrogen fluoride (HF) are the same as those of HCl.²¹³ However, HF is more potent than HCl. Acute inhalation of HF at 100 ppm can cause death in only a few minutes. Like HCl, HF can cause delayed death from cardiotoxicity and from such pulmonary sequelae as infection.²¹³

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Sulfur Dioxide

On contact with moisture, sulfur dioxide (SO₂) forms sulfurous acid. Death resulting from SO₂ exposure is usually caused by respiratory arrest and asphyxia, which culminates in massive tracheobronchial mucosal necrosis and gross pulmonary edema, with no evidence of an inflammatory reaction.⁴⁶ Delayed irreversible reactions include chronic airflow obstruction and bronchitis. Associated symptoms (dyspnea at rest and on exertion) and disability can be severe. Other common symptoms include cough, wheeze, rales, hypoxemia, marked abnormality in pulmonary function, bronchiolitis obliterans, peribronchiolar fibrosis, and a general decrease in small-airway diameters. There is one report of severe left main stem bronchial stenosis.⁴⁶ Reduced resistance or increased susceptibility to infection can ensue days or months after what is at first considered to be a mild exposure to SO₂.⁸⁸ Delayed deaths from pulmonary infection (occurring 17 days to 16 months after exposure) have also been reported.

Nitrogen Dioxide

The thermal oxidation products of nitrogen are usually found only in association with extreme combustion temperatures (about 2500°F or 1370°C).⁶¹ The immediate effect of an intense exposure to nitrogen dioxide (NO₂) is rapid death from respiratory spasm or pulmonary edema. Exposures to high, sublethal concentrations can cause severe delayed pulmonary edema and chemical pneumonitis. There is some evidence, mainly from animal experiments, that a single brief exposure can cause persistent lung damage, such as emphysema and interstitial fibrosis. Severe discomfort with lacrimation, coughing, and respiratory distress is induced by somewhat less intense exposures than those requiring hospitalization, and a milder pulmonary edema with reversible respiratory impairment is possible.¹⁴⁵ Single exposures to NO₂ at concentrations that cause slight, but tolerable, discomfort in humans have been shown in animal experiments to cause a reversible increase in susceptibility to respiratory infection and aggravated reactions to allergens. Even lower concentrations, at or below the threshold of sensory perception and below the current

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

federal occupational standard (5 ppm), can cause reversible impairment of respiratory functions.¹⁶⁶

Hydrogen Sulfide

Hydrogen sulfide (H_2S) is characterized by the smell of rotten eggs. It is an irritant gas that induces inflammation of the moist membranes of the eye and respiratory tract.¹¹ The consequences of exposure have been grouped into three phases: acute, subacute, and chronic. At high acute doses (over 1,000 ppm), H_2S causes immediate collapse with respiratory failure. Artificial respiration is required to restore the victims, who might have neurologic symptoms later. At lower doses (300-500 ppm), subacute exposures cause severe eye and respiratory tract irritation. After a few hours, pulmonary edema might set in. Exposure at 50-100 ppm is characterized by nonspecific neurologic symptoms, such as fatigue.^{161 165} Recent animal studies have shown that subacute exposures are followed by reduction in protein synthesis, probably caused by inhibition of cytochrome oxidase.¹⁹⁷ The resulting cellular hypoxia is suggested to be the critical toxic effect of H_2S intoxication. The slow dissociation of the cytochrome- H_2S complex could explain the persistence of the biochemical effects and the cumulative effects of repeated exposure.

ALIPHATIC AND AROMATIC HYDROCARBONS

Most hydrocarbons have an anesthetic or narcotic effect when inhaled. The aromatic hydrocarbons, in addition to their narcotic effect, have varied irritant properties.¹³⁶ Some can be absorbed through the skin. Some, notably benzene, are carcinogens.¹⁰⁸ Although their presence is not uncommon in fires, their concentrations are usually very low and insignificant in proportion to those of other pyrolysates.¹³⁶

FREE RADICALS

Considerable attention has recently been focused on the presence of stable free radicals formed during combustion.^{48 137} Free radicals have been identified at concentrations up to 1,200 ppm in fire environments

where CO did not exceed 500 ppm.¹³⁷ Unidentified, but stable, free radicals were found in laboratory tests to cause rapid unconsciousness, owing to the peroxidation of the pulmonary surfactant, which caused an increase in surface tension, atelectasis, and concomitant hypoxia.¹³⁷ This mechanism also potentiated CO asphyxia and might be related directly to alveolar injury of type I pneumocytes and to the inhibition of alveolar macrophages and ensuing pulmonary sepsis (Lowry et al., unpublished data).

INTERACTIONS AMONG COMPONENTS

This brief review of the toxic hazards associated with fires suggests that fatalities due to smoke inhalation are in reality caused by complex mixtures of gases, particles, and other less well-characterized products of combustion. Results of the few well-conducted fire-fatality studies available^{18 35} indicate that most fire fatalities are associated with CO poisoning and, to a much smaller extent, with HCN intoxication. Although these two chemicals have been identified with some certainty, examination of the blood of victims has revealed that in many cases neither chemical was present at a concentration sufficient to cause death. Other chemicals, such as free radicals, have recently been identified as potentially lethal, but difficulties in detecting them in human victims leave their contribution to fire fatalities uncertain. The most serious potential hazard, of course, is the combination of a mixture of combustion products with high temperatures that can increase their toxicity.

There is evidence that exposure to a combination of two or more toxic agents can have effects not completely explained by knowledge of the effects of exposure to the individual agents alone. Similarly, although data are insufficient for drawing firm conclusions, the combination of numerous respiratory irritants can be expected to induce toxic pulmonary effects not anticipated on the basis of the effects induced by exposure to any single toxicant at lower concentrations. Understanding of these interactions will increase what is now our very limited ability to extrapolate from some fire model systems to actual human experience of fire exposures.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

HEALTH EFFECTS OF SMOKE INHALATION ON HUMANS EXPOSED TO FIRES

The health effects of smoke inhalation on humans can be grouped into three phases: immediate or in-fire effects, early postexposure effects, and long-term sequelae.

IMMEDIATE EFFECTS

Immediate effects are defined as those which occur at the fire scene. They include the effects of exposure to heat, CO, increased concentration of CO₂, O₂ depletion, and irritants. Causes of death at the fire scene include heat, burns, and neurologic and cardiorespiratory collapse.^{43 79 132 153 154 180 230 231 232}

EARLY POSTEXPOSURE EFFECTS

Early postexposure effects are defined as those which are seen after rescue or entry into the emergency care system and in the resuscitation and shock phase--a period of up to several days. Myocardial infarction can be precipitated by the physical and psychologic stresses of the fire. Common sequelae are edema of the upper airways and edema and bronchospasm throughout the respiratory tract. In this period, sepsis might complicate recovery and contribute to a fatal outcome.

A moderate smoke-inhalation injury can produce chemical tracheobronchitis concentrated in the large and medium airways.^{10 77 86 153 183} Bronchorrhea is common after smoke inhalation and is often accompanied by large amounts of sooty sputum. Sloughing of the bronchial mucosa can also occur. Depending on whether sepsis occurs, the condition usually improves by the tenth day.

LONG-TERM SEQUELAE

The difficulties of identifying acute effects of single high-dose exposures to fire are compounded when one tries to define the chronic effects of such exposures. Few followup studies to characterize the nature and extent of chronic sequelae have been performed;

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

interpretation of those which have been done is limited by problems related to selected populations (selected with respect to types of exposures and extent of illness at presentation for medical care), variability of followup intervals and measured outcomes, and loss of subjects to followup. Much of our knowledge about potential and known long-term sequelae of exposures to fire has been derived from animal and human data on exposures to constituents of fire smoke in nonfire settings, such as intentional exposure to CO in automotive exhaust. It must be kept in mind that interactive effects of the numerous constituents of smoke probably increase the risk of injury.

Long-Term Sequelae after Single Exposures

Fire victims have been shown to have chronic obstructive pulmonary disease after single exposures.^{53 119} Followup studies among smoke-inhalation victims have demonstrated various types and degrees of persistent pulmonary dysfunction. Whitener et al. performed serial pulmonary function measurements in 28 patients after acute burn injury and smoke inhalation.²²⁶ Among the six with smoke inhalation only (without burns), significant pulmonary obstruction was observed within hours of exposure, and further recovery of function was seen at final followup, 5 months after injury; that all six were smokers might preclude the generalization of these persistent effects to all smoke victims. Among patients with both smoke inhalation and surface burns, the pulmonary function abnormalities were more severe than in those with either alone, and decrements of function were still resolving at the 5-month followup. Recent animal studies of pulmonary effects of wood smoke and thermal decomposition products of plasticized poly(vinyl chloride) have shown that smoke from Douglas fir diminished ventilatory response to 10% CO₂.²²⁹ Wood smoke was one-tenth as potent as smoke from poly(vinyl chloride), and animals recovered from the effects of wood smoke much more rapidly.²²⁸

Other delayed sequelae of inhalation are attributed not only to thermal injury (e.g., tracheal stenosis), but to toxic exposures of the tracheobronchial tree (tracheobronchitis, bronchiolitis obliterans, bronchial stenosis, and bronchiectasis).^{1 30 176}

Long-Term Sequelae after Repeated Exposures

Although long-term pulmonary impairment is the most commonly recognized and studied sequela of acute exposure, risk factors for chronic respiratory dysfunction, the prevalence of dysfunction among fire victims, and the degree of permanent impairment are still poorly characterized. Firefighters have been a useful cohort for investigations of respiratory morbidity. Caution needs to be applied, however, in generalizing results from studies in this group, which faces recurrent exposures and different exposures in postfire overhauls, to the population of victims of single exposures.

Although a number of studies among firefighters have identified acute pulmonary complications of smoke inhalation, the reported chronic sequelae are variable. Tashkin and co-workers' initial evaluation of 21 fire-fighters exposed to the combustion products of poly(vinyl chloride) found transient hypoxemia in 19; at 1 month, respiratory symptoms and pulmonary function abnormalities did not exceed those in matched controls.²¹⁶ Musk et al.,¹⁵⁹ however, found significant decrements in forced expiratory volume at 1 s (FEV₁) among a group of 39 Boston firefighters after intense smoke inhalation.¹⁵⁹ Loke et al.¹³⁴ found an excess of changes consistent with small-airway disease among 54 firefighters and persistent significant obstructive airway disease in one after a single severe exposure. Unger et al.²²¹ studied a group of 30 firefighters after a severe smoke exposure and found decrements in both FEV₁ and forced vital capacity (FVC) with a preserved ratio of FEV₁ to FVC, which is consistent with a restrictive ventilatory defect. This defect persisted at 6-week and 18-month followups.²²¹ Because baseline spirometric data were not available, it was not possible to establish whether the decrement in function resulted from repeated exposures or from a single intense exposure; for various reasons, the authors favored the former as the cause.

Chronic pulmonary function changes attributed to repeated smoke exposure have been found in three studies of firefighters.^{175 178 208} The largest was a study of pulmonary function among Boston firefighters: 1,430 firefighters studied in 1970 and again in 1972 were found to have greater than twice the degree of pulmonary function loss that would have been anticipated in the

intervening 2 years; the decline was significantly correlated with the frequency of fire exposures.¹⁷⁸ However, 3- and 6-year followup studies of this cohort showed no further abnormal loss of pulmonary function.^{157 158} The authors attributed the apparent resolution of accelerated functional impairment to absence of the most affected subjects from followup and to job transfer (away from continued exposures) within the fire service. A recent 1-year followup study of a cohort of London firemen showed a greater than expected decline in FEV₁ and FVC, particularly among cigarette-smokers.⁶⁷

Firefighters have also been found to have both acute and chronic respiratory and neurologic dysfunction after serious exposures to toluene diisocyanate,^{22 128} combustion products of pesticides,¹⁹⁹ and poly(vinyl chloride) wire insulation.²²⁴

Cancer

Because the products of fire contain a number of known and suspected human carcinogens, concern has been raised about the potential carcinogenic risk associated with exposures to fire. Unlike the pulmonary sequelae of fire exposure, excess cancer risk would be expected to show a dose-response relationship: the greatest risk would be among those with repeated exposures--firefighters. After his review of the literature failed to reveal any studies that showed a pattern of excess cancer risk among fire-fighters,¹⁴¹ Mastromatteo investigated the mortality experience of a cohort of city firefighters and found no evidence of excess cancer risk.¹⁴² The absence of excess cancer risk was also demonstrated some 20 years later, when Musk et al. studied the mortality experience of Boston firefighters employed from 1915 to 1975.¹⁵⁶

The possibility that firefighters have excess cancer risks has been suggested by a number of other epidemiologic studies, with no consistent pattern of excess identified. These studies reported increases in brain cancer and lymphatic and hematopoietic cancers,¹⁴⁴ in lung cancer,^{70 179} and in gastrointestinal cancer.⁷⁶ More recent studies, by Feuer and Rosenman⁷⁶ and N. J. Heyer and L. Rosenstock (personal communication), have identified an excess of lymphatic and hematopoietic

cancers in a group of city firefighters with long exposure histories.

In sum, the data available are inconsistent and contradictory and show no convincing pattern of excess risk of cancer at specific sites. Some studies have shown an absence of excess cancer risk,^{142 159} and others have shown excesses of lung, gastrointestinal, hematopoietic, and brain cancers.^{70 144 179}

SUMMARY

The most studied and best recognized chronic sequelae of exposure to fire smoke are in the respiratory system. Smoke inhalation, with and without burn injury, has been demonstrated to cause persistent and sometimes irreversible impairment in pulmonary function. The impairment is predominantly obstructive, but isolated restrictive and mixed deficits have also been observed. Tracheal and bronchial stenosis, polyposis, bronchiolitis, and bronchiectasis have been identified. The prevalence and extent of these sequelae, however, are not well known. Although there is ample evidence that toxic gases are primarily responsible for these sequelae, differences in the effects of various combustion products are not well established.

Other chronic sequelae are rare; they include the delayed neurologic effects of exposure to CO and other asphyxiants. Excess cancer risk has been suggested in studies of firefighters who sustained chronic exposures, but, although plausible, remains unmeasurable among single-episode victims.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

5

LABORATORY METHODS FOR EVALUATION OF TOXIC POTENCY OF SMOKE

Assessment of fire hazard requires that toxicity data be available for incorporation into a hazard assessment model. Therefore, methods for evaluation of toxicity are presented in some detail in this chapter. The most common end point used to assess the toxicity of inhaled combustion products in animals is death, specifically the LC_{50} , but an equally important consideration in a real fire is the propensity of the smoke to impede or prevent escape before lethal conditions are reached. Toxic events are not measured or detected in LC_{50} studies; studies that take such events into account could be useful for determining the potential human health consequences of nonfatal exposures and provide a more convincing means of predicting ability to escape from fires. Although a goal of laboratory animal toxicologic studies of inhaled combustion products is to estimate the toxicity of these materials in man, extrapolations to man are usually only qualitative.

The first section of this chapter addresses the use of combustion-product toxicity tests for screening purposes. After contrasting chemical and biologic analyses, we discuss animal test methods that use death as an end point and then methods that use nonlethal end points, including factors that can impede escape from fires and nonlethal pulmonary effects that can be extrapolated to human exposures.

USE OF COMBUSTION-PRODUCT TOXICITY TESTS: TO SCREEN OR NOT TO SCREEN

A screening test should be simple, inexpensive, and valid. One assumes that the discriminations provided by

test data can ensure some desired degree of safety. E.g., in the case of regulation, the judgment might be a pass/ fail discrimination for approval of product application; in the case of manufacturing, the judgment might be to produce or not to produce an item. The Committee takes issue with the assumption that any regulatory pass/fail judgment can be made on the basis of toxicity screening test data alone. The more complex evaluation of fire properties (fire hazard analysis) remains, in our opinion, a requirement for judgments of suitability of products for specific uses. If products for the same intended use have been shown to be very similar in composition and other fire properties, a pass/fail decision that depends on a toxicity test could be justified. Although this appears to be a screening test, it is in fact simply the final point of discrimination in a less formal hazard analysis. For uses with no regulatory component (e.g., a manufacturer's surveillance of products under development), any chosen test can be used for screening, with specific performance criteria set by the user.

CHEMICAL ANALYSIS VS. BIOLOGIC ASSAY

Awareness of the possibility of unknown fire hazards came with the Cleveland Clinic fire in 1929, in which the newly developed nitrocellulose film played a major role. For the first recorded time, a major fire produced considerable quantities of toxic combustion products in addition to the ubiquitous oxides of carbon. The incident presaged a concern that would become acute with the avalanche of new synthetic materials in the 1950s and 1960s: how to assess the risks associated with combustion products of new materials.

Modern analytic chemistry soon made it clear that many pyrolysis products could be generated by materials of relatively simple composition. Boettner and Weiss⁴⁰ identified over 50 compounds produced by the pyrolysis of a sample of poly(vinyl chloride), and different species of wood produce different combustion products.¹⁶⁹ Such complexity showed that the toxicity of smoke could not be accurately predicted simply from chemical analysis of the original material.¹⁶² In an actual fire, many different materials are involved, some of which have combustion products not readily predicted by classical chemistry and perhaps not detectable by current techniques. Some of

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

these products could prove highly toxic, and their toxicity could go undetected unless biologic tests were used. One such product was reported in 1975 by Petajan and co-workers at the University of Utah.¹⁷⁷ Eventually identified as a bicyclic organic phosphate, it arose during the combustion of a urethane foam from the reaction of a phosphorus-containing fire retardant with trimethylolpropane, one of the components of the urethane formulation.

The product was first detected by its acute effects on laboratory animals, which included grand mal seizures; it also produced observable psychomotor effects on several of the human investigators. This incident is often cited as evidence of chemical tests' potential failure to detect unanticipated, and in this case unusually toxic, combustion products.

The preference for biologic approaches to screening for combustion-product toxicity does not mean that chemical approaches have been ignored. Recent advances in analytic techniques have led to readily available systems that can separate smoke components and identify many of them.⁶¹ A combination of gas chromatography and mass spectrometry, commonly called GC/MS, is a powerful tool for analyzing smoke components. Such gases as carbon monoxide (CO), carbon dioxide (CO₂), and oxygen (O₂) can also be detected by optical or magnetic methods. The analysis of smoke, however, is much more than a simple problem in gas analysis. Condensation, selective absorption, and stratification make reliable sampling difficult. Smoke is a mixture of gases, solid particles, and liquid droplets, and a careful analysis for even the most pedestrian set of known toxicants requires that all three phases of smoke be checked. This is not a trivial job and generally requires considerable treatment of smoke components before analysis.

Spurgeon²⁰⁹ has analyzed the pyrolysis products of 75 aircraft cabin interior materials for nine gases and attempted to correlate incapacitation times, as measured in the Federal Aviation Administration Civil Aeromedical Institute (FAA/CAMI) test method,⁶⁰ with the chemical profile of these gases. He reported that the observed incapacitation time in rats can be predicted as a linear function of the concentrations of selected gases in the combustion atmosphere. It remains to be demonstrated

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

whether this method has a more generally applicable predictive value.

Purser and Woolley,¹⁸⁸ in the United Kingdom, exposed monkeys to sublethal concentrations of individual gases--hydrogen cyanide (HCN) and CO-- and Purser and Grimshaw¹⁸⁶ exposed them to combustion products of wood, polypropylene, polyurethane, polyacrylonitrile, polystyrene, and nylon. They reported that the test subjects displayed signs of intoxication typical of only one or another of the major gases tested, depending on the conditions of the burning, and suggested that animal models can be replaced by chemical analysis.

