Handbook of Environmental Genotoxicology

Volume I
Environmental Aspects

Editor
Eugene Sawicki, Ph.D.

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Director
Center for Environmental Genotoxic Studies
5033 Hermitage Drive
Raleigh, North Carolina

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THE EDITOR

Dr. Eugene Sawicki has had a widely varied experience in clinical chemistry, cancer research, and environmental analysis. He has received a B.S., magna cum laude in chemistry, and a M.S. in organic chemistry from the University of Cincinnati, and a Ph.D. in biochemical oncology from the University of Florida, Gainesville. He has spent 4 years in clinical chemistry, 4 years in cancer research, and 22 years in the EPA as one of the country's foremost pioneers in developing, evaluating, and applying methods of analysis for carcinogens, mutagens, allergens, and other pollutants in emission sources, industrial and outdoor atmospheres, and other ambient environments. He has directed or been in the forefront in the development and application of numerous analytical techniques (including thin-layer chromatography, high performance liquid chromatography, gas-liquid chromatography, electrophoresis, ion chromatography, mass spectrometry, ultraviolet, visible and infrared absorption spectrophotometry, spectrophotofluorimetry, and spectrophotophosphorimetry to the analysis of environmental pollutants. A large number of genotoxicant screening methods have been developed or utilized under his direction. He has been a Chairman of the Subcommittee on Hydrocarbons, Organic Airborne Particulates, and Industrial Carcinogens of the Intersociety Committee. Dr. Sawicki has been or is a member of the Editorial Advisory Boards of *Analytical Chemistry, Microchemical Journal, Analytical Letters, Environmental Analytical Chemistry, Toxicology and Environmental Reviews,* etc. He has been the author of over 200 scientific papers published in organic chemical, analytical, environmental, and medical journals and is the author of nine books. He has presented papers all over the world at chemical, analytical, environmental, biological, and medical symposia. He has been a consultant to the National Cancer Institute on their contracts concerned with environmental carcinogenesis. He was on the Cancer Hazards Ranking and Information System Advisory Committee and also contributed to the Carcinogen Metabolism and Toxicology Segment Advisory Group.

He has also consulted and contributed to several publications of the International Agency for Research against Cancer. He is a member of Phi Beta Kappa and has received government superior service awards in 1959 and 1960, the "Cincinnati Chemist of the Year Award" in 1968, the Detroit Anachem Award in 1968, the Benedetti-Pichler Award from the Microchemical Society in 1974, the U.S. Government Bronze Medal in 1978, and the Distinguished Career Award from the U.S. Government in 1979.

He has an intense interest in exploration, poetry, literature, photography, environmental pollution, environmental analytical chemistry, mutagenesis, carcinogenesis, chromosome aberrations, evolution, aging, and other aspects of genotoxicology.

TABLE OF CONTENTS

Volume I

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INTRODUCTION

The central themes around which this volume is constructed are the changes undergone by the double helical DNA molecule and its entities — the genes and the 46 human chromosomes — when attacked by environmental pollutants or their highly reactive metabolites. For such changes to take place the defenses of the mortal phenotype must be bypassed. These changes can be even more serious to the species when the phenotype's defenses of the germ cell DNA's attempt at continuing immortality are breached.

All life forms are related; they all have the same genetic code. In this genetic code of life there are only four letters (A,T,G,C) and all the words are three-letter words. The arrangement of "words" or genetic codes in linear sequence on an alternating polymeric background of 2-deoxyribose and phosphoric acid imparts to all living things their genetic potential. Essentially, DNA carries the organism's genetic information, which is then passed on to RNA during the synthesis of RNA from the DNA blueprint. The RNA then migrates to cellular sites and acts as the blueprint for the synthesis of the proteins, each triplet of RNA bases coding for each protein amino acid. It is these proteins which give form, shape, and function to organisms and, since their ultimate structure is encoded in the DNA, then any change in the DNA (or in the transfer of genetic information) will affect the form, shape, and function of the organism itself.