However, chemical test methods pose substantial problems. For example, the sensitivity of a test is limited by the detection limits of the analytic instrument. Chemical tests often do not discriminate among the physical forms of a toxicant, such as its adsorption on particles and its occurrence as an aerosol, even though toxicity might depend on form. Correlations between human health response and combustion-product mixture dosage are poorly understood, so the health consequences of exposure to a fire gas at a given concentration over a given duration are poorly predicted. Finally, the human-effects data that have been cataloged are relevant almost exclusively to single chemical exposures, and little guidance is available for prediction of the effects of exposures to mixtures of chemicals.

Biologic test methods expose living systems to chemicals or mixtures of chemicals, to induce changes in the performance of those systems. The results can be useful even if the components of the test material are not identified, but the biologic changes chosen for observation must be measurable and preferably are easy to extrapolate and interpret.

The most commonly used end point in bioassays of smoke potency has been death, usually expressed as the concentration that causes death of 50% of the exposed animals (LC₅₀) in a specified period. With a bioassay for smoke potency, it is possible to describe a set of conditions under which the predetermined end point is known to occur. The bioassay, then, is the obverse of the chemical analysis: the effect can be defined even if the cause remains unknown.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

In summary, the advantages of chemical tests are that many are quick to perform and that they avoid the use of test animals. The main advantage of biologic tests is that they produce data of high validity. The major potential danger of a chemical test is that it could miss unanticipated, and perhaps unusually toxic, combustion products, whereas in principle there is less danger of missing a biologically relevant response in a biologic test. It is important to note, however, that the actual occurrences of toxic combustion products whose formation was not predicted by chemistry (e.g., by measurements of CO₂, CO, HCN, and O₂) are rare.¹⁷⁷ One other problem with the application of data derived solely from chemical tests to fire hazard models is that most current models are designed to accommodate toxicity data in units of concentration. The use of chemical data alone in a fire hazard model would require development and verification of a scheme to summarize and add the various measured concentrations in a useful way. Many of these concerns (e.g., over the use of analytic methods as an alternative to animal testing) have been addressed by other groups, such as the European Chemical Industry Ecology and Toxicology Centre⁷¹ and the International Standards Organization (B. Levin, personal communication).

A toxicity-testing strategy that avoids the uncertainties of chemical analysis while exploiting its advantages could follow these steps:

- Chemically analyze the test material's smoke for expected major toxicants, such as CO, HCN, and HCl.
- Calculate an "expected" LC₅₀ for the smoke, on the basis of the response of test animals to the toxicants identified in the chemical analysis.
- Perform a bioassay of the material's smoke at, slightly above, and slightly below the expected LC₅₀. If all the important toxicants have been identified in the chemical analysis, this test should be sufficient to confirm the identification and to yield an approximate LC₅₀. If the observed LC₅₀ is very different from the expected LC₅₀, this will also be apparent; in such a case, more extensive bioassays must be carried out.

TEST METHODS THAT USE DEATH AS AN END POINT

BIOASSAY OF SMOKE POTENCY

The chemistry of smoke and therefore presumably its toxic potency change with the conditions of burning. Fires typically evolve through a continuum of general conditions; the temperature, the ventilation, and the makeup of the fuel change over the duration of the process. These changes can modify the potency of smoke^{117 125} and make the development of smoke potency evaluations difficult.

The design of a test fire must include an abstract and simplified representation that will not only substitute for actual events, but also provide reproducible test conditions. The fire surrogates that are incorporated into test methods are most plausibly viewed as phases of a fire process, rather than as scaled-down replicas of specific fires. Exposure of a test sample at a fixed temperature has been compared with exposure to a fully developed fire, and exposure at an increasing temperature, to a growing fire. However, a test protocol that incorporates a single, arbitrarily chosen temperature is subject to question, because of the temperature dependence of smoke chemistry. Fire products generated at one temperature might be relatively innocuous, and products formed from the same material at another temperature might be significantly more potent.¹¹⁷

Generic toxicity testing is usually designed to reveal the worst-case response to a test agent, and the use of a single, arbitrarily chosen temperature for decomposition of samples is unlikely to achieve this condition. The fire surrogate for a test system is improved by choosing a series of fixed temperatures or programed temperature increases for decomposition of test samples.

Any exposure system used for evaluation of toxic potency must be considered representational, because actual human exposures vary widely and cannot be fully modeled in a test system. The major categories of exposure that have been incorporated in smoke toxicity tests can be described as static, in which smoke collects in a closed compartment, and dynamic, in which smoke streams from its source past the test subjects.^{4 107} Whether these differences are important is not known.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

LIMITATIONS IN USE OF BIOLOGIC DATA

The time at which an effect occurs, as measured in these tests, depends on sample size and heat transfer by the furnaces used. None of the tests under discussion provides data that can be directly extrapolated to predict a duration of safety or a time at which human death would occur in an actual fire.

If a single test chemical has been thoroughly studied and its mechanism of action in humans defined, a most appropriate animal species can be chosen for further evaluations. In studies of combustion-product potency during which mixtures of unknowns are administered in unknown quantities, the perfect surrogate for humans clearly does not exist. Instead, data must be collected from a well-characterized species that is typical of living systems.

The most commonly used organisms are rodents. In the absence of definite, specific biochemical parallels, the use of other animals offers no advantages. The use of an animal (a "typical" living organism) should not be expected to predict the actual dose at which some event would occur in humans, but rather to define relative potency of a series of materials. Because, in the bioassay of smoke, the actual interactions of unknown test materials with biologic pathways are uncertain, it is important to interpret the data with a margin of error for individual, as well as species, differences. A variety of numerical safety factors have been invoked by regulatory agencies for this purpose.

Integration of data from any toxicity test into a currently available numerical fire hazard model requires the expression of the test results as concentrations of smoke at which a specific end point is attained. Examples of end points might be change in attention span, lacrimation, loss of postural tonus, or, at the extreme, death. For the purposes of the numerical model, the specific biologic end point chosen is not critical. The choice depends on the desired degree of protection, which varies with the user. For some, the desire is simply to ensure escape by fire victims; for others, it might be to ensure continuation of peak performance in the presence of continued smoke exposure.

Although for some purposes it remains desirable to know as much as possible about the chemistry of the test material, that is not required in numerical fire hazard models. Present hazard analysis models call for a unit of smoke concentration. Smoke concentration is not generally measured, but the value can be numerically derived by dividing the mass of sample by the volume of space filled. The resulting value is represented in the tests discussed below as the LC₅₀. (The routine measurement of CO in smoke or of carboxyhemoglobin, COHb, in the blood of exposed animals, however useful such measures are for research purposes, provides no information of utility to hazard assessment efforts that is not provided with more certainty by the LC₅₀ itself.) Because of the requirement for data expressed in units of concentration, only three test methods are candidates for use with current numerical models of fire hazard: the National Bureau of Standards (NBS) method, the University of Pittsburgh (Pittsburgh) method, and the Deutsche Industrie-Norm. (DIN) 53 436 method. These are the most completely developed and documented of the available methods, have been published in peer-reviewed literature, and have been accepted or are under evaluation as consensus standards.

GERMAN DIN 53 436 METHOD

The DIN 53 436 method⁶⁵ was developed in Germany to provide a standardized procedure for the generation of combustion products, as well as for animal exposure.

The DIN method uses a dynamic airflow system. The furnace is a quartz tube 110 cm long fitted with a variable-temperature annular oven that moves along the tube at 1 cm/min. The furnace is designed to hold a cylindrical sample of equal volume or weight per unit length. Pyrolysis gases are swept out of the tube and into the exposure chamber by a 100-L/min airstream, with one further dilution possible between furnace and exposure chamber. The test animals are rats, and the duration of exposure (head-only or whole-body) is 30 min. LC₅₀ and the pyrolysis temperatures associated with specific lethal concentrations are determined. Death rates, necropsy results, and blood COHb values are the primary biologic data collected. Continuous chemical monitoring of CO, CO₂, and O₂ and periodic measurements of HCN, HCl, and other gases are performed.

In a series of papers, Klimisch, Kimmerle, and co-workers have described the procedure and results based on exposure of rats to a variety of materials.^{118 122 123} The data indicate that consistent and reproducible results can be obtained with this method within a limited range of temperatures (300-600°C). At temperatures above 600°C, when pyrolysis occurs, the variability of the results increases significantly. This method has been adopted as the German national standard, but has not been tested in the United States.

NATIONAL BUREAU OF STANDARDS METHOD

The NBS combustion system^{129 130} consists of a cup furnace in which heating elements surround a well that contains a 1-L quartz beaker, the sample holder. The exposure chamber is a 200-L airtight box connected directly to the furnace. All smoke is collected in the exposure chamber. Six rats are restrained in individual holders, with their heads extending into the exposure chamber.

In this static test, the cup furnace is heated to a temperature 25°C lower than the autoignition temperature of the sample (nonflaming or smoldering condition). A weighed sample is dropped into the cup for decomposition. The animals are exposed to the resulting fumes for 30 min, starting at the introduction of the sample, and observed for survival at 30 min and up to 14 days. The test is repeated with progressively larger samples and additional animals. For estimating the LC₅₀*, the

*Although data produced by all the combustion-product toxicity test methods are expressed as LC₅₀s, strictly speaking they are not LC₅₀s, but they can be considered surrogate LC₅₀s. LC₅₀ is defined as the concentration of a toxicant that causes death in 50% of the exposed animals in a specified period; concentration is defined as the relative content of a substance, e.g., milligrams per milliliter or milligrams per kilogram. The “concentration” as NBS uses it is grams of sample charged relative to the volume of the exposure chamber (200 L). The “concentration” as the Pittsburgh method uses it is grams of sample charged. In neither test is the real exposure concentration known. However, both measures are commonly used to represent LC₅₀.

exposure concentration is calculated by dividing the sample weight (grams charged) by the chamber volume (200 L). The entire series of tests is repeated with the cup preheated to a temperature 25°C above autoignition (flaming condition). For most uses, no correction is made for the portion of the sample that remains in the beaker on completion of the test, although modification of the system to collect these data would be possible. The resulting data are therefore nominal concentrations, rather than measured concentrations.

For summarizing smoke potency, the statistical method of Litchfield and Wilcoxon¹³¹ is used to estimate the LC₅₀. Data from the smoldering or flaming condition, whichever is more potent, are used for comparison with data on other materials, if a worst case is being described.

The nominal exposure concentration, in milligrams per liter, is assumed to remain constant for most of any exposure period, because sample decomposition is expected to be rapid.

Routine chemical monitoring of the exposure atmosphere includes evaluation of CO, CO₂, and O₂. The test atmosphere is sampled continuously during the test and returned to the chamber after filtering and analysis. Approximately 30% of the volume of the chamber is recirculated during the 30-min exposure; for some materials, this could be a source of important error.

End points other than or in addition to death have been studied as part of this test system. The available data suggest that the other end points selected were not more sensitive or more useful than lethality alone.¹²⁹ (Nonlethal end points are discussed more fully later in this chapter.)

UNIVERSITY OF PITTSBURGH METHOD

The combustion system of the Pittsburgh method^{5 6 19 20} is a box furnace that is heated at 20°C/min. The exposure chamber is a 2.2-L glass box with ports to allow monitoring of the test atmosphere and placement of mice for head-only exposure.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Smoke from the furnace is diluted with chilled air and drawn through the exposure chamber. The total airflow and the dilution air are measured. In each test, four male mice are exposed for 30 min. Animals are observed for 10 min after the test exposure. The test is repeated with larger samples and additional test animals, to estimate the LC_{50} by the method of Thompson and Weil.²⁰ In addition to the LC_{50} , the time required to kill 50% of the animals (LT_{50}) is recorded in this method.

Results from this test are most frequently expressed in terms of furnace loading weight. However, because sample weight is recorded continuously and air and smoke volumes are measured, it is equally convenient to express the results in terms of nominal concentration.²⁰ CO , CO_2 , and O_2 are monitored in air pulled from the exhaust line immediately after animal exposure; this air is not recycled.

COMPARISON OF TEST METHODS

Acute Toxicity

The LC_{50} protocols from the NBS and Pittsburgh tests are similar in several respects. The primary information is the number of animal deaths within a fixed period. For the NBS test, the time is 14 days after exposure. For the Pittsburgh test, the time is within the 30-min exposure period and 10-min recovery period; the Pittsburgh test has also been used with the longer animal observation period. In each case, the LC_{50} is calculated with a statistical method that provides confidence limits and the slope of the regression line. For both the NBS and Pittsburgh tests, the limited data available indicate acceptable reproducibility within and between laboratories.

Anatomic Changes

Necropsy data collected 24 h after exposure in the two tests have not been remarkably informative. With some important exceptions, reported gross necropsy findings have been limited to increased lung weights and corneal opacification.^{5 138 139} Anatomic changes are time-dependent and would not be expected to be visible at

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

the times selected for termination of either test. If observation periods were changed for specific research needs, both test protocols could yield more information pertaining to pathologic changes after animal exposure.

Test Subjects

The choice of test subject constitutes a problem for all toxicity tests. Similarities have been shown between rodent and human in sensitivity to toxic agents, such as CO¹¹⁴ and HCN.⁴ However, in rodents, which breathe through the nose, water-soluble materials might be retained in or absorbed through the nasal mucosa, thereby decreasing lung exposure. Rodents, for this reason, might be less acutely sensitive than other species to the effects of a water-soluble corrosive gas.¹⁹ As a result, products that release a water-soluble corrosive gas as a primary toxicant could be evaluated differently from some other materials.⁴ It is necessary to remain alert to this potential problem.

Physical Test Characteristics

The physical characteristics of these two tests need to be carefully compared and contrasted to identify differences in the fire and exposure models used and artifacts to be encountered. The impact of physical characteristics on smoke potency remains mostly unknown.

Differences in physical characteristics between the NBS and Pittsburgh tests might make these tests suitable for different uses. The validity and importance of this intuitive conclusion have not been carefully analyzed.

Furnace Dimensions

The Pittsburgh furnace is large enough (42 L) to accommodate a wide range of sample sizes, configurations, and orientations. The NBS sample holder is much smaller (1 L); as a result, some low-density foams cannot be evaluated in the NBS system.

For some products, it might be desirable to use sample holders that allow end products to be tested in an

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

orientation similar to that of the final use. It is more convenient to position a sample and a holder in the Pittsburgh apparatus than in the NBS system.

Heat Transfer and Oxygen Availability

The two test methods approach combustion in very different ways. In the Pittsburgh test, the sample sits on a stage in a relatively large oven; heating is primarily convective in the early stages, with radiative heat transfer to the sample becoming increasingly important as the oven's interior surfaces get hotter. In the NBS test, heat to the sample is conductive where the sample is in contact with the hot container; if the preset temperature is high enough, radiation from the surfaces of the container is also important.

If the sample is large, heating produces substantial thermal gradients within it, according to its physical form, thermal conductivity, and so on. When low-density materials are used in the NBS test, they can occupy a sizable fraction of the hot container, making local O₂ availability a problem. The Committee notes, however, that both these concerns diminish in importance as the size of the sample decreases; testing a product with an LC₅₀ of 5 mg/L with the NBS method requires only 1 g of sample. Even if the material is a low-density foam, 1 g of sample occupies 30-50 cm³, which constitutes only a few percent of the 1-L volume of the cup furnace. The extent, therefore, to which the details of heat and O₂ transfer control differences between the smoke toxicities measured by the two methods is expected to be less important for more toxic materials. To put it another way, the difference is most pronounced when it is least important.

Heating Regimen

A more important difference between the two tests is probably in the way in which energy is delivered to the sample. In the Pittsburgh test, the material is heated at a constant rate. Heating continues well beyond the ignition temperature; after ignition, therefore, the sample receives energy both from its own flame radiation and from the oven. Thermoplastic materials, once ignited,

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

usually burn relatively quickly, so the amount of such materials exposed to the high postignition heat load is comparatively small. In contrast, materials that contain fillers, which char or otherwise decompose more slowly after ignition, might lose most of their mass later. Douglas fir, for example, loses about 80% of its weight in this relatively high-energy environment.⁶

The NBS test provides no such high-energy period. The room-temperature sample is dropped into the cup furnace, which has been preheated to just below or just above the ignition point. Early heating is very fast in comparison with that in the Pittsburgh test; the sample surface goes from 25°C to 300-500°C rapidly, instead of at 20°C/min. However, it is never given energy at a rate appreciably greater than that required to ignite it. If ignition occurs, the sample is irradiated by its own flame, but there is no increasing energy flux from an exterior source.

Measurements of large-scale test fires indicate that heating rates immediately preceding ignition are likely to be very steep, perhaps tens of degrees per second, unless energy flux from an exterior source has been sufficient to preheat the sample surface well in advance of the flame's arrival. The external component of flux that a combustible sample receives is greatest near and after flashover. At earlier stages, the fire spreads as unignited combustible material is heated to its ignition point by an adjacent flame.

From the preceding analysis, it appears that the thermal conditions of the NBS test more closely resemble those of a young fire while it is still growing. In contrast, the Pittsburgh test's thermal conditions before sample ignition are more similar to those near and after flashover. As noted below, neither test provides enough O₂ to guarantee a well-ventilated fire.

Combustion Chamber Atmosphere

The atmosphere surrounding the sample in the furnace is important for the availability of O₂ to the combustion process. Because of the relative sizes of the cup furnace and the sample, the atmosphere in the NBS cup can be low in O₂ in some tests and higher in others. In

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

the Pittsburgh system, the atmosphere has a greater likelihood of being rather uniform, despite changes in sample size. However, according to the rule of thumb that a well-ventilated fire needs 5 or more times the air required for stoichiometric burning and the stoichiometric O₂ requirements for burning LC₅₀ samples of Douglas fir in the two systems, both systems are “under-ventilated”; consequently, fluctuations in chamber atmospheres might be relatively unimportant.

Exposure System

The static exposure chamber of the NBS test is representative of conditions in which smoke accumulates and mixes as the fire progresses, and it allows interactions among products that might be given off sequentially. The dynamic exposure system of the Pittsburgh test can be considered representative of human exposure to a moving stream of fire gas. The gases tend to be delivered to the animal as a series of concentration peaks, with low potential for interactions between fire products. This could have a toxic impact different from that of the mixed atmosphere of a static system, but the differences have not been studied.

Physical Artifacts

Artifacts inherent in both methods have been identified, but not quantified. In the Pittsburgh test, the surface-to-volume ratio of the delivery tube from furnace to exposure chamber is high; that allows deposition of potentially toxic products on the tube surface. Moreover, the introduction of cold air into the smoke stream, which cools and dilutes the furnace products, might alter the chemical composition of the smoke. Some products might condense or precipitate out of the stream, because of the sudden change in temperature.

In the NBS test, the gas monitoring procedure requires the removal and recycling of gas at 2 L/min for 30 min, for a total of one-third of the chamber volume. The air is recycled back into the chamber only after it passes through a series of filters and analytic equipment. The resulting change in test atmosphere has not been quantified. An additional possible artifact associated with

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

this system is the interaction of thermal decomposition products with the surfaces of the plastic exposure chamber. Because of the long residence time in the static chamber, it is possible for substantial quantities of reactive products to adhere to the inner surfaces of this chamber.⁴⁵

Comparison of Data from NBS and Pittsburgh Tests

Two kinds of comparison are of interest: broad classification and the actual measured values of toxic potency. The former is of interest if the tests are to have utility as screening devices; the latter is related to the estimate of overall hazard. In comparing test data, it is important to point out that lack of agreement of test values implies not that either test is in error, but only that they are different. Considering the substantial differences in the test characteristics, lack of agreement should not be surprising.