The infinite ways the code may be constructed gives to the four bases (A,T,G,C) the capability of producing all forms of life from viruses to humans. Since all life forms are related, many of the things that happen to the DNA, genes, and chromosomes of nonhuman life forms could happen to similar entities in humans. For this reason genotoxic effects on nonhuman life have also been considered in this volume from the model or extrapolative viewpoint.

The chemicals or radiations that can alter DNA, genes, or chromosomes are called genotoxicants. The wide variety of genotoxicants which have been postulated for many human physiological problems include aging factors, atherogens, behavior modifiers, carcinogens, cataractogens, clastogens, diabetogens, memory modifers, mutagens, teratogens, and turbogens. These genotoxicants and their gene-environmental interactions will be discussed in these volumes. For many of the physiological effects postulated as resulting from exposures to these materials epigenetic mechanisms have also been shown to operate or postulated to take place in addition to the genetic mechanism or sometimes as the predominant mechanism. These genotoxic effects have been called a Devil's book of genesis and would seem to include almost all serious human afflictions from infancy to old age and even evolution itself. Also of interest for purposes of understanding and prevention are the many types of cofactors and antifactors which enhance and inhibit, respectively, the physiological actions of the genotoxicants. These factors are discussed.

In respect to exposure or the potential gene-environment interaction, Steinbach, in one of his papers, has postulated that the attitude of man with regard to the environment is different from animals in at least two respects: (1) man in terms of human society keeps changing the environment humanity lives in and (2) as an individual, man can consciously choose to some extent his ecologic niche. This ecologic behavior is called ecopoiesis. The most important chronic diseases of modern man are the result of an inadequate ecopoiesis.

In the last 10 to 20 years there has been a remarkable growth in the production of a wide variety of chemicals. The production of key chemicals has increased drastically, e.g., nitric acid, 6.6, 12.9, and a predicted 25 billion lb in 1960, 1970, and 1980, respectively. The production of synthetic organic materials is now greater than 120 million tons (in 1970), and of this material 20 million tons are estimated to be released into the environment. Similarly, radiation sources have been and will be increasing at a rapid rate. People receive varying levels of exposure to many potential genotoxicants at home, at work, in the marketplace, in the streets, or during recreation. These exposures are often uncontrolled and most often undocumented. Essentially, these exposures can be considered to be industrial, iatrogenic, lifestyle, or transplacental. Because of their importance to genotoxicity, production and exposure are discussed in the present volumes.

In respect to the control of environmental pollution, the major industries now have departments concerned with the solution of environmental problems. On the other hand, with the cutback in government regulations in the 1980s, much more data on the genotoxic effects of chemicals and radiation on humans will probably be developed. Systems will need to be perfected to collect the massive amounts of human data that are and will be accumulating to a probably accelerating extent in the future. This increasing exposure would also seem to be indicated on the basis of Edelman's postulate wherein regulatory agencies have a common life cycle starting with the enforcement of whatever the agency is regulating for the common good to the last phase of staffing the agency with administrators drawn from the ranks of the regulated industries. In this respect the future direction of OSHA, NIOSH, and EPA will be of great interest.

Another argument for the belief that human experimental genotoxic data will be accumulating at a fairly rapid rate has been advanced by Hardin in his discussion of the tragedy of the commons. This tragedy is defined as a remorseless working of things, i.e., public property is plundered by the group in power; public highways are littered with garbage, paper, metal, bottles and cans; industrial waste is dumped into the air, water, and soil; and radioactive waste is stored on public land. Hardin believes that "Ruin is the destination toward which all men rush, each pursuing his own best interest in a society that believes in the freedom of the commons," while Crowe states that "the best answer to the question of who watches over the custodians of the commons is the regulated interests that make incursions on the commons." Iatrogenic problems derived from adverse use of the medical commons have been briefly discussed by Hiatt and will also be covered in this volume. Another reason for the continuation of many exposures has been given by Levin. He states that many of the known causes of cancer in man do involve some particular industrial and economic interest which is often highly effective in attacking the significance of the observations or in blocking their fullest application. Numerous systems which can be used in investigating this ongoing human experimentation are available and are discussed in our volumes.

The conclusion seems to be inescapable. It would seem that there are and will be many human exposures to chemicals and radiations, the collected data of which could be very useful in genotoxic studies especially since extrapolation from animal to man is not necessary. Some of this material will be discussed in these volumes. But somehow we should do a better job of collecting the forthcoming data and make better use of it in preventing and solving our cancer and other genotoxic problems.

The primary question in all genotoxic studies is the identity of the various key factors in the human environment and the human body which play important roles in human carcinogenesis and mutagenesis. For purposes of prevention of genotoxic effects the first question that needs to be answered is what numbers do we collect, and specifically, what individual pollutants, pollutant families, and mixtures do we measure and bioassay. This is important, for as Miller has stated, the generation of information which is not needed and not used raises the bill without contributing significantly to the value of the service. These are problems which are considered in these volumes.

Cancer has risen from the eighth most important cause of death in the U.S. in 1900 to the number two cause of death in 1972, second only to diseases of the heart. Lung cancer is now epidemic; the mortality rate for men went up 1400% in the past 40 years and it was still rising for both women and men in 1974. About 53 million Americans now living (one in four persons) will eventually have cancer. Cancer will be experienced by two of three families. Four out of five Americans who get cancer will die of it sooner or later no matter how early the diagnosis or how vigorous the treatment. More than half of all cancer deaths occur in persons over 65, more than three quarters over 50. Cancer, particularly leukemia, is the largest disease killer of children between the ages of 1 and 15 years. Cancer is the leading cause of death among women between the ages of 30 and 54, many of them mothers. After accidents, cancer racks up the highest mortality rate in people under 35. A large majority of American males will get cancer of the prostate if they live into their eighties.

The main interest is not whether cancer is decreasing or increasing, but in improving the quality of life over a longer period of a person's lifetime. If we believe in the improvement of the quality of life, especially among the aged, we cannot evade a much more thorough investigation of human carcinogenesis because of the awesome magnitude of two facts: (1) about one out of four persons living today will die of malignant tumors and (2) about 69% of people who will die after age 65 will die with cancer. These are the reasons so much emphasis is placed on cancer and carcinogenesis in these volumes.

A tremendous amount of important and interesting information has been accumulated from cancer research and genotoxic studies of the past 40 years. And yet Greenberg has stated that "cancer survival rates have shown little improvement over the past two decades or so, and that the frequent claims of markedly improved survival rates ignore or blur the fact that most of the changes occurred before 1950, and can probably be attributed to lower mortality from operations." In addition, Dao has been quoted by Greenberg as stating that breast cancer mortality has not changed in the last 70 years despite improved surgical techniques, sophisticated radiotherapies, and massive chemotherapeutic treatments. However, some cancers have been successfully treated. But where the treatment has been the most successful, we may have our most interesting genotoxic developments. Thus treatment of cancer patients with massive amounts of carcinomutagens means that DNA-containing entities present in the body, such as bacteria, viruses, fungae, parasitic organisms, mitochondria, etc. are probably mutated on a grand scale. This is one of the reasons why, in addition to secondary cancer effects on the cancer patients, these volumes also consider the various genotoxic effects of these cancer therapeutic agents on nonhuman life forms.

We will discuss various aspects of the three types of exposure (lifestyle, industrial, and iatrogenic) which are so complex, pervasive, intertwined, and so shallowly investigated that it is difficult to delineate any one of these as the most important factor of environmental carcinogenesis. The belief is that the elimination of the two major causes of death, cancer and cardiovascular disease, would add only about 10 years of additional life expectancy to Americans. This is another reason why these volumes will consider the action of the various genotoxicities on the quality and longevity of aging, for surely there are many other factors that affect life expectancy besides the two major causes of death.

Other facets of our gene-environment interaction problems to be discussed will be somatic and germ cell mutagenesis and the bioassays for the various types of genotoxicants and their cofactors and antifactors. It has been reported that 25% of our health burden is of genetic origin and that this genetic legacy is growing and that prenatal elimination may be the rule rather than the exception and that malformation may be the norm rather than the exception. It is estimated that in England and Wales married women aged 20 to 29 may abort 78% of their conceptions. It would seem that genetic mutations resulting in life-threatening abnormalities are not rare but are very common. It would appear that knowledge of the mutagen content of our environment is more than highly desirable; it is absolutely necessary.