A published range of LC₅₀ values obtained with the Pittsburgh test for a wide variety of end products is 126 g (starting weight) for gypsum board, which is decomposed minimally by heat, to 2.7 g for a PVC pipe.¹⁹ The data were not corrected for residue weight. The same series of products tested with the NBS method resulted in a range of 45 to 1.8 g (225 to 9 mg/L). More and less potent materials have been studied in each test system.^{4 5 6 130 139}

Anderson and co-workers¹⁹ have compared LC₅₀ values obtained by the two methods on various materials and concluded that they agree fairly well for thermoplastic materials, but not for materials that can char or otherwise leave behind substantial residue on decomposition. This conclusion is consistent with the observations made above on the differing thermal environments of the two methods.

Actual values of toxic potency can be compared only if the data can somehow be normalized. Traditionally, this has been done by comparing the measured potency of a material with that of some reference material measured in the same test. The results of such a comparison, with Douglas fir as the reference, show relatively poor agreement; most materials are more toxic when evaluated

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

by the Pittsburgh method.⁵¹ However, the Committee believes that Douglas fir is a poor choice for a reference material, because its decomposition appears to be particularly susceptible to the differences noted between the two tests, and agreement of test data would be assessed differently if a less variable reference material were chosen.

Because time of exposure is so different between static and flow-through systems, comparisons might be more informative if time-weighting were used. Alexeeff and Packham⁸ have calculated $L(Ct)_{50}$ s from published data on the Pittsburgh test for cases in which information on sample weight loss was also available. In many cases, LC_{50} s obtained on the same materials by the NBS test are also available, but the needed corresponding data on weight loss generally are not. In their absence, a limiting assumption is that all the material loaded in the NBS test is rapidly converted to smoke, so the nominal LC_{50} would be the actual concentration encountered, and it would be experienced for the entire 30-min exposure period. The $L(Ct)_{50}$ for the NBS test would then be the product of 30 min and the measured LC_{50} . Values obtained by Alexeeff and Packham are compared with those obtained with the NBS test in [Table 5-1](#).

With two exceptions (cotton fabric and Douglas fir), the values obtained with the NBS test are systematically higher than those obtained with the Pittsburgh test. If the NBS test results were corrected for nonvolatile residues and for the time it takes for the samples (particularly the large ones) to decompose, the agreement would probably be much improved.

COMPARISON OF TEST METHODS WITH GUIDELINES FROM 1977 NATIONAL RESEARCH COUNCIL REPORT

The previous National Research Council Committee on Fire Toxicology¹⁶⁴ presented guidelines for combustionproduct toxicity testing, which provide another point of reference for comparing the NBS and Pittsburgh methods. The reader should note, however, that those guidelines were to address the development and improvement of methods for “first-level screening of materials.” The present Committee is addressing methods that provide data to be incorporated in hazard analysis, not screening, of

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

materials. The Committee believes that toxicity data alone--e.g., data from screening tests--are not sufficient for the complete and accurate assessment of fire hazard.

TABLE 5-1

Material ^a	L(Ct) ₅₀ ⁸	L(Ct) ₅₀ (NBS) ¹⁹
PTFE wire*	85 ^b	176 ^b
C-PVC pipe*	127	480
Thin wire*	165 ^b	240 ^b
PVC conduit	212	885
Intumescent paint	230	615
Paper wallcover	260	1,125
ABS pipe	280	855
Nylon-carpet foam backing	298	3,240
Vinyl wallcover	313	1,179
Mineral-base ceiling tile	384	3,390
Latex paint	403	5,280
Nylon-carpet jute backing	465	1,710
Asphalt felt	486	648
Wood-base tile	486	1,371
Cotton fabric	561	387
Reprocessed paperboard	883	1,500
Douglas fir	1,440	873
Gypsum wallboard	1,521	6,750

^aAll materials except those marked with an asterisk reported to leave substantial residue after burning.

^bBased only on weight of insulation.

The first guideline recommended the use of both pyrolysis and flaming decomposition conditions. Both are used in the Pittsburgh and NBS procedures. The discussion preceding this recommendation, however, assumed that changes in airflow would accompany different burning conditions. As currently used, both tests have kept ventilation constant at the fire site.

The second guideline recommended the use of specific test animals and exposure conditions: exposure of rodents for 15-30 min at temperatures not exceeding 35°C and with

About this PDF file: This new digital representation of the original work has been reproduced from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

O₂ at no less than 16%. Both tests comply with this guideline to some extent. Because of the dynamic conditions of the Pittsburgh test, which result in the exposure of animals to different combustion products at different times, it has been run for a 30-min interval, but the time of exposure to specific smoke components cannot be fixed. Because of the addition of air for dilution, O₂ is seldom low. In the NBS static system, a 30-min test has been selected. Concentrations initially rise and then are relatively stable for the remainder of the 30 min. O₂ in the animal exposure chamber is monitored, and O₂ is added if the concentration falls below the desired point. Each test provides for cooling smoke before animal exposure.

The third guideline recommended inclusion of a suitable measure of incapacitation, followed by 2 weeks of observation for behavioral and physical changes. The recommendation regarding incapacitation has not been successfully met by either test. Incapacitation tests, designed to detect and measure a dose-related effect on some behavior, were found to be incapable of detecting such an effect at a time after exposure much different from the time when death occurs; and the behavioral and physical monitoring selected did not prove satisfactory for screening purposes.¹²⁹ Although tests that have been developed were not successful in providing the required evaluations, the Committee considers that this remains a desired test end point. The 2-week observation period is incorporated in the NBS test; the Pittsburgh test calls for a 10-min postexposure recovery time.

The fourth guideline pertained to evaluation of the test atmosphere. In both tests, temperature, CO₂, CO, and O₂ are monitored routinely. Humidity and smoke density are not monitored. Other gases are measured when specific questions warrant it.

The previous committee recommended that data be compared with equivalent test results from reference materials, rather than being used as absolute values. Douglas fir has been used as a reference material in both procedures, but is not specified by either. NBS, which has done considerable study with Douglas fir, recommends that it not be used as a reference material.¹²⁹ The same conclusion has been drawn by others.¹⁹ No other product has been recommended as a reference material for these tests.

TEST METHODS THAT USE NONLETHAL END POINTS

FACTORS THAT IMPEDE ESCAPE

Circumstantial evidence suggests that many fire deaths are related to victims' failure to escape before lethal conditions are encountered (perhaps evidence of incapacitation) and that characteristics of the smoke might have been responsible. As a result, the earlier National Research Council Committee on Fire Toxicology¹⁶⁴ recommended that small-scale animal test protocols include a measure of the loss of ability to escape, termed "incapacitation." "Incapacitation," however, was not defined, and the methods later developed for measuring incapacitation, some of which are described in this chapter, have reflected the various investigators' interpretations.

The characteristics of smoke that might impede or prevent escape cover a wide range of effects, from relatively minor to severe, including:

- Blocking of visibility, which makes escape routes more difficult to find and use.
- "Burning" and tearing of eyes, burning of nasal passages, and respiratory irritation (coughing and choking), which, even if not serious, could slow escape by causing distraction, discomfort, and panic.
- Pharmacologic and toxicologic properties that impair sensorimotor function, alertness, and judgment.
- Psychologic responses that cause panic, which could result in "freezing" and inappropriate choices of actions.
- Physiologic effects on other organ systems, especially the respiratory system, that would render a person incapable of life-saving actions.

Of these characteristics, the two that have received the most experimental attention are impairment of sensorimotor performance and sensory irritation. Various methods for measuring these effects are described briefly below.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

OBSERVATIONAL METHODS

Observational methods have enjoyed considerable success in various screening programs. However, when applied to the evaluation of smoke toxicity, they have a potentially serious limitation: the presence of smoke imposes a restriction on the exposure-observation environment that could compromise other aspects of the test protocol, and the exposure-observation unit must be small enough so that smoke obscuration does not constitute a major problem.

Hilado and co-workers^{99 100 101 102 103 104} have been the major proponents of observational methods that use time to incapacitation as the observed end point. They define time to incapacitation (T_i) as the time to the first observation of loss of equilibrium (staggering), collapse, or convulsions. They also record time to death (T_d), the time to cessation of movement and respiration.

In the exposure protocol that they have used most extensively, a 1-g sample of material is heated in the absence of forced external air, and the temperature is increased from 200 to 800°C at 40°C/min. Data abstracted from several reports^{100 101 102 104} showed that the $T_d:T_i$ ratios for the 15 materials tested were all less than 2.0:1, except that for poly(vinyl chloride), which was 2.8:1. The correlation between T_i and T_d for these 15 materials was 0.89. Thus, the two end points apparently would provide similar information for ranking materials.

Motorized Activity Wheels

Several investigators have used motorized activity wheels to measure the capacity of rats or mice to perform a motor act during exposure to products of thermal degradation.^{60 85 91 146 193 210} Crane et al.⁶⁰ tested rats in motor-driven exercise wheels housed in an exposure chamber. The rats were exposed to smoke from a 0.75-g sample heated at 600°C for up to 10 min in a Lindberg tube furnace with recirculating flow. T_i was taken as the time when they could no longer walk and began to slide or tumble. T_d was recorded when respiratory and body movements could no longer be seen. The average difference between T_i and T_d for 71

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

materials was 7.3 min, and the average $T_d:T_i$ ratio was 2.0:1. For 15 of the materials, there were no deaths during the 30-min exposure period. An incapacitating combustion or pyrolysis product that is not lethal will generate what appears to be a very large difference between T_i and T_d ; however, if there is no T_d (no lethality), then differences and ratios are meaningless or indeterminate. Indeed, the time to effect for any end point cannot be considered adequate as a measure of smoke toxicity if only one concentration is evaluated.

Hind-Leg Flexion

In this procedure, developed by Packham and co-workers,^{74 75 170} a rat's hind leg is wired so that a shock is delivered if the foot touches a metal plate below it, and the task is to keep the foot raised. The rat is considered incapacitated when it can no longer avoid the shock. This test was adopted by NBS as its measure of incapacitation, thoroughly evaluated, and incorporated into an interlaboratory study to examine the reliability of the NBS test protocol.^{129 130} Comparisons of effective concentrations (EC_{50} s) for hind-leg flexion and 14-day LC_{50} s led to the conclusion that the hind-leg flexion response did not provide any great increase in sensitivity over the 14-day LC_{50} . Indeed, because of delayed deaths, the 14-day LC_{50} was more sensitive than the hind-leg flexion response for three of the 11 materials in the nonflaming mode and for one of the 12 materials in the flaming mode. Moreover, the materials were ordered in essentially the same way by both end points. The correlations were 0.88 and 0.95 for the nonflaming and flaming modes, respectively. As a result of these studies, NBS eliminated the hind-leg flexion response from its protocol.

Sensory Irritation and Physiologic Stress

The use of plethysmography to measure sensory irritation in laboratory animals was developed by Alarie and co-workers.^{2 3 5 28 112} A mouse is exposed to the products of thermal decomposition of a material in a chamber into which its head protrudes (described earlier as the Pittsburgh method). The rest of its body is sealed in a plethysmograph that continuously records

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

respiratory and other body movements before, during, and after the test period. Two quantitative measures are obtained from the plethysmographic records--sensory irritation and physiologic stress. For sensory irritation, the maximal decrease in respiratory rate in each test is recorded and plotted against the amount of material decomposed in that test. The amount of material decomposed that is associated with a 50% decrease in respiratory rate (i.e., the RD_{50}) is estimated from the concentration-response curve obtained from a series of such tests. Physiologic stress (stress index, SI) is obtained from the plethysmographic records minute by minute throughout the exposure and recovery periods. The SI reflects the severity of a series of physiologic adjustments that are made to compensate for the reduction in breathing rate and apneic periods.²⁸ For most materials, respiratory depression in mice is seen at much lower concentrations than is death.⁵ The correlation between the RD_{50} and the LC_{50} was only 0.14, indicating relative independence of the two end points.

Kane et al.¹¹² compared the RD_{50} s for 11 sensory irritants with effects reported in the literature for humans and animals. They proposed that the RD_{50} is equivalent to an intolerable degree of sensory irritation for humans and would probably cause incapacitation within 3-5 min.^{3 28 112} They also proposed that use of 10 times the RD_{50} would be lethal or cause severe injury to the respiratory tract. This prediction has been verified.⁴¹ Prediction of safe exposure of humans to 40 industrial chemicals from RD_{50} values found with mice was excellent (correlation, 0.92).⁷

Furthermore, Potts and Lederer¹⁸¹ used this model to examine the tolerability of an RD_{50} of red oak smoke in humans for 3 min. They used a strain of mice (HAICR) with different sensitivity from that of the Swiss Webster mice used by Alarie.⁴ At the RD_{50} concentration for HAICR mice, humans reported irritation, but this concentration was not intolerable and certainly not incapacitating. However, at RD_{75} , Potts and Lederer¹⁸¹ reported that the irritancy was high and that the person inhaling the smoke believed that it could not be tolerated for 3 min, but whether incapacitation would be caused by a 3-min exposure was conjectural.

OTHER METHODS

Several other methods have been used to investigate sublethal effects of products of thermal degradation. Available data are not sufficient to compare them adequately with tests that use death as an end point, nor are the data that most tests produce expressed in terms of concentration. Therefore, they are described only briefly here. Although these tests do describe behavioral or biochemical changes that result from exposure to combustion-products, it is not clear how they correlate with human health effects described in [Chapter 4](#).

Unsignaled-Shock Avoidance/Escape

Several investigators have used this procedure to monitor the behaviorally disruptive effects of thermal-decomposition products. The general procedure, known as Sidman avoidance,²⁰¹ involves training rats to press a lever to postpone a scheduled, but unsignaled, painful foot shock (i.e., avoidance) or to terminate the shock if it is received (escape).

McGuire and Annau¹⁴³ used the procedure to evaluate the effects of exposures to the smoke from a flexible polyurethane foam. They found significant increases in shocks received in 4- and 8-g exposures, both during exposure and during a 30-min recovery period. Trends in the same direction were seen in 2-g exposures, but the differences were not significant. The effects seen could be attributed to the amounts of CO generated. Sette and Annau²⁰⁰ used the method to investigate the interactive effects of pure CO (1,666 ppm) and heat. Heat alone and CO alone caused an increase in shocks received, and the combination appeared to be additive. Russo et al.¹⁹⁶ compared the effects of the smoke from polyimide and flexible polyurethane foams. The thermally less stable polyurethane foam caused greater performance decrements at the lower heating temperature at which greater amounts of CO were evolved. The reverse was true of the thermally more stable polyimide foam.

Water-Reinforced Task

McGuire and Annau¹⁴³ compared the effects of the smoke from a flexible polyurethane foam on licking

behavior. Water-deprived rats were trained and stabilized on a water-spout licking task. The authors found disruption of performance at 1.0-g exposures. Although exposure to the smoke from 0.1 g did not cause a significant decrease in the total number of responses during the 30-min exposure, a clear and progressive decrease began after 15 min and progressed for about 10 min. The authors suggested that the temporary decrease in licking behavior was caused by irritant components of the smoke to which the rats had adapted by the end of the exposure period.

Rotorod with Electrified Grill Floor

This procedure has elements in common with methods that use motorized exercise wheels and shock avoidance/escape. Hartung et al.⁹¹ trained rats to walk on a rod 3 in. (7.6 cm) in diameter rotating at 6 rpm. The incentive for remaining on the rod was the presence of an electrified grill floor under it. A rat was considered incapacitated during exposure when it fell off the rotorod and remained on the electrified grill floor for at least 2 min, after which the current was turned off in that compartment. Variability in T_1 was relatively low, and that permitted statistically significant differences to be found among materials with the use of only eight or 12 rats in each group. This method was also used by Mitchell et al.¹⁴⁶ in full-scale tests of the effects of combustion products of natural fiber and synthetic polymeric furnishings. Their results showed that the smoke from synthetic polymeric materials impaired performance faster than smoke from natural-fiber materials.

Multisensory Conditioned Pole-Climb Avoidance

In this procedure, rats were trained to escape foot shock by climbing or pulling a response pole suspended from the ceiling of the test chamber. They were then trained to avoid the aversive foot shock, which was preceded by one of three warning stimuli--a light, a tone, or a nonaversive electric current on the floor. All of a series of 10 flexible, flame-retarded polyurethane foams^{172 184} caused impairment of the avoidance response, but none was incapacitating (as defined by loss of the escape response). Nor were these materials, in

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

the exposure ranges tested, associated with acute mortality during the exposures and 30-min recovery phases. However, delayed deaths attributed to pulmonary or cardiovascular complications¹⁷¹ followed exposure to eight of the 10 materials. In tests with five potential aircraft interior materials,⁶⁶ almost all deaths occurred within the exposure-recovery test period. The correlation between the concentration of smoke that caused a 50% decrease in avoidance and LC₅₀ for this small series of materials was 0.91. Thus, each end point would predict the other, and they would provide about the same answers with respect to the relative toxicity of smoke.

Analysis of Use of Bronchoalveolar Lavage Fluid to Detect Acute Nonlethal Lung Toxicity

Many short-term tests have been developed to assess the potential toxicity of various materials that might be inhaled by humans. These include the use of isolated pulmonary alveolar macrophages,²²⁵ bacterial cultures,¹⁵ and cultured mammalian cells.⁴⁷ These tests have proved useful in rapid screening of a large number of materials, but the general approach of each test does not allow monitoring of the integrated response of the whole animal to the inhalable agent in question. One *in vivo* method, which is also relatively rapid, uses biochemical and cytologic evaluation of bronchoalveolar lavage (BAL) fluid from the lungs of exposed animals to detect lung damage. Many investigators have used this technique to evaluate the potential chronic lung toxicity of inhaled materials, on the basis of the hypothesis that animals exposed by inhalation to a pulmonary toxin will suffer subtle acute lung damage that can be measured by various biochemical and cytologic changes. A correlative hypothesis is that inhaled toxicants will cause specific types of damage to respiratory tract tissues and cells, altering various biologic and biochemical processes that can be measured with assays of BAL fluid. Sampling the bronchoalveolar region of the respiratory tract by saline washing (lavage) of lungs is relatively simple. The lavage is usually done on excised lungs in toxicity screening tests. However, it can be done *in vivo* if the experimental objectives of the study require repeated sampling of BAL fluid.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Changes in BAL fluid characteristics are postulated to be specific and indicative of later-developing pathologic alterations in respiratory tract tissues.⁹⁴ A number of studies have shown good correlation between these early toxicity indicators and later-developing lesions. The application of these methods to combustion-product toxicity testing programs might also prove useful. Not only do analyses of BAL fluid potentially provide information on the development of chronic lung disease, but these techniques might provide a highly sensitive method for ranking the acute toxicity of inhaled combustion products.

SUMMARY

Efforts have been made to use analytic chemistry to predict the toxic potency of smoke. However, chemical analysis is not now an acceptable substitute for bioassay of smoke toxicity. Biologic tests of smoke toxicity are most useful in defining relative toxicity of combustion products; direct extrapolation to humans is seldom appropriate.

For an accurate assessment of fire hazard, toxicity data alone are not sufficient, but should be incorporated into a fire hazard assessment. The two biologic methods for evaluating the lethal potency of smoke that have been compared here appear to provide data that can be incorporated into a numerical model for fire hazard evaluation. Neither test successfully addresses the possible sublethal effects of smoke exposure. Each test yields reproducible results. These bioassays represent different fire and exposure conditions, but the relationship between small-scale test conditions and real fire conditions is not well understood.

Analysis of toxicity data on nonlethal end points in animals exposed to products given off by different burning materials might provide data for the prediction of potential health effects of these products in humans. These analytic methods could also prove useful for application to overall hazard assessment and selection of materials of greater safety with respect to fire. However, at present none has been developed and validated to the extent necessary for incorporation into such an assessment.

6

GUIDELINES FOR HAZARD ASSESSMENT: CASE STUDIES

Setting a level of performance using hazard assessment requires four steps. The first two steps establish a quantitative measure of hazard, the time available for escape (TAE), and relate it to the controllable fire and smoke properties of the product under study. These steps are sufficient to allow products intended for a given application to be compared on the basis of hazard. Then if a regulation setting a required level of performance is contemplated, the last two steps are carried out.

1. Identification of the environment and conditions of use of the product being assessed (i.e., definition of the various scenarios in which the product will be used and is likely to be involved in a fire).
2. Determination for each important scenario, through numerical modeling or full-scale experiments, of how the fire and smoke properties of the product affect its fire hazard, on the basis of TAE as a measure of hazard.
3. Selection of the minimal acceptable TAE, ideally by comparison with the time needed for escape (TNE), for each important scenario.
4. Specification of the fire and smoke properties of the product that are needed to provide the minimal acceptable TAE or to increase TAE.

The general procedure for hazard assessment can take very different forms, depending on the kind of product under consideration. For example, an article of upholstered furniture in a room poses a kind of threat different from that of a combustible pipe in a chase

behind a wall. To illustrate the procedure, these two situations are examined in the following case studies.

CASE STUDY 1: BURNING OF AN UPHOLSTERED CHAIR

STEP 1: DEFINING SCENARIOS

Environment

The chair and the people exposed to the fire are assumed to be in a compartment of about 650 ft² (about 60 m²). A compartment of this size is typical of the combination of a small sitting room, hall, and bedroom. It is assumed that doors are open and that the interior walls provide no barrier to the smoke; thus, conditions throughout the compartment, once the fire has begun, are essentially the same. This assumption is the “worst case” for the occupants of the compartment as a whole. A closed door exacerbates the situation for anyone in the room with the fire, but gives rise to a lower degree of threat to someone in another room. The walls of the compartment are assumed to be 0.5-in.-thick gypsum board, a material whose thermal properties are well known. The detailed numerical data used in the room-fire calculation are shown in the first part of [Table 6-1](#).

The example here is typical, but a compartment of different dimensions or construction could just as easily have been chosen. In most cases, it will be hard to avoid some arbitrary choices in developing the scenario. The advantage of hazard assessment by computation is that the effects of truly arbitrary choices can be tested by computing TAE for a series of assumptions, and the results can be examined to determine which, if any, of those assumptions most influence the outcome.

Fuel and Ignition

In this scenario, the chair is the first item to ignite, it is the principal item of fuel, and it is assumed that the entire surface becomes involved quickly. It is clear that many other sequences of events can be envisioned, but in few will it be possible to relate the burning of the item of furniture quantitatively to the outcome of the fire. The weight of the combustible material is chosen as 25 kg (55 lb), which is typical of a large easy chair.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TABLE 6-1

Room:

Length, m	7.6
Width, m	7.6
Height, m	2.4

Wall:

Thickness, cm	1.6
Density, kg/m ³	960
Conductivity, kW/m ² per kelvin	1.7 x 10 ⁻⁴
Specific heat, kJ/kg per kelvin	1.1

Fuel:	Chair		
	1	2	3
Heat release rate, bench scale, kW/m ²	100	200	300
Mass, kg	25	25	25
Burning time, s	600	300	200
Heat of combustion, MJ/kg	18.1	18.1	18.1

Upholstered furniture is often ignited, not by flaming ignition, but by a dropped cigarette. Such ignitions usually lead to smoldering, a qualitatively different kind of combustion. A smoldering item might burn in that mode until it is completely consumed or might, after a time, make a transition to flaming combustion. Both smoldering and flaming combustion are considered here.

Conditions of Exposure

In addition to those in direct contact with the compartment fire, “bystanders” can be affected, and it is of interest to know how. Such bystanders could be in adjacent compartments and could be partially (but not completely) protected from smoke leakage by fire-resistant construction.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Another requirement for this exercise is to determine when conditions would become untenable for those exposed, i.e., when escape would no longer be possible:

- The hot smoky layer has descended to within approximately 1 m (3.3 ft) above the floor. At this time, the occupants are likely to breathe the airborne fire products and endure the temperature of the layer.
- The hot layer has reached a temperature high enough to cause physical injury immediately. Observing the work of other investigators,⁸² the Committee has chosen 183°C (at which skin burns) as this temperature.
- The occupants are exposed to an incapacitating or lethal amount of combustion products. Determination of when those exposed would have been exposed to a lethal amount of combustion products is straightforward in principle; it assumes that laboratory-measured lethal doses approximate those encountered in real fires. But deciding on what is incapacitating is more difficult. (The lack of a good test for incapacitation is discussed in [Chapter 5](#).) One approach to approximating incapacitating dose would be simply to use, say, 20% of the lethal dose of combustion products as the “incapacitating dose.”

STEP 2: COMPUTING TAE AS A FUNCTION OF FIRE AND SMOKE PROPERTIES

Fire Model

The model selected for this computation was the Fifth Harvard Computer Fire Code (5.2).¹⁴⁸ This model was designed for a fire in a room that does not involve the walls or ceiling, and it is the only available model that couples the time-variant thermal conditions in the room to the intensity of the fire. In this case, the burning rate of the upholstered furniture was supplied, on the basis of laboratory measurements of the heat release rate of the furniture cushioning. Of particular concern are the accumulation of hot gaseous combustion products in the upper region of the room, the increase in thickness of this layer as the fire grows, and the temperature of the layer. Because the burning rate of the fuel is an input to the model, the concentration of smoke in the

upper region in this particular case can be calculated if the volume of the upper layer is known. Computations were carried out on a minicomputer with the values listed in [Table 6-1](#) as input.

Burning of Upholstered Furniture

The objective of the study is to relate fire conditions in the room to the fire performance of the fuel, so it is necessary to direct attention to how the heat release of the upholstered furniture is related to its burning in the room. It has been demonstrated²⁴ that the peak heat release rate of a piece of upholstered furniture is proportional to its total mass and to the heat release per unit area, as measured in the laboratory. This proportionality holds for a series of furniture items that are similar in style and shape and have a high heat release rate. However, if the cushioning and fabric used are extremely difficult to ignite, such as neoprene foam covered by woolen fabric, the fire is not vigorous enough to fit this proportionality. Three chairs are used in the simulation. All have the same weight, 25 kg, and are chosen to represent a threefold range in heat release rate. The three chairs have the same total mass and the same average heat of combustion, so an increased rate of heat release has to be accompanied by a correspondingly shorter burn time: the chair with the lowest heat release rate burns for 600 s, and that with the highest heat release rate burns for 200 s.

Use of the Harvard code for this scenario requires some compromises with reality. For example, burning of the chair requires that combustion air be admitted to the room from outside; hence, the model provides for a vent. However, vents also allow smoke to leave the room; this is undesirable when one is calculating a worst-case scenario in which virtually all the smoke is contained in the room. The vent chosen for this calculation is therefore wide, but close to the floor, so enough air is admitted to burn the chair; but it is low enough (1 m high) to prevent smoke leakage out of the room until the hot layer has extended down substantially. As the layer approaches the floor, it interferes with the ventilation of the fire, leading to computational difficulties. For larger fires, 2-3 MW, stable solutions to the burn algorithms cannot be obtained past 300 and 200 s,

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

respectively. However, because the hot layer has descended to an untenable level long before these times, this result does not affect TAE. Once the smoke has reached the height of the vent and the fire has begun to decrease in intensity, the hot-layer thickness can actually decrease, as seen in Figure 6-1. Using a normal 1 x 2-m (3.3 x 6.5-ft) doorway as the vent would slow the growth of the hot layer substantially and would not yield a good prediction of its growth in the worst case.

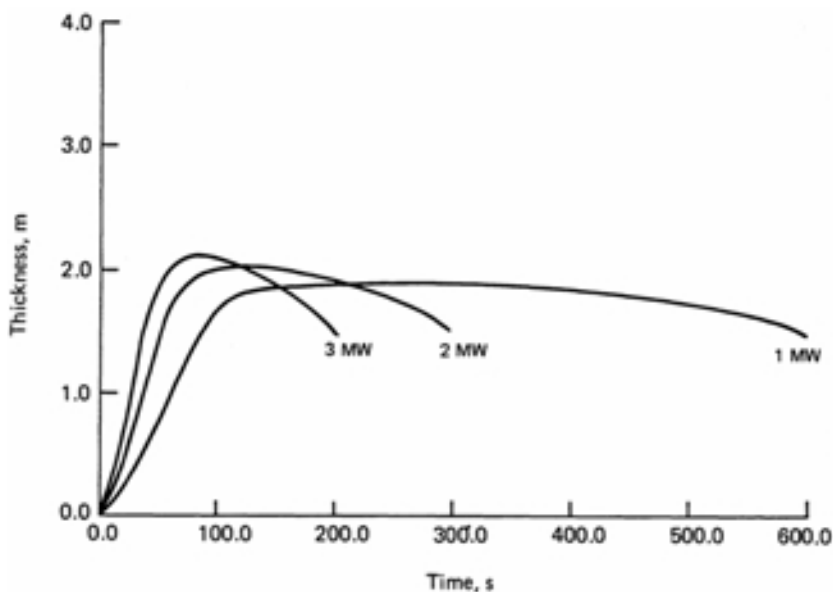


FIGURE 6-1 Thickness of hot layer vs. time of burning of three chairs. Curves are for burning chairs with peak heat release rates of 1, 2, and 3 MW.

Just how thick the hot layer is in the late stages of the fire depends on the exact height chosen for the vent. (However, a vent that is too close to the floor might not allow enough air into the room for free burning of the chair.) In real cases, the hot layer might extend down closer to the floor in the late stages of the fire than is predicted here. A hotter layer means a correspondingly larger smoke volume and hence a lower concentration of smoke than shown in Figure 6-2, Figure 6-3, and Figure 6-4. However, the

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

concentrations shown are good estimates of the worst-case smoke concentrations; actual concentrations might be slightly less than predicted, owing to a larger smoke volume, but they will not be greater. Moreover, these potential errors will not be encountered until after the smoke has descended.

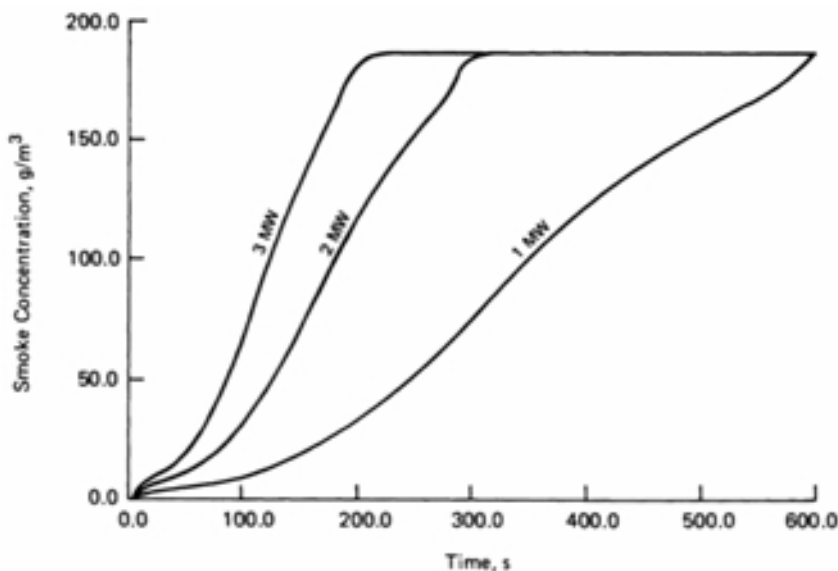


FIGURE 6-2 Smoke concentration in upper layer vs. time of burning of three chairs.

Results of Calculations

The Harvard code was run with the three simulated chair fires as input. The thickness of the upper layer and its temperature are calculated at 2-s intervals throughout the burning of the chair, and results are reported at 20-s intervals. Figure 6-1 shows the thickness of the hot layer as it grows down from the ceiling, Figure 6-5 shows the temperature of the hot layer. Figure 6-2 shows the increase in smoke concentration in the compartment as a function of time, and Figure 6-3 shows the increase in smoke dose (the product of concentration and time) over the same period. (A discussion of smoke dose may be found in Chapter 2.)

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

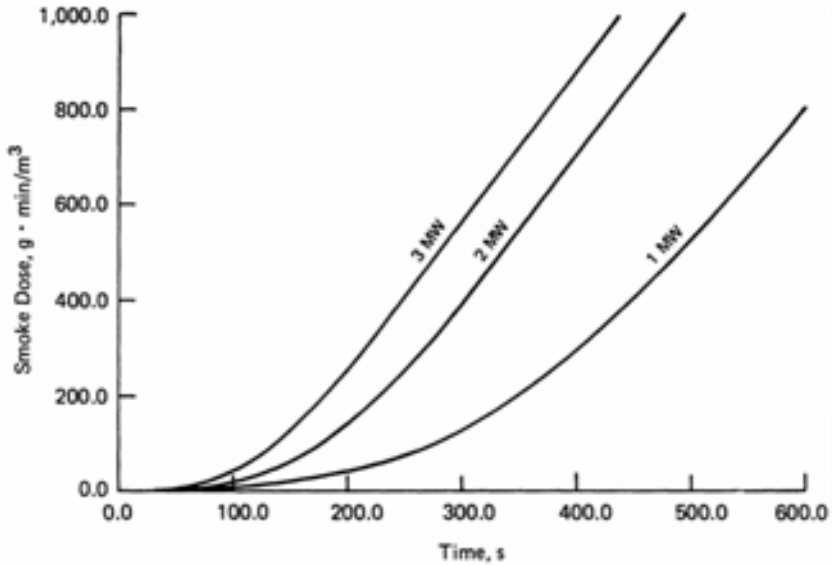


FIGURE 6-3 Smoke dose in room of origin from hot layer vs. time of burning of three chairs.

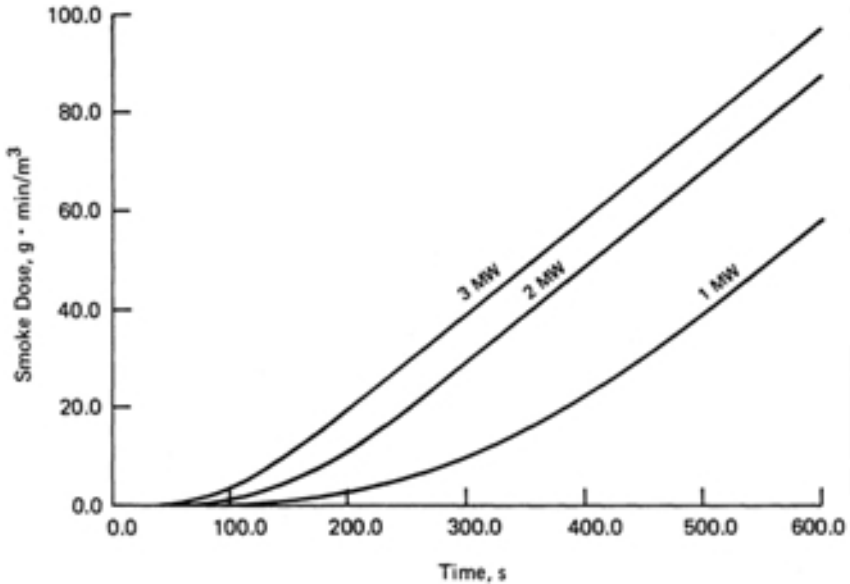


FIGURE 6-4 Smoke dose in adjacent room vs. time of burning of three chairs.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

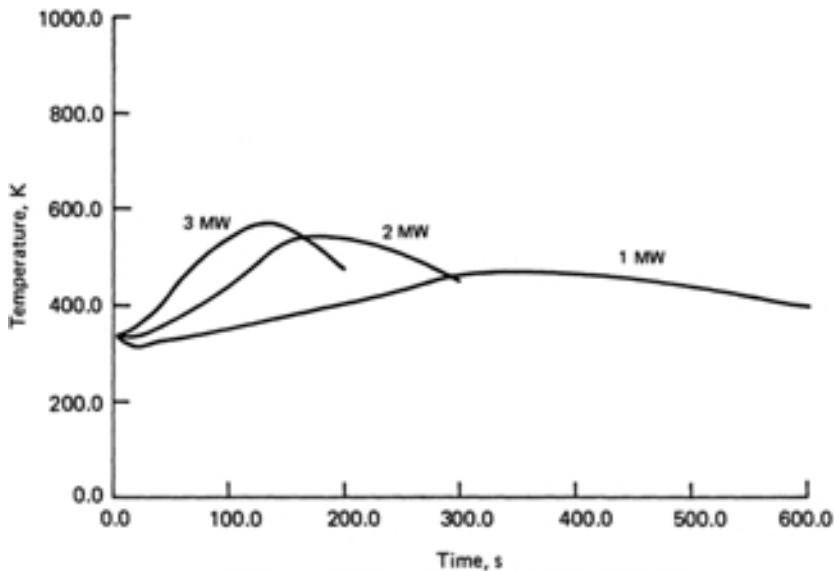


FIGURE 6-5 Temperature of hot layer vs. time of burning of three chairs.

Fires like those discussed above provide ample smoke density for rapid detector actuation, if the detector is situated so that it is exposed to the developing smoke layer at the earliest time, e.g., mounted on the ceiling or the top of the wall, not far below the ceiling. At

TABLE 6-2

Dimension	Chair		
	1	2	3
Peak heat release rate, MW	1	2	3
Time, s, when hot layer reaches 1 m above floor	85	50	35
Time, s, when hot layer reaches 180°C	275	110	60
Smoke dose encountered before onset of lethal temperature, g-min/m ³	30	22	7

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

20 s, the first interval for which a complete set of data are reported in this calculation, the smoke density from even the smallest of the three fires is 0.31 optical density unit per meter, and the thickness of the hot layer is 0.2 m (8 in.) or greater. Those conditions should be sufficient to trigger the alarm. Therefore, detectors in all three fire scenarios should alarm no later than 20 s after the fire begins.

Each of the three chair fires can be divided into intervals. First is a period of relatively unhindered escape between the time when the detector alarms and the time when the hot smoke layer extends down to 1 m above the floor. This interval is approximately 65 s for chair 1, 30 s for chair 2, and 15 s for chair 3. Second is an interval during which occupants must be presumed to be in direct contact with the hot layer, but the hot layer is not warm enough to be instantly injurious. This interval is 190 s for chair 1, 60 s for chair 2, and 25 s for chair 3. Because those exposed will be breathing smoke during this period, it is possible to estimate the amount of smoke (i.e., the dose) they will encounter (see [Table 6-2](#)). If the occupants encounter so much smoke that it hinders their escape, smoke toxicity can become important. Smoke from the chairs is unlikely to be lethal, because the dose received before the occurrence of a lethal temperature is too small. $L(Ct)_{50s}$ of most common cushioning materials appear to be about 300-1,000 g-min/m³,⁸ and the concentrations encountered here are less than one-tenth this amount. Put another way, the heat in the compartment would be so severe that the toxicity of the smoke would be relatively unimportant. Imposition of a toxicity limit on the smoke for chairs 2 and 3, however stringent, would not provide an environment as survivable as that for chair 1. In summary, a single chair, flaming in a small compartment, usually produces such heat that the compartment is untenable, or nearly so, before a substantial amount of smoke can be inhaled.