We will discuss the effect of various portals of entry of carcinogens on organotropy, the utilization of comparative genotoxicology in extrapolation, and the effect of various environmental genotoxicants on high-risk groups. Other factors we have considered to be of special importance are the estimations of human environmental risk for cancer, mutation and other genotoxicities, and the two main problems in all genotoxic studies as applied to humans, the total body burden and the explosive growth of knowledge.

Much of the material in these volumes should be read with the understanding that there is a total body burden with many unknown factors involved in the genotoxic problem. For example, there seems to be many difficulties with our perspective in the study of human carcinogenesis because the sparsity of our environmental data and the simplicity of our carcinogenesis model of purebred animal and pure chemical (from which we have obtained a large amount of useful knowledge) so clouds our horizon so as to obscure the real world of diverse human beings and widely varied types of exposures to materials, radiations, and tissue damages. According to Lee, the detection and measurement of chemicals in the environment, their movement through environmental compartments, their physicochemical behavior at the portal of entry, their pharmacological fate within the body, and the resultant physiological disturbances lie within the purview of different types of specialist. The specialists are interested mainly in their own field of work, so that the data obtained from these various disciplines is poorly coordinated. This problem is exacerbated through the scattering of responsibility for the regulation and investigation of environmental carcinogens throughout many government agencies, e.g., in regulation, the Environmental Protection Agency, the Nuclear Regulatory Commission, the Food and Drug Administration, and the Occupational Safety and Health Administration with other agencies playing some role in regulation ranging from the Army Corps of Engineers to the Department of Transportation. In investigation we have EPA, FDA, NIOSH, NCI, NIEHS, etc.

It must be emphasized just as a thorough analysis of the hazardous environment is vital so is knowledge about the total environment. Most mutagens and carcinogens and their co- and antifactors are not exclusively residents in one type of environment, but can be found in varying quantities in air, water, soil, food, and medicinal, industrial, and consumer products. The multiplicity of types of exposures and the resultant body burden acquired from all portals of entry make it difficult for the media-oriented authorities to consider the total exposure of an individual to a given carcinogen and its co- and antifactors, a consideration necessary for understanding the various processes of mutagenesis and carcinogenesis. Consequently in these volumes we have attempted to bring together these specialized studies of the various investigative fields in a coordinated manner so as to make the material more useful to the reader.

The second major problem in organizing this material is the explosive growth of knowledge in the various fields of environmental genotoxicology, as well as in allied fields necessary to the study, understanding, and solution of these problems. This explosive growth has made it difficult for any single individual specializing in one of these fields to keep up with the advances in his field, never mind in allied fields. The difficulties in handling and utilizing this knowledge stems from our inability to read, assimilate, integrate, and remember all relevant technical literature for prompt, effective use. Even with the shallow help of bibliographies, the professional finds that he (or she) can no longer keep up with the literature of his profession and also do his everyday work. The most promising solution for the individual research worker, if he is to maintain an adequate knowledge of advances in his area (and allied areas), is continued specialization with the use of responsible, coordinated surveys of background information and advances in his field and allied fields. These volumes attempt to do this in environmental genotoxicology, in which field there is an intense interest by a wide variety of specialists.

I have attempted to cover the literature as best I could. I am sure some important papers have been omitted; I can only plead the excuse of the overwhelming vastness of the fields and the necessity to choose from a vast richness of material from many fields of endeavor. I am indebted to a large number of researchers and reviewers whose work I have covered in this volume. I have attempted to use the phraseology of the originators of the many thoughts, ideas, and facts in this volume. It was a humbling experience assembling the data in this book to realize that one's own contributions are so insignificant compared to the extremely interesting contributions of the large number of researchers who have made this book possible. Credit for the data and ideas in any section of this volume should be given to the authors in the references to that section.