The situation is different for the scenario involving “bystanders.” [Figure 6-4](#) shows the buildup of smoke dose in a room adjacent to the fire room. It is assumed that 10% of the smoke mass leaks, through poorly sealed seams and fire wall penetrations, into an adjacent compartment of the same size as the original one. In leaking, the smoke loses most of its heat, so it mixes uniformly with

the air in the adjacent compartment, rather than forming a hot upper layer. Because the compartment is large, in comparison with the amount of smoke it contains, the nonlinearity of a growing fire accounts for only a very small portion of the total smoke; most of it leaks into the adjacent compartment after the chair fire is over. The leakage of smoke into the adjacent compartment is therefore constant with time, and the buildup of the smoke is nearly linear. Note that, for most materials, the $L(Ct)_{50}$ is 300-1,000 g-min/m³. This would take some 20-30 min to reach, regardless of which chair is burning. Thus, in contrast with the situation in the room of fire origin, the growth of the fire has a relatively small impact on fire hazard, and smoke toxicity can have a major impact.

Toxicity plays an important role in the room of fire origin only if the fire is so small that heat buildup is unimportant with respect to life safety. That is the case during smoldering combustion, but not flaming combustion. The rate of smoldering can, in principle, be measured in the laboratory. For this discussion, let it be assumed that the mass loss rate of the smoldering chair increases linearly with time. If so, Equation 5 of Chapter 2 can be used to calculate TAE from the smoldering fuel's $L(Ct)_{50}$ and rate of mass loss increase, k .

STEP 3: DECIDING ON MINIMAL ACCEPTABLE TAE

In these relatively simple scenarios, the time needed to escape is simply that required to get out of the immediate compartment. A potential regulator of furnishings must decide, either by calculation or by judgment, how much time is required and what safety factor should be allowed. Let it be supposed that for those in the compartment of origin 2 min is required for escape, and a 100% safety factor is to be applied to provide a margin of error. Then, 4 min becomes the target figure.

If ignition is by smoldering, burning will occur more slowly and smoke detectors cannot always be relied on. It is possible to envision those exposed sleeping while the chair smolders to completion--over a period of perhaps several hours. If it is desired to protect against such an occurrence, an escape time about this great might be needed; 1 h is chosen here as reasonable.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

For those exposed in an adjacent compartment, a flaming fire would probably be required, to provide the leakage rate necessary for the smoke to penetrate a fire-resistant assembly. In such a case, it might be reasonable to assume that 1 h of protection should be available to those on the other side of a fire-endurance wall, regardless of whether the wall leaks.

These quantities are summarized in the following table of needed TAEs:

Scenario Variant	Required TAE, min
1. Flaming fire--occupants in same compartment	4
2. Flaming fire--occupants on other side of fire wall; 10% of smoke leaks through	60
3. Smoldering fire--no heat; automatic detection uncertain	60

STEP 4: SPECIFYING FIRE AND SMOKE PROPERTIES

Let it be supposed that all three of the situations discussed above are deemed equally important and that fire and smoke properties that meet each one should be specified.

For variant 1, a 4-min TAE for the product under consideration means that it must be constructed of material with a bench-scale heat release rate of no more than about 100 kW/m². A rate any higher will result in lethal temperatures in less than 4 min. To keep those exposed from being overcome by smoke when they otherwise could escape, it is necessary that the smoke dose they encounter, 30 g·min/m³, not be debilitating. If one arbitrarily asserts the debilitating dose to be 20% of the lethal dose, then the minimal L(Ct)₅₀ allowed is 150 g·min/m³.

If the same chair is ignited by a source that gives rise to smoldering ignition, the situation is different. One can envision the chair smoldering for a long period,

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

until the concentration of smoke is high enough to be rapidly debilitating, and then bursting into flame. In such a case, it is not possible to ensure that 4 min will be available. If the fire remains in a smoldering state, Equation 5 of Chapter 2 can be used. For a TAE of 60 min, the quotient $L(Ct)_{50}/k$ must be above $150 \text{ min}/\text{m}^3$, where k is the rate of mass loss increase. This is a stringent requirement. An alternative is to control smoldering in some other way, such as by making the chair resistant to cigarette ignition.

Protecting those on the other side of a leaking fire-endurance wall requires that the atmosphere there be kept sublethal for 60 min. The requirements of variant 1 already stipulate a heat release rate of $100 \text{ kW}/\text{m}^2$ or less, so it can be calculated from the data giving rise to Figure 6-4 that the smoke dose reaching an adjacent compartment in 1 h is $660 \text{ g}\cdot\text{min}/\text{m}^3$ or less. Therefore, an $L(Ct)_{50}$ greater than this amount (multiplied, perhaps, by a safety factor) would be required. This is a more stringent requirement than that for escape from the fire compartment.

In sum, the hypothetical chair could have three restrictions, one each to deal with each aspect of the scenario. They are listed here.

Scenario Variant	Required Performance
1. Flaming fire--occupants in same compartment	Heat release rate no more than $100 \text{ kW}/\text{m}^2$ and $L(Ct)_{50}$ no less than $150 \text{ g}\cdot\text{min}/\text{m}^3$
2. Flaming fire--occupants on other side of fire wall; 10% of smoke leaks through	$L(Ct)_{50}$ no less than $660 \text{ g}\cdot\text{min}/\text{m}^3$
3. Smoldering fire--no heat; automatic detection uncertain	Smoldering ignition resistance: $L(Ct)_{50}/k$ no less than $250 \text{ min}^3/\text{m}^3$

If one wishes to respond equally to all three scenario variants, the requirements must be combined. Meeting the

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

requirement of variant 2 automatically satisfies the toxicity requirement of variant 1. The requirement of variant 3 is satisfied as well when k is less than 2.6. Hence the composite specifications for the chair are as follows:

Heat release rate no more than 100 kW/m^2

Smoke toxicity no less than 660 g-min/m^3

Rate of increase of mass loss (smoldering) no more than 2.6 g/min^2 .

It bears repeating that the case study is simplified, in that the heats of combustion, flame spread, and ignitability are assumed to be invariant. The scenarios chosen and the escape times estimated are for illustration and are probably not the product of systematic analysis that true regulatory decisions would be. The exercise is intended to illuminate the method, not to suggest a regulation.

CASE STUDY 2: CONCEALED COMBUSTIBLE MATERIAL

STEP 1: DEFINING SCENARIOS

Assessment of the fire hazard associated with combustible material concealed behind a wall or ceiling is more complicated than the assessment in the previous example, which focused on upholstered furniture. The first thing to be considered is whether the combustible material can be ignited by a small ignition source. Electric codes have helped to minimize both the sources of ignition in and the ignitability of electric products, except for electric ignitions (sparking and shorting); the principal threat to building occupants from concealed combustible material arises from its possible contribution to a fire that originates in a compartment and has sufficient intensity and duration to ignite material in a concealed space.

In the scenario chosen, a fire is burning in a room of typical size--13 ft (4 m) on a side with a normal 30-in.-wide (76-cm-wide) doorway. The compartment is faced with relatively noncombustible 0.5-in. gypsum board. The fuel in the room is not specified in detail, it being assumed only that there is enough to drive the room to flashover--about 30 kg/m^2 . A typical 3.5-in.

(8.9-cm) cavity behind the gypsum board contains combustible materials. The side of the cavity away from the room is also assumed to be faced with 0.5-in. gypsum board. Because the focus is on the combustible material behind the wall, details of ignition and spread of fire in the room are relatively unimportant.

STEP 2: COMPUTING TAE AS FUNCTION OF FIRE AND SMOKE PROPERTIES

Four tasks are required: determining the thermal conditions that the room contents (exposed fuel) will create, determining how thermal conditions in the room influence those behind the wall, determining how the concealed combustible material responds to the thermal conditions behind the wall, and assessing the contributions of the two fuels (exposed and concealed) to the hazard.

Fire Buildup in Room

The buildup of fire in the room can be calculated with the Harvard fire code as in the previous example or calculated on the basis of experimental data. In this case, experimental results are available. The time-temperature curve for the fire in this scenario has been measured as part of a study at the National Bureau of Standards;⁷³ the temperature profile of the hot upper layer is shown in [Figure 6-6](#). The shape of the profile is typical of fires that reach flashover. Changes in fuel characteristics and thermal properties of the wall linings influence the details of the curve, but the curve is essentially similar to that shown.

Thermal Condition Behind Wall

Once the temperature profile is known, the heat flow through the wall can be calculated with a one-dimensional finite-element analysis, such as presented in any elementary heat-transfer text.¹⁰⁵ The details of the calculation differ with the nature of the material exposed. If the combustible material is thermal or acoustic insulation that fills the cavity, heat will be transmitted primarily by conduction and radiation from

the gypsum board. If the material, such as pipe or electric wiring, occupies only a fraction of the cavity, its heating will depend (especially in the early stages) on its precise location in the cavity. In an effort to circumvent this kind of uncertainty, we calculated the temperature rise in the cavity by assuming that it was empty and that it lost heat only by conduction through the outside gypsum board. This assumption allows for the most rapid temperature rise, in that anything in the cavity will raise the heat capacity and slow the temperature buildup. Figure 6-7 shows the results of the temperature calculation. It is common in fire science to express a material's fire performance in terms of the energy imposed (flux), rather than the temperature. Thus, although the data in Figure 6-7 can be applied directly to estimation of how the concealed material behaves on being heated, it is more convenient to express the imposition of heat in terms of total energy flux. Figure 6-8 shows the estimated total flux striking a target in the middle of the cavity.

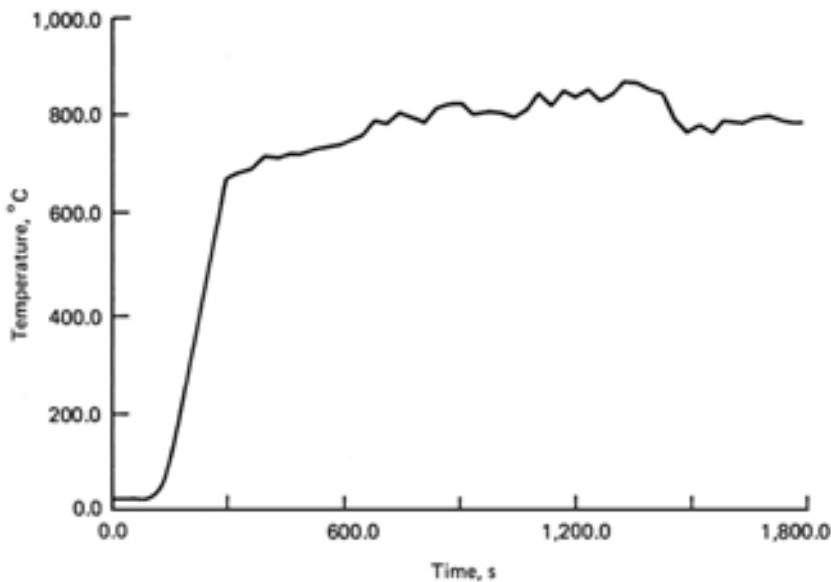


FIGURE 6-6 Temperature vs. time in full-scale room burn.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

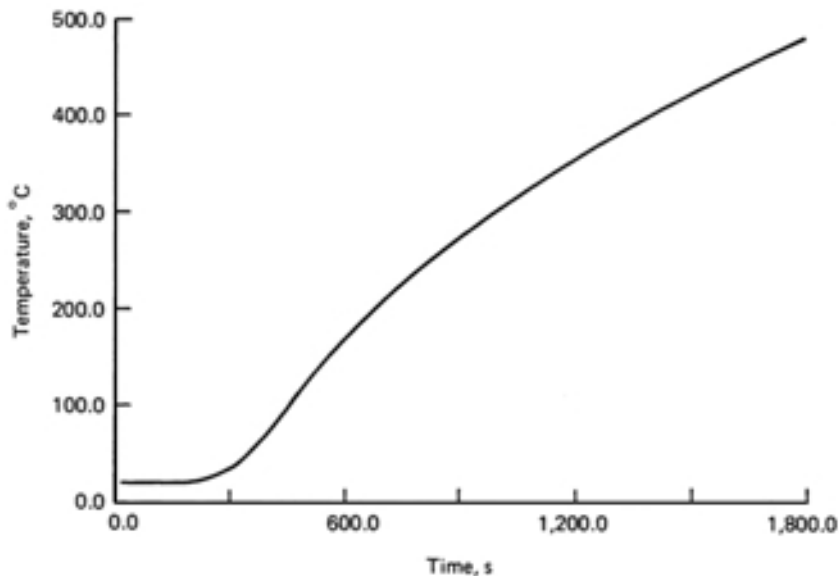


FIGURE 6-7 Temperature in wall cavity vs. time.

Response to Thermal Conditions Behind the Wall

It is necessary to know how readily the material decomposes. This is determined from a small-scale measurement. Material performance is gauged by exposing the material in the laboratory to a range of imposed radiant flux $q \cdot$ ” (in which q denotes heat, the dot denotes its derivative with respect to time, and the double prime denotes “per unit area”) and determining the rate of mass loss ($\dot{\cdot}$) at each flux. Plots of $\dot{\cdot}$ ” against $q \cdot$ ”, an idealized example of which is shown in Figure 6-9, have a positive slope and are usually fairly linear. In the simplest case, a material’s performance can be described in terms of the slope and X intercept of the plot. The X intercept, q_0 , is generally taken to be the minimal flux at which mass is lost. The slope is the reciprocal of the apparent heat of gasification (L), which governs how readily a material loses mass under a given heat load.

Contribution of Two Fires to Smoke

In this example, we are dealing with a fire after flashover. Conditions in the room of origin and nearby

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

will become rapidly lethal long before the concealed material is involved. An alternative time-dependent formulation of hazard is more useful; the contributions of concealed and exposed fuel are compared in terms of the relative volumes made lethal by their smoke in a given time. This is the time-based approach developed in [Chapter 2](#).

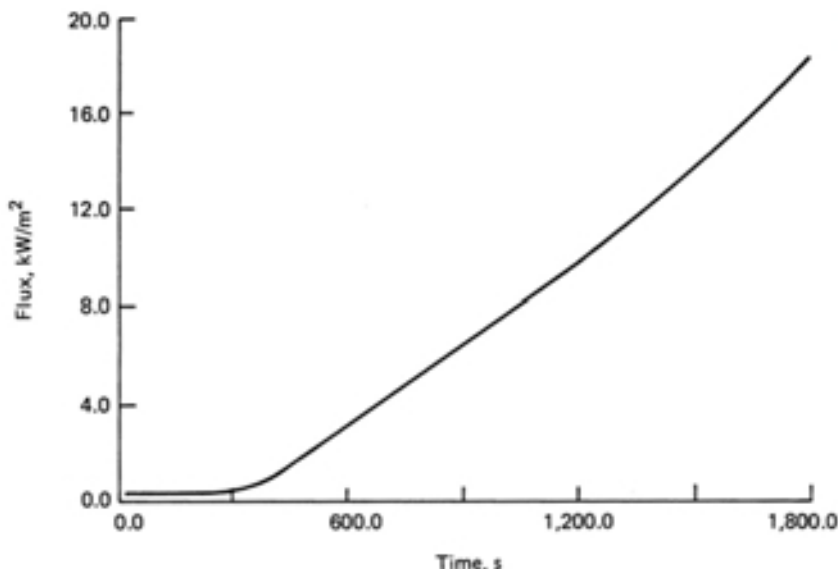


FIGURE 6-8 Thermal flux to material in wall cavity.

Equation 6 of Chapter 2 can be rewritten as follows:

$$\begin{aligned}
 V &= \frac{1}{L(Ct)_{50(1)}} \int \int_0^x \dot{m}_1(t) dt^2 + \frac{1}{L(Ct)_{50(2)}} \int \int_0^x \dot{m}_2(t) dt^2 \\
 &= \frac{f1(x)}{L(Ct)_{50(1)}} + \frac{f2(x)}{L(Ct)_{50(2)}},
 \end{aligned}$$

where V is the volume of space in which a lethal dose of smoke has been produced in time x. The contribution of the concealed combustible material, f2(x), is zero until

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

x is equal to the time (Figure 6-8) when $q \cdot t = \dot{q}_0^*$. V is then expressed in terms of a contribution from the room, given by the first term, and a contribution from the concealed material, given by the second.

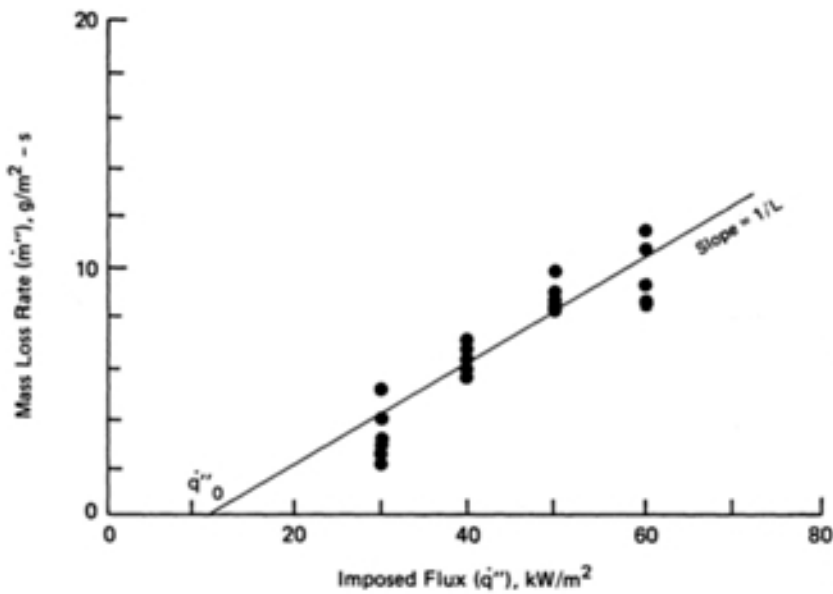


FIGURE 6-9 Rate of mass loss vs. imposed flux (idealized). L = apparent heat of vaporization.

The material burning in the room produces the smoke production curve of Figure 6-10. The curve can be approximated by series of relatively linear regions:

\dot{V} , g/s	Interval, s
0	0-180
48	180-1,080
32	1,080-1,800
0	71,800 (fuel exhausted)

Knowing the mass loss in the room permits calculation of $f_1(x)$. $f_2(x)$ is obtained by calculating the value of the expression:

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

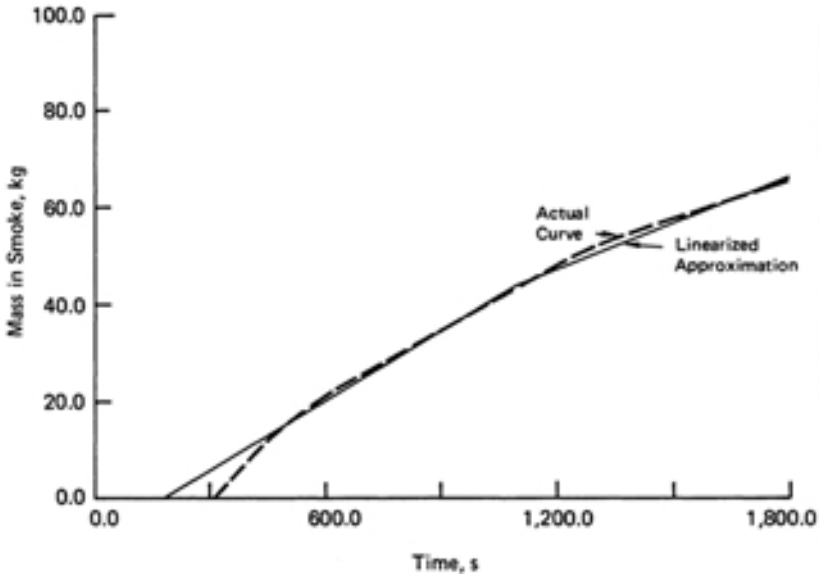


FIGURE 6-10 Mass loss in room fire.

$$\frac{A}{L} \int \int_{t_0}^x [\dot{q}''(t) - \dot{q}_o''] dt^2$$

where

A = area of concealed combustible material receiving energy in cavity,

L = apparent heat of vaporization of concealed combustible material,

$\dot{q}''(t)$ = flux reaching combustible material (from Figure 6-7), and

t_0 = time when $\dot{q}''(t) = \dot{q}_o''$.

Unless an analytic expression is available for $\dot{q}''(t)$, the integration must be performed numerically. Values of $f_2(x)$ per square meter of concealed combustible

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

material are listed in Table 6-3 for various values of \dot{q}_0'' and L and are in the range of 1-3,000 g-min. For comparison, integrating the mass curve of Figure 6-10 over 30 min to obtain $f_1(x)$ gives a value of 980,000 g-min.

TABLE 6-3

Mass-Time Product, g-min/m², for \dot{q}_0'' of:

L, kJ/g	10 kW/m ²	12 kW/m ²	14 kW/m ²	16 kW/m ²	18 kW/m ²
3	2,400	1,000	320	48	0
6	1,200	500	160	24	0
9	800	330	110	18	0
15	480	200	64	10	0

This does not complete the computation, however. Obviously, the wall eventually will be physically breached. The combustible material behind the wall is then exposed to the same thermal conditions as the interior of the room. The room fire discussed here burns out in 30 min, but it could burn longer if it had enough fuel.

If it is assumed that the fire is still burning vigorously, the volume rendered lethal after collapse can be approximated:

$$V \text{ after collapse} = \frac{t^2}{2} \left(\frac{A_1[\dot{q}'' - \dot{q}_0''(1)]}{L_1[L(Ct)_{50}(1)]} + \frac{A_2[\dot{q}'' - \dot{q}_0''(2)]}{L_2[L(Ct)_{50}(2)]} \right),$$

where A and L are the combustible-material surface area and flammability, respectively, of the room fuel and wall contents. The time, t, is measured from the time of wall failure, and \dot{q}'' is average flux in the room. After flashover, \dot{q}'' will be 60-100 kW/m², which is large in comparison with \dot{q}_0'' , which exceeds 30 kW/m² for only a few organic materials. Thus, as a first approximation, the ratio of the contributions of the two fuels to smoke toxicity is simply:

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

$$\text{Ratio} = \frac{A_1}{A_2} \left(\frac{L_2}{L_1} \right) \left(\frac{L(Ct)_{50}(2)}{L(Ct)_{50}(1)} \right).$$

(1)

STEPS 3 AND 4: DECIDING ON MINIMAL ACCEPTABLE TAE AND SPECIFYING FIRE AND SMOKE PROPERTIES

The scenario under discussion in this case study is a large, well-developed fire, which reaches high intensity and is thus easily detectable in a few minutes. Until the concealed combustible material becomes involved, the fire analysis is similar to the one in Case Study 1: the buildup is controlled by the flammability properties of the room fuels. Note that conditions in the neighborhood of the fire become untenable in just a few minutes. Thus, the influence of the concealed combustible material on TAE is zero, unless the compartment that the smoke fills is large. Hence, this scenario is of only limited interest in small buildings. In larger structures, where alarm and evacuation procedures can be complex, it is not unreasonable to focus on conditions well after the fire has begun. The time selected here--30 min--typifies the focus for escape and evacuation in large structures, such as a high-rise building.

Suppose that one wishes to assess the contribution of concealed combustible piping in this scenario. A generous estimate of the surface area of the pipe is 3 m². Table 6-4 shows the contribution to V of both the room fuel and the pipe for several values of pipe flammability and smoke toxicity. A smoke toxicity of 750 g·min/m³ (i.e., an LC₅₀ of 25 g/m³ over a 30-min exposure) is assumed for the room fuel. Note that the contribution of the pipe is extremely sensitive to \dot{q}_0 --compare parts a and c of Table 6-4. However, even at a very low \dot{q}_0 and a toxicity 10 times that of the room fuel, the pipe contributes less than 10% of the total toxicity for the first 30 min of burning. If it is desired to eliminate the contribution of the pipe entirely during this period, the simplest way is to specify a

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

\dot{q}_0 above 18 kJ/g. Then, according to the data in Figure 6-7, the pipe would not reach its decomposition point during the interval under study.

TABLE 6-4

L, kJ/g	Total Volume Made Lethal	Room Contribution	Pipe Contribution	Room: Pipe Ratio
(a) $\dot{q}_0 = 10 \text{ kW/m}^2$; $L(\text{Ct})_{50}(\text{pipe}) = 750 \text{ g}\cdot\text{min/m}^3$:				
2	1,315	1,300	15	90:1
3	1,310	1,300	10	100:1
6	1,305	1,300	5	300:1
9	1,303	1,300	3	400:1
15	1,302	1,300	2	700:1
(b) $\dot{q}_0 = 10 \text{ kW/m}^2$; $L(\text{Ct})_{50}(\text{pipe}) = 75 \text{ g}\cdot\text{min/m}^3$:				
2	1,450	1,300	150	9:1
3	1,400	1,300	100	13:1
6	1,350	1,300	50	26:1
9	1,330	1,300	30	43:1
15	1,320	1,300	20	65:1
(c) $\dot{q}_0 = 16 \text{ kW/m}^2$; $L(\text{Ct})_{50}(\text{pipe}) = 750 \text{ g}\cdot\text{min/m}^3$:				
3	1,300.2	1,300	0.2	6,500:1
6	1,300.1	1,300	0.1	13,000:1
9	1,300.07	1,300	0.07	19,000:1
15	1,300.04	1,300	0.04	33,000:1
(d) $\dot{q}_0 = 16 \text{ kW/m}^2$; $L(\text{Ct})_{50}(\text{pipe}) = 75 \text{ g}\cdot\text{min/m}^3$:				
3	1,302	1,300	2	700:1
6	1,301	1,300	1	1,000:1
9	1,300.7	1,300	0.7	2,000:1
15	1,300.4	1,300	0.4	3,000:1

The contribution of the concealed combustible material that is “acceptable” is arbitrary. Let it be supposed

About this PDF file: This new digital representation of the original work has been reproduced from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

that one chooses to limit the pipe's toxicity contribution to 10% of that of the room, regardless of whether the wall has collapsed--a very stringent requirement. To hold the pipe's contribution below this in the pre-collapse case (interpolating values in the last column of Table 6-4) it can be seen that at 10 kW/m² pipe having an L value of about 2.3 kJ/g and an L(Ct)₅₀ of 75 g·min/m³ produces a toxic-volume ratio of 10:1. For any given critical flux, the toxic contribution of the pipe is proportional to the product of its apparent heat of gasification and its L(Ct)₅₀. Thus, to provide a maximum of the toxic contribution, a pipe having a critical flux of 10 kW/m² will show [L (pipe) x L (Ct)₅₀ (pipe)] of at least 173 kJ·min/m³.

For the postcollapse case, Equation 1 may be used. A conservative estimate of the combustible surface area in the room is 10 m²; an L value of 2 kJ/g is reasonable and, as above, an L(Ct)₅₀ of 750 g·min/m³. To maintain the concealed pipe's contribution at less than 10% of the total, on the basis of these assumptions, requires that [L (pipe) x L(Ct)₅₀ (pipe)] be at least 4,500 kJ·min/m³. This is a much more restrictive requirement than that dictated by precollapse conditions.

The choice of requirements is reflected in the time chosen for protection and can be summarized as follows:

Option	Required Performance
1. No contribution from pipe for 30 min (precollapse)	\dot{q}_0 no less than 18 kW/m ²
2. Less than 10% of toxic volume from pipe before collapse	\dot{q}_0 no less than 10 kW/m ² and [L x L (Ct) ₅₀] no less than 175 kJ·min/m ³
3. Less than 10% of toxic volume from pipe after collapse	[L x L(Ct) ₅₀] no less than 4,500 kJ·min/m ³

The most severe restrictions would be to allow no pipe contribution to toxicity before collapse and to limit it to 10% after collapse, in which case the provisions of Options 1 and 3 apply. A somewhat less restrictive pre-collapse condition is obtained by using a lower \dot{q}_0 , as in Option 2.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Note that the product of $L(Ct)_{50}$ and L is restricted, not toxicity by itself. Thus, a higher toxicity can be offset by a lower L , and vice versa. This approach provides the most freedom of choice among pipe materials without changing the pipe hazard in this scenario.

SUMMARY

DEFINING THE SCENARIO

The scenario of concern should be developed as completely as possible. Specifying the scenario of concern includes specifying the fire properties of the material being assessed, its conditions of use, and the fire conditions to which it will be assumed to be exposed. The hypothetical worst case is often chosen for this exercise, but is not necessarily the most informative example. The appropriate scenario is sometimes obvious, but more commonly is a consensus contributed to by persons with substantial knowledge of the product, the product use, and the modeling process.

RELATING TAE TO FIRE PROPERTIES OF MATERIALS

Most of the well-documented mathematical fire models available can be adapted to various situations. For scenarios in which those exposed are relatively close to the fire--e.g., in the same room--a single-room model will often suffice. If those exposed are farther away, a model that treats smoke transport in more detail might be required. In either case, however, the key component of the model is the part that relates the flammability characteristics of the fuel to the rates of smoke and heat production.

Requirements will eventually be set for flammability characteristics, as well as for smoke toxicity, so it is crucial that the model be able to provide quantitative relationships between a given set of fire and smoke properties and TAE. Specific models are discussed in detail in [Chapter 3](#).

SELECTING MINIMAL ACCEPTABLE TAE

In general, one wishes to ensure that TAE is larger than TNE, but selecting the degree to which TAE exceeds TNE is a matter of choice. Some margin is desirable, if for no other reason than to allow for inadequacies of the model.

SPECIFYING PRODUCT PERFORMANCE

In principle, the hazards associated with smoke are controlled only if both the smoke toxicity and the properties that determine smoke production are controlled. The modeling process and selection of a TAE should make it apparent that various combinations of toxicity and smoke production can provide acceptable overall performance. Performance specifications should reflect that fact.

The toxic potency of the smoke produced by the burning product is assessed with a standard toxicity test (see [Chapter 5](#)), possibly with a chemical test of the smoke to assist in estimating the LC_{50} or $L(Ct)_{50}$.

REFERENCES

1. Adams, C., T. Moisan, A. J. Chandrasekhar, and R. Warpeha. 1979. Endobronchial polyposis secondary to thermal inhalational injury. *Chest* 75:643-645.
2. Alarie, Y. 1966. Irritating properties of airborne materials to the upper respiratory tract. *Arch. Environ. Health* 13:433-449.
3. Alarie, Y. 1973. Sensory irritation by airborne chemicals. *CRC Crit. Rev. Toxicol.* 2:299-363.
4. Alarie, Y. 1985. The toxicity of smoke from polymeric materials during thermal decomposition. *Ann. Rev. Pharmacol. Toxicol.* 25:325-347.
5. Alarie, Y., and R. C. Anderson. 1979. Toxicologic and acute lethal hazard evaluation of thermal decomposition products of synthetic and natural polymers. *Toxicol. Appl. Pharmacol.* 51:341-362.
6. Alarie, Y., and R. C. Anderson, 1981. Toxicologic classification of thermal decomposition products of synthetic and natural polymers. *Toxicol. Appl. Pharmacol.* 57:181-188.
7. Alarie, Y., and J. E. Luo. 1986. Sensory irritation by airborne chemicals: A basis to establish acceptable levels of exposure, pp. 91-100. In C. S. Barrow, Ed. *Toxicology of the Nasal Passages*. New York: Hemisphere Publishing Corporation.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

8. Alexeeff, G. V., and S. C. Packham. 1984. Evaluation of smoke toxicity using concentration-time products. *J. Fire Sci.* 2:362-379.
9. Alpert, R. L. 1972. Calculation of response time of ceiling-mounted fire detectors. *Fire Technol.* 18:181-195.
10. Ambiavagar, M., J. Chalon, and I. Zargham. 1974. Tracheobronchial cytologic changes following lower airway thermal injury. A preliminary report. *J. Trauma* 14:280-298.
11. American Conference of Governmental Industrial Hygienists. 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio: ACGIH.
12. American Society for Testing and Materials. 1983. Standard methods of fire tests of building construction and materials, pp. 337-363. In *Annual Book of ASTM Standards*. Vol. 04.07. ASTM E119-83. Philadelphia, Pa.: ASTM.
13. American Society for Testing and Materials. 1985. Standard terminology relating to fire standards, pp. 428-432. In *Annual Book of ASTM Standards*. Vol. 04.07. ASTM E176-85. Philadelphia, Pa.: ASTM.
14. American Society for Testing and Materials. 1985. Standard test method for heat and visible smoke release rates for materials and products, pp. 906-927. In *Annual Book of ASTM Standards*. Vol. 04.07. ASTM E906-83. Philadelphia, Pa.: ASTM.
15. Ames, B. N., J. McCann, and E. Yamasaki. 1975. Methods for detecting carcinogens and mutagens with the *Salmonella*/mammalian-microsome mutagenicity test. *Mutat. Res.* 31:347-363.
16. Anderson, E. W., R. J. Andelman, J. M. Strauch, N. J. Fortuin, and J. H. Knelson. 1973. Effect of low-level carbon monoxide exposure on onset and duration of angina pectoris: A study in ten patients with ischemic heart disease. *Ann. Intern. Med.* 79:46-50.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

17. Anderson, R. A., and W. A. Harland. 1982. Fire deaths in the Glasgow area. III. The role of hydrogen cyanide. *Med. Sci. Law* 22:35-40.
18. Anderson, R. A., A. A. Watson, and W. A. Harland. 1981. Fire deaths in the Glasgow area. II. The role of carbon monoxide. *Med. Sci. Law* 21:288-294.
19. Anderson, R. C., P. A. Croce, F. G. Feeley, III, and J. D. Sakura. 1983. Study to Assess the Feasibility of Incorporating Combustion Toxicity Requirements into Building Material and Furnishing Codes of New York State. Vols. 2 and 3. Final Report to Department of State, Office of Fire Prevention and Control. Reference 88712. Cambridge, Mass.: Arthur D. Little. 260 pp.
20. Anderson, R. C., M. F. Stock, and Y. C. Alarie. 1978. Toxicologic evaluation of thermal decomposition products of synthetic cellular materials. *J. Combust. Toxicol.* 5:111-129.
21. Ator, N. A., W. H. Merigan, and R. W. McIntire. 1976. The effects of brief exposures to carbon monoxide on temporally differentiated responding. *Environ. Res.* 12:81-91.
22. Axford A., C. McKerrow, A. Jones, and P. LeQuesne. 1976. Accidental exposure to isocyanate fumes a group of firemen. *Br. J. Ind. Med.* 33:65-71.
23. Ayres, S. M., S. Giannelli, and H. Mueller. 1970. Myocardial and systemic responses to carboxy-hemoglobin. *Ann. N. Y. Acad. Sci.* 174:268-293.
24. Babrauskas, V. 1984. Bench-scale methods for prediction for full-scale fire behavior of furnishing and wall linings. In Society of Fire Protection Engineers Technology Report. 84 (10):1-25. Gaithersburg, Md.: National Bureau of Standards Center for Fire Research.
25. Babrauskas, V. 1984. Development of the cone calorimeter. A bench-scale heat release rate apparatus, based on oxygen consumption. *Fire Mater.* 8:81-95.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

26. Baker, S. P., B. O'Neill, and R. S. Karpf. 1984. *The Injury Fact Book*. Lexington, Mass.: D. C. Heath and Co. 313 pp.
27. Balmes, J., R. M. Cullen, and R. A. Matthay. 1983. Occupational and environmental lung diseases, pp. 361-402. In R. George, R. Light, and R. Matthay, Eds. *Chest Medicine*. New York: Churchill Livingstone.
28. Barrow, C. S., Y. Alarie, and M. F. Stock. 1978. Sensory irritation and incapacitation evoked by thermal decomposition products of polymers and comparisons with known sensory irritants. *Arch. Environ. Health* 33:79-88.
29. Baum, H. R., and R. G. Rehm. 1984. Calculations of three-dimensional buoyant plumes in enclosures. *Combust. Sci. Technol.* 40:55-77.
30. Beal, D. D., J. T. Lambeth, and G. H. Conner. 1968. Follow-up studies on patients treated with steroids following pulmonary thermal and acrid smoke injury. *Laryngoscope* 78:396-403.
31. Benjamin, I., G. Heskestad, R. Bright, and T. Hayes. 1979. *An Analysis of the Report on Environments of Fire Detectors*. New York, N.Y.: Fire Detection Institute. 33 pp.
32. Benjamin/Clarke Associates, Inc. 1984. *Fire Deaths: Causes and Strategies for Control*. Lancaster, Pa.: Technomic Publishing Co. 72 pp.
33. Bevan, D. R., and J. R. Lakowitz. 1984. Mediation of toxicological properties of chemicals by particulate matter, pp. 141-180. In *Hazard Assessment of Chemicals: Current Developments*. New York: Academic Press.
34. Bevan, D. R., S. C. Riemer, and J. R. Lakowitz. 1981. Effects of particulate matter on rates of membrane uptake of polynuclear aromatic hydrocarbons. *J. Toxicol. Environ. Health* 8:241-250.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

35. Birky, M., B. M. Halpin, Y. H. Caplan, R. S. Fisher, J. M. McAllister, and A. M. Dixon. 1979. Fire fatality study. *Fire Mater.* 3:211-217.
36. Birky, M., D. Malek, and M. Paabo. 1983. Study of biological samples obtained from victims of MGM Grand Hotel fire. *J. Anal. Toxicol.* 7:265-271.
37. Birky, M., M. Paabo, and J. Brown. 1980. Correlation of autopsy data and materials involved in the Tennessee Jail fire. *Fire Saf. J.* 2:17-22.
38. Birky, M., and K. J. Voorhees. (In press.) Use of soot analysis as an investigative tool in aircraft fires.
39. Boettner, E. A., G. Ball, and B. Weiss. 1969. Analysis of the volatile combustion products of vinyl plastics. *J. Appl. Polym. Sci.* 13:377-391.
40. Boettner, E. A., and B. Weiss. 1967. An analytical system for identifying the volatile pyrolysis products of plastics. *Am. Ind. Hyg. Assoc. J.* 28:535-540.
41. Buckley, L. A., X. Z. Jiang, R. A. James, K. T. Morgan, and C. S. Barrow. 1984. Respiratory tract lesions induced by sensory irritants at the RD₅₀ concentration. *Toxicol. Appl. Pharmacol.* 74:417-429.
42. Bukowski, R. (In press.) Evaluation of furniture fire hazard using a hazard assessment computer model. *Fire Mater.*
43. Bull, J. P., and A. J. Fisher. 1954. A study of mortality in a burn unit: A revised estimate. *Ann. Surg.* 139:269-274.
44. Cahalane, M., and R. H. Demling. 1984. Early respiratory abnormalities from smoke inhalation. *J. Am. Med. Assoc.* 251:771-773.
45. Carroll, W. F., J. J. Beitel, A. F. Grand, C. A. Bertelo, G. F. Smith, and R. O. Gardner. 1984. Thermal Decomposition of PVC in Large Apparatus: The Generation, Transport and Decay of Hydrogen