This book can be considered as a concise summary of significant past contributions in environmental genotoxicology arranged alphabetically, a useful guide for environmental genotoxicity, and a stimulus to the application through analogy of the data and ideas in one field to entirely different fields of endeavor in environmental research. Parochial jargon promotes communication among the specialists, but does nothing to enhance the understanding of the general public. And yet, we're all specialists in one or a few limited subjects and nonspecialists in a vast number of disciplines. The present dictionary type of handbook has the best of all possible worlds since it uses a variety of jargons but also explains them. In this sense, the following Table of Acronyms is a good beginning in understanding the many jargons in this volume.

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TABLE OF ACRONYMS

A = Adenine
A = Anthracene
AAA = Acetazolamide
4AAABi = *N*-Acetoxy-4-acetylaminobiphenyl
2AAAF = *N*-Acetoxy-2-acetylaminofluorene
AAAFF = *N*-Acetoxy-*N*-acetyl-2-amino-7-fluorofluorene
AAAIF = *N*-Acetoxy-*N*-acetyl-2-amino-7-iodofluorene
2AAAPh = *N*-Acetoxy-2-acetylaminophenanthrene
4AABi = 4-Acetylaminobiphenyl
4AAAS = *N*-Acetoxy-4-acetylaminostilbene
4AABi = 4-Acetylaminobiphenyl
2AAF = 2-Acetylaminofluorene
4AAS = 4-Acetylaminostilbene
AB = Azobenzene
ABVD = Adriamycin, bleomycin, vinblastine, and DTIC
AC = Adenomatous coli
AcO = Acetoxy
Ac = Acetyl
Acr = Acridine
ACR = Adenomatosis of the colon and rectum
ActD = Actinomycin D
ACTH = Adrenocorticotropic hormone
AD = Adriamycin
ADA = Adenosine deaminase
AdH = Adenomatous hyperplasia
ADP = Adenosine diphosphate
AdV = Adenovirus
AE = *S*-Adenosylethionine
AEEN = *N*-α-Acetoxyethyl-*N*-ethylnitrosamine
AEL = Acute erythroleukemia
AETT = 6-Acetyl-7-ethyl-1,1,4,4-tetramethyltetralin
2AF = 2-Aminofluorene
AF^+ = Acriflavine
4AFABi = *N*-Acetyl-4′-fluoro-4-aminobiphenyl
AFB_1 = Aflatoxin B_1
AFB_2 = 2,3-DihydroAFB_1
AFB_{2a} = 2-HydroxyAFB_2
AFH_1 = 9-Hydroxyaflatoxicol = Aflatoxin H_1
AFL = Aflatoxicol
$AFLM_1$ = 4-Hydroxyaflatoxicol = Aflatoxicol M_1 = Aflatoxin LM_1
AFG_1 = Aflatoxin G_1
AFM_1 = 4-HydroxyAFB_1 = Aflatoxin M_1
AFP_1 = DemethylAFB_1 = Aflatoxin P_1
AFQ_1 = Aflatoxin Q_1 = 9-HydroxyAFB_1
AGL = Acute granulocytic leukemia; see AML
AH = Acetylhydrazine
2AHAF = *N*-acetyl-*N*-hydroxy-2-aminofluorene
4AHAS = *N*-Acetyl-*N*-hydroxy-4-aminostilbene
AHC = Acute hemorrhagic cystitis
AHH = Aryl hydrocarbon hydroxylase