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

- Chloride, pp. 6-16. Presented to the Third U.S.-Japan-Canada Cooperative Study Group on the Toxicity of Combustion Products, October 23-25. Ottawa, Canada.
46. Charan, N. B., C. G. Myers, S. Lakshminarayan, and T. M. Spencer. 1979. Pulmonary injuries associated with acute sulfur dioxide inhalation. *Am. Rev. Respir. Dis.* 119:555-560.
 47. Chu, E. H. Y., and H. V. Malling. 1968. Mammalian cell genetics. II. Chemical induction of specific locus mutations in Chinese hamster cells *in vitro*. *Proc. Nat. Acad. Sci. U.S.A.* 61:1306-1312.
 48. Church, D. F., and W. A. Pryor. 1985. Free radical chemistry of cigarette smoke and its toxicological implications. *Environ. Health Perspec.* 64:111-126.
 49. Clark, C. J., D. Campbell, and W. H. Reid. 1981. Blood carboxyhaemoglobin and cyanide levels in fire survivors. *Lancet* 1:1332-1335.
 50. Clarke, F. B., III. 1986. Fire hazard assessment, pp. 21-2-21-9. In A. E. Cote, Ed. *Fire Protection Handbook*. 16th ed. Quincy, Mass.: National Fire Protection Association.
 51. Clarke, F. B., I. A. Benjamin, and J. W. Clayton. 1982. *An Analysis of Current Knowledge in Toxicity of the Products of Combustion*. Prepared for the Committee on the Toxicity of Combustion of the National Fire Protection Association Standards Council. Quincy, Mass.: National Fire Protection Association. 165 pp.
 52. Clarke, F. B., III, and J. Ottoson. 1976. Fire death scenarios and firesafety planning. *Fire J.* 70:20-22,117-118.
 53. Colardyn, F., M. Vanderstraeten, H. Lamont, and T. van Peteghem. 1976. Acute inhalation-intoxication by combustion of polyvinylchloride. *Intl. Arch. Occup. Environ. Health* 38:121-127.
 54. Coleman, W. E., D. S. Lester, and C. H. Gorski. 1968. The particles resulting from polytetra

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

- fluoroethylene (PTFE) pyrolysis in air. *Am. Ind. Hyg. Assoc. J.* 29:54-60.
55. Cooper, L., and D. Stroup. 1982. Calculating Available Safe Egress Time (ASET)--A Computer Program and User's Guide. NBSIR 82-2578. Washington, D.C.: U.S. Department of Commerce. 131 pp.
 56. Cooper, L. Y., and D. W. Stroup. 1985. ASET-A computer program for calculating available safe egress time. *Fire Saf. J.* 9:29-45.
 57. Coppock, R. 1984. *Social Constraints on Technological Progress*. Brookfield, Vt.: Gower Publishing Co. 291 pp.
 58. Cornish, H. H., and E. L. Abar. 1969. Toxicity of pyrolysis products of vinyl plastics. *Arch. Environ. Health* 19:15-21.
 59. Cox, G., S. Kumar, and N. C. Markatos. 1985. Some field model validation studies, pp. 159-172. In *Fire Safety Science. Proceedings of the First International Symposium*, October 7-11. New York: Hemisphere Publishing Corporation.
 60. Crane, C. R., D. C. Sanders, B. R. Endecott, J. K. Abbott, and P. W. Smith. 1977. *Inhalation Toxicology. I. Design of a Small-Animal Test System. II. Determination of the Relative Toxic Hazards of 75 Aircraft Cabin Materials*. FAA-AM-77-9. Washington, D.C.: U.S. Federal Aviation Administration, Office of Aviation Medicine. 49 pp.
 61. Cullis, C. F., and M. M. Hirschler. 1981. *The Combustion of Organic Polymers*. Oxford, England: Clarendon Press.
 62. Curtis, M. H., J. R. Hall, and P. R. LeBlanc. 1985. Analysis of multiple-death fires in the United States during 1984. *Fire J.* 79:18-81.
 63. Curtis, M. H., and P. R. LeBlanc. 1984. Multiple-death fires in the United States. *Fire J.* 78:33-54.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

64. Demling, R. H. 1985. Medical progress: Burns. *N. Engl. J. Med.* 313:1389-1398.
65. Deutsche Industrie-Norm. 1981. Erzeugung Thermischer Zersetzungsprodukte von Werkstoffen unter Luftzufuhr und ihre Toxikologische Prufung. 1. Zersetzungsgerat und Bestimmung der Versuchstemperatur. 2. Verfahren zur thermischen Zersetzung. DIN 53436. Berlin: Beudh-Verlag Valrggrafen. (distributed in United States by International Publishers Services)
66. Dilley, J. V., G. T. Pryor, and S. B. Martin. 1978. Evaluation of the Potential Toxicity of Candidate Aircraft Materials: Final Report. Menlo Park: SRI International.
67. Douglas, D. B., R. B. Douglas, D. Oakes, and G. Scott. 1985. Pulmonary function of London firemen. *Br. J. Ind. Med.* 42:55-58.
68. Drinker, C. K. 1938. Carbon Monoxide Asphyxia. New York: Oxford University Press. 276 pp.
69. Drysdale, D. 1985. An Introduction to Fire Dynamics. New York: John Wiley and Sons. 424 pp.
70. Enterline, P., and M. McKiever. 1963. Differential mortality from lung cancer by occupation. *J. Occup. Med.* 5:283-290.
71. European Chemical Industry Ecology and Toxicology Centre. 1985. Acute toxicity tests, LD₅₀ (LC₅₀) determinations and alternatives. D-1985-3001-30. Brussels: European Chemical Industry Ecology and Toxicology Centre. 38 pp.
72. Evans, D. D. 1985. Calculating sprinkler actuation time in compartments. *Fire Saf. J.* 9:147-155.
73. Fang, J., and J. N. Breese. 1980. Fire Development in Residential Basement Rooms. National Bureau of Standards NBSIR 80-2120. Washington, D.C.: U.S. Department of Commerce. 97 pp. (available from National Technical Information Service, Springfield, Va., as PB81-141509)

74. Farrar, D. G. 1980. The effect of the sensory irritant component of a combustion atmosphere derived from Douglas-fir on the leg-flexion avoidance-response of the rat. *J. Combust. Toxicol.* 7:3-22.
75. Farrar, D. G., and W. A. Galster. 1980. Biological end-points for the assessment of the toxicity of the products of combustion of materials. *Fire Mater.* 4:50-58.
76. Feuer, E., and K. Rosenman. 1986. Mortality in police and firefighters in New Jersey. *Am. J. Ind. Med.* 9:517-527.
77. Fineberg C., B. J. Miller, and F. F. Allbritten, Jr. 1954. Thermal burns of the respiratory tract. *Surg. Gynecol. Obstet.* 98:318-323.
78. Fisher, F. L., F. W. Mowrer, and R. B. Williamson. 1983. A room fire screening test procedure. *Fire Technol.* 19:238-250.
79. Foley, F. D., J. A. Moncrief, and A. D. Mason, Jr. 1968. Pathology of the lung in fatally burned patients. *Ann. Surg.* 167:251-264.
80. Forbes, W. H., D. B. Dill, H. De Silva, and F. M. Van Deventer. 1937. The influence of moderate carbon monoxide poisoning upon the ability to drive automobiles. *J. Ind. Hyg. Toxicol.* 19:598-603.
81. Forster, R. E. 1969. Reaction of carbon monoxide with heme proteins, pp. 10-13. In National Research Council, Committee on Effects of Atmospheric Contaminants on Human Health and Welfare. *Effects of Chronic Exposure to Low Levels of Carbon Monoxide on Human Health, Behavior, and Performance.* Washington, D.C.: National Academy of Sciences.
82. Fowell, A. J. 1985. Assessing toxic hazard as it relates to overall fire hazard. *Fire Technol.* 21:199-212.
83. Friedman, R. 1985. Some unresolved fire chemistry problems, pp. 349-360. In *Fire Safety Science. Proceedings of the First International Symposium.*

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

- October 7-11. New York, N.Y.: Hemisphere Publishing Corporation.
84. Friedman, R. 1981. Status of Mathematical Modeling of Fires. FMRC RC81-BT-5. Norwood, Mass.: Factory Mutual. 9 pp.
 85. Gad, S. C., and A. C. Smith. 1983. Influence of heating rates on the toxicity of evolved combustion products: Results and a system for research. *J. Fire Sci.* 1:465-479.
 86. Getzen, L. C., and E. W. Pollak. 1981. Fatal respiratory distress in burned patients. *Surg. Gynecol. Obstet.* 152:741-744.
 87. Ginsberg, M. D. 1979. Delayed neurological deterioration following hypoxia. *Adv. Neurol.* 26:21-44.
 88. Gordon, J. 1943. Acute tracheobronchitis complicated by bronchial stenosis following the inhalation of sulfur dioxide. *N.Y. State J. Med.* 43:1054-1056.
 89. Gosink, B. B., P. J. Friedman, and A. A. Liebow. 1973. Bronchiolitis obliterans: Roentgenologic pathologic correlation. *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 117:816-832.
 90. Guyton, A. C. 1986. *Textbook of Medical Physiology*. 7th ed. Philadelphia: Saunders. 1088 pp.
 91. Hartung, R., G. L. Ball, E. A. Boettner, R. Rosenbaum, and Z. R. Hollingsworth. 1977. The performance of rats on a rotorod during exposure to combustion products of rigid polyurethane foams and wood. *J. Combust. Toxicol.* 4:506-522.
 92. Hartzell, G. E., S. C. Packham, A. F. Grand, and W. G. Switzer. 1985. Modelling of toxicological effects of fire gases. III. Quantification of post-exposure lethality of rats from exposure to HCl atmospheres. *J. Fire Sci.* 3:195-207.

93. Hartzell, G. E., S. C. Packham, and W. G. Switzer. 1983. Toxic products from fires. *Am. Ind. Hyg. Assoc. J.* 44:248-255.
94. Henderson, R. F., J. M. Benson, F. F. Hahn, C. H. Hobbs, R. K. Jones, J. L. Mauderly, R. O. McClellan, and J. A. Pickrell. 1985. New approaches for the evaluation of pulmonary toxicity: Bronchoalveolar lavage fluid analysis. *Fundam. Appl. Toxicol.* 5:451-458.
95. Heskestad, G. 1981. A Fire Products Collector for Calorimetry into the MW Range. Factory Mutual Research Corporation Technical Report J.I. OC2E1.RA. Norwood, Mass.: Factory Research Corporation. 100 pp.
96. Heskestad, G. 1973. Physical modeling of fire. *J. Fire Flammability* 6:253-273.
97. Higgins, E. A., V. Fiorca, A. A. Thomas, and H. V. Davis. 1972. Acute toxicity of brief exposures to HF, HCl, NO₂, and HCN with and without CO. *Fire Technol.* 8:120-130.
98. Hilado, C. J. 1973. *Flammability Test Methods Handbook*. Vol. I. Westport, Conn.: Technomic. 420 pp.
99. Hilado, C. J., and C. R. Crane. 1977. Comparison of results with the USF/NASA and FAA/CAMI toxicity screening test methods. *J. Combust. Toxicol.* 4:56-60.
100. Hilado, C. J., H. J. Cumming, A. M. Machado, J. E. Schneider, C. R. Crane, D. C. Sanders, B. R. Endecott, and J. K. Abbott. 1977. Comparison of animal responses to the combustion products generated by the two test procedures, the USF/NASA methodology and the FAA/CAMI system. *J. Combust. Toxicol.* 4:325-359.
101. Hilado, C. J., and L. A. Gall. 1977. Relative toxicity of pyrolysis products of some wood samples. *J. Combust. Toxicol.* 4:193-199.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

102. Hilado, C. J., N. V. Huttlinger, and B. A. O'Neill. 1978. Effect of heating rate on toxicity of pyrolysis gases from some wood samples. *J. Combust. Toxicol.* 5:25-38.
103. Hilado, C. J., W. H. Marcussen, A. Furst, and H. A. Leon. 1976. Effect of species on relative toxicity of pyrolysis products. *J. Combust. Toxicol.* 3:125-134.
104. Hilado, C. J., and J. E. Schneider. 1979. Toxicity of pyrolysis gases from polytetrafluoroethylene. *J. Combust. Toxicol.* 6:91-98.
105. Holman, J. P. 1981. *Heat Transfer*. 5th ed. New York: McGraw-Hill. 672 pp.
106. Howell, J. B. L. 1975. Primary or idiopathic alveolar hypoventilation, pp. 819-820. In P. B. Beeson and W. McDermott, Eds. *Textbook of Medicine*. Vol I. 14th ed. Philadelphia: Saunders.
107. Huggett, C. 1984. Combustion conditions and exposure conditions for combustion product toxicity testing. *J. Fire Sci.* 2:328-347.
108. International Agency for Research on Cancer. 1982. *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Industrial Chemicals and Dye Stuffs*, pp. 93-148. Vol. 29. Lyon, France: IARC.
109. Jin, T. 1979. Human behavior in fire smoke experiments on emotional instability under increasing smoke density, pp. 323-339. In U.S. Japan Cooperative Program in Natural Resources, Fourth Joint Panel Meeting of the UJNR Panel on Fire Research and Safety, Tokyo, Japan, Feb. 5-9, 1979. *Proceedings*. Gaithersburg, Md.: National Bureau of Standards, Center for Fire Research.
110. Jones, W. H. 1985. A multicompartment model for the spread of fire, smoke, and toxic gases. *Fire Saf. J.* 9:55-79.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

111. Jones, W. W. 1983. A Review of Compartment Fire Models. National Bureau of Standards NBSIR 83-2684. Washington, D.C.: U.S. Department of Commerce. 38 pp.
112. Kane, L. E., C. S. Barrow, and Y. Alarie. 1979. A short-term test to predict acceptable levels of exposure to airborne sensory irritants. *Am. Ind. Hyg. Assoc. J.* 40:207-229.
113. Kaplan, H. L., A. F. Grand, and G. E. Hartzell. 1983. *Combustion Toxicology: Principles and Test Methods*. Lancaster, Pa.: Technomic Publishing Co. 174 pp.
114. Kaplan, H. L., A. F. Grand, W. R. Rogers, W. G. Switzer, and G. E. Hartzell. 1984. A Research Study of the Assessment of Escape Impairment by Irritant Combustion Gases in Postcrash Aircraft Fires. DOT/FAA/CT-84/16. Atlantic City, N.J.: U.S. Department of Transportation, FAA Technical Center. 65 pp.
115. Karter, M. J. 1985. Fire loss in the United States during 1984. *Fire* 79:14-76.
116. Kawagoe, K. 1958. *Fire Behaviour in Rooms*. Report of the Building Research Institute. Report No. 27. Tokyo: Building Research Institute. 72 pp.
117. Kimmerle, G. 1974. Aspects and methodology for the evaluation of toxicological parameters during fire exposure. *J. Fire Flammability Combust. Toxicol.* 1:4-51.
118. Kimmerle, G., and F. K. Prager. 1980. The relative toxicity of pyrolysis products. I. Plastics and man-made fibers. II. Polyisocyanate based foam materials. *J. Combust. Toxicol.* 7:42-68.
119. Kirkpatrick, M., and J. Bass. 1979. Severe obstructive lung disease after smoke inhalation. *Chest* 76:108-110.
120. Kishitani, K., and K. Nakamura. 1974. Toxicities of combustion products. *J. Fire Flammability Combust. Toxicol. Suppl.* 1:104-123.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

121. Kisko, T. M., and R. L. Francis. 1985. EVACNET+: A computer program to determine optimal building evacuation plans. *Fire Saf. J.* 9:211-220.
122. Klimisch, H. J., H. W. M. Hollander and J. Thyssen. 1980. Comparative measurements of the toxicity to laboratory animals of products of thermal decomposition generated by the method of DIN 53 436. *J. Combust. Toxicol.* 7:209-230.
123. Klimisch, H. J., H. W. M. Hollander, and J. Thyssen. 1980. Generation of constant concentrations of thermal decomposition products in inhalation chambers: A comparative study with a method according to DIN 53 436. *J. Combust. Toxicol.* 7:243-263.
124. Laties, V. G., and W. H. Merigan. 1979. Behavioral effects of carbon monoxide on animals and man. *Ann. Rev. Pharmacol. Toxicol.* 19:357-392.
125. Lawrence, W. H., R. R. Raje, A. R. Singh, and J. Autian. 1978. Toxicity of pyrolysis products: Influence of experimental conditions. The MSTL/UT and NASA/JSC procedures. *J. Combust. Toxicol.* 5:39-53.
126. Lawson, J., W. Walton, and W. Twilley. 1983. Fire performance of furnishings as measured in the NBS Furniture Calorimeter. Part I. National Bureau of Standards NBSIR 83-2787. Washington, D.C.: U.S. Department of Commerce.
127. Lee, T. G. 1972. Field Measurement of HCl Concentration from PVC Electrical Conduit Involved in Fire. National Bureau of Standards Project 4219229. NBS Rept. 10-870. Washington, D.C.: U.S. Department of Commerce. 17 pp.
128. LeQuesne P., A. Axford, C. McKerrow, and A. Jones. 1976. Neurological complications after a single severe exposure to toluene di-isocyanate. *Br. J. Ind. Med.* 33:72-78.
129. Levin, B. C., A. J. Fowell, M. M. Birky, M. Paabo, A. Stolte, and D. Malek. 1982. Further Development of a Test Method for the Assessment of the Acute

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

- Inhalation Toxicity of Combustion Products. NBSIR 82-2532. Washington, D.C.: U.S. Department of Commerce, National Bureau of Standards, Center for Fire Research. 133 pp.
130. Levin, B. C., M. Paabo, and M. M. Birky. 1983. An Interlaboratory Evaluation of the Version of the National Bureau of Standards Test Method for Assessing the Acute Inhalation Toxicity of Combustion Products. NBSIR 83-2678. Washington, D.C.: U.S. Department of Commerce, National Bureau of Standards, Center for Fire Research. 82 pp.
 131. Litchfield, J. T., Jr., and F. Wilcoxon. 1949. A simplified method of evaluating dose-effect experiments. *J. Pharmacol. Exper. Ther.* 96:99-113.
 132. Lloyd, E. L., and W. R. MacRae. 1971. Respiratory tract damage in burns: Case reports and review of the literature. *Br. J. Anaesth.* 43:365-379.
 133. Loftus, J. J. 1978. Back-Up Report for the Proposed Standard for the Flammability (Cigarette Ignition Resistance) of Upholstered Furniture. PFF 6-76. National Bureau of Standards NBSIR 78-1438. Washington, D.C.: U.S. Department of Commerce. 239 pp.
 134. Loke J., W. Farmer, R. Matthay, C. Putman, and G. Smith. 1980. Acute and chronic effects of fire fighting on pulmonary function. *Chest* 77:369-373.
 135. Loke, J., and R. A. Matthay. 1981. Managing victims of smoke inhalation. *J. Respir. Dis.* 2:87-98.
 136. Lowry, W. T., L. Juarez, C. S. Petty, and B. Roberts. 1985. Studies of toxic gas production during actual structural fires in the Dallas area. *J. Forens. Sci.* 30:59-72.
 137. Lowry, W. T., J. Peterson, C. S. Petty, and J. L. Badgett. 1985. Free radical production from controlled low-energy fires: Toxicity considerations. *J. Forens. Sci.* 30:73-85.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

138. Lucia, H. L., C. S. Barrow, M. F. Stock, and Y. Alarie. 1977. A semi-quantitative method for assessing anatomic damage sustained by the upper respiratory tract of the laboratory mouse, *Mus musculus*. *J. Combust. Toxicol.* 4:472-486.
139. Lucia, H. L., A. K. Burton, R. C. Anderson, M. F. Stock, and Y. C. Alarie. 1978. Renal damage in mice following exposure to the pyrolysis products of polytetrafluoroethylene. *J. Combust. Toxicol.* 5:270-277.
140. MacArthur, C. D., and J. R. Myers. 1978. Dayton Aircraft Cabin Fire Model Validation, Phase I. Produced by the University of Dayton Research Institute. Prepared for the U.S. Department of Transportation Federal Aviation Administration, Washington, D.C. 167 pp. (available from National Technical Information Service, Springfield, Va., as AD-A058 547)
141. Mastromatteo, E. 1959. Mortality in city firemen. I. A review. *AMA Arch. Ind. Health* 20:1-7.
142. Mastromatteo, E. 1959. Mortality in city firemen. II. A study of mortality in firemen of a city fire department. *AMA Arch. Ind. Health* 20:227-233.
143. McGuire, P. S., and Z. Annau. 1980. Behavioral effects of exposure to the combustion products of flexible polyurethane foam. *Neurobehav. Toxicol.* 2:355-362.
144. Milham, S., Jr. 1976. Occupational Mortality in Washington State 1950-1971. Vol. III. Division of Surveillance, Hazard Evaluations, and Field Studies. Contract CDC-99-74-26. NIOSH 76-175-C. Cincinnati, Ohio: National Institute for Occupational Safety and Health. 926 pp.
145. Milne, J. E. H. 1969. Nitrogen dioxide inhalation and bronchiolitis obliterans. *J. Occup. Med.* 11:538-547.
146. Mitchell, D. S., W. R. Rogers, W. R. Herrera, and W. G. Switzer. 1978. Behavioral incapacitation of rats during full-scale combustion of natural-fiber

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

- and synthetic polymeric furnishings. *Fire Res.* 1:187-197.
147. Mitler, H. 1985. The Harvard fire model. *Fire Saf. J.* 9:7-16.
148. Mitler, H. E., and H. W. Emmons. 1981. Documentation for CFC V, the Fifth Harvard Computer Fire Code. National Bureau of Standards NBS-GCR-81-344. Washington, D.C.: U.S. Department of Commerce. 189 pp. (available from National Technical Information Service, Springfield, Va., as PB82-139486)
149. Modern Plastics. 1960. Market for materials--1959. *Modern Plast.* 37:91-112,182-226.
150. Modern Plastics. 1980. Materials. 1980. *Modern Plast.* 57:71-108.
151. Modern Plastics. 1950. Materials supply and demand. *Modern Plast.* 27:57-70,129-140.
152. Modern Plastics. 1970. The materials and markets. *Modern Plast.* 47:64-102.
153. Moritz, A. R., F. C. Henriques, F. R. Dutra, and J. R. Weisiger. 1947. Studies of thermal injury. IV. An exploration of the casualty-producing attributes of conflagration; local and systemic effects of general cutaneous exposure to excessive circumambient (air) and circumradiant heat of varying duration and intensity. *Arch. Pathol.* 43:466-488.
154. Moritz, A. R., F. C. Henriques, and R. McLean. 1945. The effects of inhaled heat on the air passages and lungs: An experimental investigation. *Am. J. Pathol.* 21:311-331.
155. Moss, R. H., C. F. Jackson, and J. Seiberlich. 1951. Toxicity of carbon monoxide and hydrogen cyanide gas mixtures. *AMA Arch. Ind. Hyg. Occup. Med.* 4:53-64.
156. Musk, A. W., R. R. Monson, J. M. Peters, and R. K. Peters. 1978. Mortality among Boston fire fighters 1915-1975. *Br. J. Ind. Med.* 35:104-108.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

157. Musk, A. W., J. M. Peters, L. Bernstein, C. Rubin, and C. B. Monroe. 1982. Pulmonary function in firefighters: A six-year follow-up in the Boston Fire Department. *Am. J. Ind. Med.* 3:3-9.
158. Musk, A. W., J. M. Peters, and D. H. Wegman. 1977. Lung function in firefighters. I. A three year follow-up of active subjects. *Am. J. Public Health* 67:626-629.
159. Musk, A. W., T. J. Smith, J. M. Peters, and E. McLaughlin. 1979. Pulmonary function in firefighters: Acute changes in ventilatory capacity and their correlates. *Br. J. Ind. Med.* 36:29-34.
160. National Center for Health Statistics. 1950-1980. *Vital Statistics of the United States*. Hyattsville, Md.: U.S. Department of Health and Human Services.
161. National Institute for Occupational Safety and Health. 1977. *Criteria for a Recommended Standard: Occupational Exposure to Hydrogen Sulfide*. DHEW (NIOSH) Publ. 77-158. Washington, D.C.: U.S. Government Printing Office.
162. National Research Council, Committee on Fire Research. 1976. *Physiological and Toxicological Aspects of Combustion Products*. International Symposium held at the University of Utah. March 18-20, 1974. Washington, D.C.: National Academy of Sciences. 244 pp.
163. National Research Council, Committee on Fire Safety Aspects of Polymeric Materials. 1979. *Fire Safety Aspects of Polymeric Materials. Vol. 2. Test Methods, Specifications and Standards*. National Academy of Sciences Publ. NMAB 318-2. Westport, Conn.: Technomic.
164. National Research Council, Committee on Fire Toxicology. 1977. *Fire Toxicology: Methods for Evaluation of Toxicity of Pyrolysis and Combustion Products*. Report No. 2. Washington, D.C.: National Academy of Sciences. 34 pp.
165. National Research Council, Committee on Medical and Biologic Effects of Environmental Pollutants. 1979.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

- Hydrogen Sulfide. Baltimore, Md.: University Park Press.
166. National Research Council, Committee on Medical and Biologic Effects of Environmental Pollutants. 1977. Nitrogen Oxides. Washington, D.C.: National Academy of Sciences.
 167. National Research Council, Committee on Safety to Life from Fire in Elementary and Secondary Schools. 1960. School Fires: An Approach to Life Safety. Washington, D.C.: National Academy of Sciences. 59 pp.
 168. Oldenburger D., W. J. Maurer, E. S. Beltaos, and G. E. Magnin. 1972. Inhalation lipid pneumonia from burning fats: A newly recognized industrial hazard. *J. Am. Med. Assoc.* 222:1288-1289.
 169. O'Mara, M. M. 1974. The combustion products from synthetic and natural products. Part 1. *Wood. J. Fire Flammability* 5:34-53.
 170. Packham, S. C., R. B. Jeppsen, J. B. McCandless, T. L. Blank, and J. H. Petajan. 1978. The toxicological contribution of carbon monoxide as a component of wood smoke. *J. Combust. Toxicol.* 5:11-24.
 171. Parent, R. A., J. V. Dilley, S. B. Martin, and R. G. McKee. 1979. Acute toxicity in Fischer rats of smoke from non-flaming combustion of ten flexible polyurethane foams. *J. Combust. Toxicol.* 6:185-197.
 172. Parent, R. A., G. H. Y. Lin, G. T. Pryor, S. B. Martin, R. G. McKee, and J. V. Dilley. 1979. Behavioral toxicity in Fischer rats exposed to smoke from non-flaming combustion of ten flexible polyurethane foams. *J. Combust. Toxicol.* 6:215-227.
 173. Paul, I. 1984. Development of knowledge about means of egress. *Fire Technol.* 20:28-40.
 174. Paulsen, R. L. 1984. Human behavior and fires: An introduction. *Fire Technol.* 20:15-27.

175. Peabody, H. 1977. Pulmonary function and the firefighters. *J. Combust. Toxicol.* 4:8-15.
176. Perez-Guerra, F., R. E. Walsh, and S. S. Sagel. 1971. Bronchiolitis obliterans and tracheal stenosis: Late complications of inhalation burn. *J. Am. Med. Assoc.* 218:1568-1570.
177. Petajan, J. H., K. J. Voorhees, S. C. Packham, R. C. Baldwin, I. N. Einhorn, M. L. Grunnet, B. G. Dinger, and M. M. Birky. 1975. Extreme toxicity from combustion products of a fire-retarded polyurethane foam. *Science* 187:742-744.
178. Peters, J., G. Theriault, L. Fine, and D. Wegman. 1974. Chronic effect of fire fighting on pulmonary function. *N. Engl. J. Med.* 291:1320-1322.
179. Petersen, G. R., and S. Milham, Jr. 1980. Occupational Mortality in the State of California 1959-1961. DHEW (NIOSH) Publ. 80-104. Cincinnati, Ohio: National Institute for Occupational Safety and Health.
180. Phillips, A. W., and O. Cope. 1962. Burn therapy. II. The revelation of respiratory tract damage as a principal killer of the burned patient. *Ann. Surg.* 155:1-19.
181. Potts, W. J., and T. S. Lederer. 1978. Some limitations in the use of the sensory irritation method as an end-point in measurement of smoke toxicity. *J. Combust. Toxicol.* 5:182-195.
182. Products Research Committee. 1980. Fire Research on Cellular Plastics: The Final Report of the Products Research Committee. Washington, D.C.: Products Research Committee.
183. Pryor A. J., D. E. Johnson, and N. N. Jackson. 1969. Hazards of smoke and toxic gases produced in urban fires. *J. Fire Flammability/Combust. Toxicol. Suppl.* 2:64-112.
184. Pryor, G. T., J. V. Dilley, R. G. McKee, and S. B. Martin. 1979. Behavioral Techniques in Fire Toxicology. Presented at California Conference on Fire Toxicity, San Francisco, August 21-22.25 pp.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

185. Purser, D. A., and K. R. Berrill. 1983. Effects of carbon monoxide on behavior in monkeys in relation to human fire hazard. *Arch. Environ. Health* 38:308-315.
186. Purser, D., and P. Grimshaw. 1984. The incapacitative effects of exposure to thermal decomposition products of polyurethane foams. *Fire Mater.* 8:10-16.
187. Purser, D. A., P. Grimshaw, and K. R. Berrill. 1984. Intoxication by cyanide in fires: A study in monkeys using polyacrylonitrile. *Arch. Environ. Health* 39:394-400.
188. Purser, D., and W. Woolley. 1982. *Biological Studies of Combustion Atmosphere: Smoke and Toxic Gases from Burning Plastics*. A Conference Organized by QMC Industrial Research Ltd. and the Fire Research Station, January 6 and 7. London: QMC. 32 pp.
189. Quintiere, J. 1982. An Assessment of Correlations Between Laboratory and Full-Scale Experiments for the FAA Aircraft Fire Safety Program. Part 1. Smoke. NBSIR 82-2508. Washington, D.C.: National Bureau of Standards. 54 pp. (available from National Technical Information Service, Springfield, Va., as PB83-113522)
190. Quintiere, J. G., and M. Harkleroad. 1984. New concepts for measuring flame spread properties. National Bureau of Standards NBSIR 84-2943. Washington, D.C.: U.S. Department of Commerce. 144 pp.
191. Radford, E. P., B. P. Pitt, B. Halpin, Y. Caplan, R. Fisher, and P. Schweda. 1976. Study of fire deaths in Maryland. Sept. 1971-Jan. 1974, pp. 26-35. In *Physiological and Toxicological Aspects of Combustion Products*. Washington, D.C.: National Academy of Sciences.
192. Rasbash, D. J. 1966. Smoke and toxic gas. *Fire* 59:175-179.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

193. Reid, J. B., and T. F. Brecht. 1979. A unique system for quantitation of incapacitation time for rats exposed to products of combustion. *Toxicol. Appl. Pharmacol.* 48:A31. (abstract 62)
194. Rentoul, E., and H. Smith, Eds. 1973. Carbon monoxide, pp. 565-570. In *Glaister's Medical Jurisprudence and Toxicology*. 13th ed. London: Churchill Livingstone.
195. Rowe, W. D. 1977. *An Anatomy of Risk*. New York, N.Y.: John Wiley and Sons. 488 pp.
196. Russo, D. M., P. Sgro, and H. J. Schneider. 1981. Effects of polyurethane and polyimide thermal decomposition products on shock escape and avoidance behavior. *Neurobehav. Toxicol. Teratol.* 3:265-270.
197. Savolainen, H., R. Tenhunen, E. Elovaara, and A. Tossavainen. 1980. Cumulative biochemical effects of repeated subclinical hydrogen sulfide intoxication in mouse brain. *Int. Arch. Occup. Environ. Health* 46:87-92.
198. Schaenman, P. S., and E. F. Seits. 1982. *International Concepts in Fire Protection*. Arlington, Va.: Tri-Data. 63 pp.
199. Schulte, P. A. 1980. Technical Assistance Report. International Association of Fire Fighters, Alliance, Ohio. Hazard Evaluation and Technical Assistance Branch. DHEW (NIOSH) Publ. 79-50. Cincinnati, Ohio: National Institute for Occupational Safety and Health. 15 pp.
200. Sette, W. F., and Z. Anna. 1979. Effects of heat and carbon monoxide on continuous avoidance performance. *Toxicol. Appl. Pharmacol.* 46:A203. (abstract 407)
201. Sidman, M. 1953. Avoidance conditioning with brief shock and no exteroceptive warning signal. *Science* 118:157-158.
202. Siesjo, B. K., L. Berntman, and S. Rehncrona. 1979. Effect of hypoxia on blood flow and

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

- metabolic flux in the brain. *Adv. Neurol.* 26:267-283.
203. Silverstein, P., and D. P. Dressler. 1970. Effect of current therapy on burn mortality. *Ann. Surg.* 171:124-129.
204. Skornik, W. A., D. P. Dressler, and R. S. Robinson. 1976. The Relationship Between a Scale-Model Laboratory Bioassay Method for the Determination of Smoke Toxicity and a Full-Scale Fire Test. Sponsored by the Committee on Fire Research, National Academy of Sciences, and the Flammability Research Center of the University of Utah. International Symposium on Toxicity and Physiology of Combustion Products. March 22-26. University of Utah, Salt Lake City. 11 pp.
205. Smith, R. P. 1980. Toxic responses of the blood, pp. 311-331. In J. Doull, C. D. Klaassen, and M. O. Amdur, Eds. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 2nd ed. New York: Macmillan.
206. Snell, J. 1984. Summary: Preliminary report of the NFPA Advisory Committee on the Toxicity of the Products of Combustion. *Fire J.* 78:69-76.
207. Society of the Plastics Industry. 1984. *Facts and Figures of the U.S. Plastics Industry*. Prepared by H. Kibbel. New York: Society of the Plastics Industry, Inc. 125 pp.
208. Sparrow, D., R. Bosse, B. Rosner, and S. Weils. 1982. The effect of occupational exposure on pulmonary function: A longitudinal evaluation of firefighters and nonfirefighters. *Am. Rev. Respir. Dis.* 125:319-322.
209. Spurgeon, J. 1978. The correlation of animal response data with the yields of selected thermal decomposition products for typical aircraft interior materials. FAA-NA-78-45. Atlantic City, N.J.: National Aviation Facilities Experimental Center. 40 pp. (available from National Technical Information Service, Springfield, Va., as AD-A062-938/6)

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

210. Spurgeon, J. C., R. A. Filipczak, R. E. Feher, and S. J. Sternik. 1979. A procedure for electronically monitoring animal response parameters using the rotating wheel. *J. Combust. Toxicol.* 6:198-207.
211. Stahl, F. I. 1982. BFIREs-II: A behavior-based computer simulation of emergency egress during fires. *Fire Technol.* 18:49-65.
212. Stewart, R. D. 1975. The effect of carbon monoxide on humans. *Ann. Rev. Pharmacol.* 15:409-423.
213. Stokinger, H. E. 1981. The halogens and the nonmetals boron and silicon, pp. 2937-2954. In *Patty's Industrial Hygiene and Toxicology*. Vol. 2B. Toxicology. New York: Wiley.
214. Stoll, A. M., and L. C. Greene. 1959. Relationship between pain and tissue damage due to thermal radiation. *J. Appl. Physiol.* 14:373-382.
215. Tanaka, T. 1983. A Model of Multi Room Fire Spread. National Bureau of Standards NBSIR 83-2718. Washington, D.C.: U.S. Department of Commerce. 168 pp.
216. Tashkin, D., M. Genovesi, S. Chopra, A. Coulson, and M. Simmons. 1977. Respiratory status of Los Angeles firemen. One-month follow-up after inhalation of dense smoke. *Chest* 71:445-449.
217. Terrill, J.B., R. R. Montgomery, and C. F. Reinhardt. 1978. Toxic gases from fires. *Science* 200:1343-1347.
218. Tewarson, A., J. L. Lee, and R. F. Pion. 1981. The influence of oxygen concentration fuel parameters for fire modeling, pp. 563-570. In *Eighteenth International Symposium on Combustion*. Pittsburgh, Pa.: The Combustion Institute.
219. Tewarson, A., and J. S. Newman. 1985. Scale effects on fire properties of materials, pp. 451-462. In *Fire Safety Science. Proceedings of the First International Symposium*. October 7-11. New York: Hemisphere Publishing Corporation.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

220. Thompson, W. R., and C. S. Weil. 1952. On the construction of tables for moving average interpolation. *Biometrics* 8:51-54.
221. Unger, K. R. Snow, J. Mestas, and W. Miller. 1980. Smoke Inhalation in Firemen. *Thorax* 35:838-842.
222. U.S. Fire Administration. 1982. Fire in the United States. Executive Summary. Prepared for the Federal Emergency Management Agency. 5th ed. Emmitsburg, Md.: U.S. Fire Administration. 61 pp.
223. Vogel, F. S. 1979. The morphological consequences of cerebral hypoxia. *Adv. Neurol.* 26:147-154.
224. Wallace D. N. 1981. Dangers of polyvinyl chloride wire insulation decomposition. I. Long-term health impairments: Studies of firefighters of the 1975 New York Telephone fire and of survivors of the 1977 Beverly Hills Supper Club fire. *J. Combust. Toxicol.* 8:205-232.
225. Waters, M. D., D. E. Gardner, C. Aranyi, and D. L. Coffin. 1975. Metal toxicity for rabbit alveolar macrophages in vitro. *Environ. Res.* 9:32-47.
226. Whitener, D. R., L. M. Whitener, K. J. Robertson, C. R. Baxter, and A. K. Pierce. 1980. Pulmonary function measurements in patients with thermal injury and smoke inhalation. *Am. Rev. Respir. Dis.* 122:731-739.
227. Winter, P. M., and J. N. Miller. 1976. Carbon monoxide poisoning. *J. Am. Med. Assoc.* 236:1502-1504.
228. Wong, K. L., M. F. Stock, and Y. Alarie. 1983. Evaluation of the pulmonary toxicity of plasticized polyvinyl chloride thermal decomposition products in guinea pigs by repeated CO₂ challenges. *Toxicol. Appl. Pharmacol.* 70:236-248.
229. Wong, K. L., M. F. Stock, D. E. Malek, and Y. Alarie. 1984. Evaluation of pulmonary effects of wood smoke in guinea pigs by repeated CO₂ challenges. *Toxicol. Appl. Pharmacol.* 75:69-80.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

230. Zikria, B. A., D. C. Budd, F. Floch, and J. M. Ferrer. 1975. What is clinical smoke poisoning? *Ann. Surg.* 181:151-156.
231. Zikria, B. A., J. M. Ferrer, and H. F. Floch. 1972. The Chemical factors contributing to pulmonary damage in "smoke poisoning." *Surgery* 71:704-709.
232. Zikria, B. A., G. C. Weston, M. Chodoff, and J. M. Ferrer. 1972. Smoke and carbon monoxide poisoning in fire victims. *J. Trauma* 12:641-645.
233. Zukoski, E. E., and T. Kubota. 1980. Two-layer modeling of smoke movement in building fires. *Fire Mater.* 4:17-27.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.