AK = Acetylkynurenine
AL = Acute leukemia
ALL = Acute lymphoblastic leukemia
ALM = Acral lentiginous melanoma
ALP = Abetalipoproteinemia
AM = *S*-Adenosylmethionine
AMAAB = *N*-Acetoxy-*N*-methyl-4-aminoazobenzene
AMAF = *N*-Acetoxy-*N*-myristoyl-2-aminofluorene
AMD = *S*-Adenosylmethionine decarboxylase
AMEN = *N*-Acetoxymethyl-*N*-ethylnitrosamine
AML = Acute myelocytic leukemia
AMML = Acute myelomonocytic leukemia
AMNU = *N*′-Acetyl-*N*-methyl-*N*-nitrosourea
AMoL = Acute monocytic leukemia
5′-AMP = 5′-Adenosine-monophosphate
AMPH = *N*′-Acetyl-4-(hydroxymethyl)phenylhydrazine
AN = Acrylonitrile
ANLL = Acute nonlymphocytic leukemia
Ant = Anthanthrene
AO = Acridine orange
AOB = Azoxybenzene
3AP = 3-Acetoxypurine
APH = 1-Acetyl-2-phenylhydrazine
APiH = 1-Acetyl-2-picolinoylhydrazine
APL = Acute promyelocytic leukemia
APM = Acute postpartum mastitis
1APPN = *N*-(1-Acetoxypropyl)-*N*-1-propylnitrosamine
APUD = (A = amines, PU = precursor uptake, D = L-aromatic amino acid decarboxylase)
APL = Acute promyelocytic leukemia
1′AS = 1′-Acetoxysafrole
4AS = 4-Aminostilbene
ASA = Acetylsalicylic acid
1′ASO = 1′-Acetoxysafrole-2′,3′-oxide
ASBP = Age-specific biological parameter
AT = Ataxia telangiectasia
ATP = Adenosine triphosphate
ATT = Acute thermic trauma
AUC = Adenocarcinoma of the uterine cervix
AUL = Acute undifferentiated leukemia
AV = Acne vulgaris
3AX = 3-Acetoxyxanthine
AY = Acridine yellow
B = Benzene
BaA = Benz(*a*)anthracene
BaAcr = Benz(*a*)acridine
BaC = Benz(*a*)chrysene or Picene (preferred)
BaCar = 11H-Benzo(*a*)carbazole
BaF = 11H-Benzo(*a*)fluorene
BaP = Benzo(*a*)pyrene
BbC = Benzo(*b*)chrysene
BbCar = 6H-Benzo(*b*)carbazole

BbF	= 11H-Benzo(*b*)fluorene
BbFT	= Benzo(*b*)fluoranthene
BcAcr	= Benz(*c*)acridine
BcCar	= 7H-Benzo(*c*)carbazole
BcCin	= Benzo(*c*)cinnoline
B-cells	= Bone marrow-derived lymphoid cells
BcF	= 7H-Benzo(*c*)fluorene
BCME	= Bis(chloromethyl)ether
BCNU	= 1,3-Bis-(2-chloroethyl)-1-nitrosourea
BcPH	= Benzo(*c*)phenanthrene
BdefDT	= Benzo(*def*)dibenzothiophene
BeAP	= Benz(*e*)acephenanthrylene or Benzo(*b*)fluoranthene
BeP	= Benzo(*e*)pyrene
BfQ	= Benzo(*f*)quinoline
BghiPer	= Benzo(*ghi*)perylene
BgQ	= Benzo(*g*)quinoline
BghiFt	= Benzo(*ghi*)fluoranthene
BghiPer	= Benzo(*ghi*)perylene
BHA	= 2-(+ 3-) *tert*-Butyl-4-methoxyphenol
BhQ	= Benzo(*h*)quinoline
BHT	= 2,6-Di-*tert*-butyl-4-methylphenol
Bi	= Biphenyl
BjFt	= Benzo(*j*)fluoranthene
BkFt	= Benzo(*k*)fluoranthene
BO	= 7H-Benz(*de*)anthracen-7-one or Benzanthrone
BrstPep	= Benzo(*rst*)pentaphene
BUdR	= 5-Bromo-2′-deoxyuridine
C	= Chrysene
C	= Cytosine
CA	= Chromotropic acid
cAMP	= Adenosine 3′,5′-cyclic monophosphate
Car	= Carbazole
CBG	= C-bands by barium hydroxide using Giemsa
CC	= Column chromatography
CCNU	= 1-(2-Chloroethyl)-3-cyclohexyl-1-nitrosourea
CGL	= Chronic granulocytic leukemia
cGMP	= Guanosine 3′,5′-cyclic monophosphate
Ch	= Cholanthrene
CHO	= Chinese hamster ovary
CIMS	= Chemical ionization mass spectrometry
CML	= Chronic myelogenous leukemia
CMME	= Chloromethyl methyl ether
CMML	= Chronic myelomonocytic leukemia
CMV	= Cytomegalovirus
CNS	= Central nervous system
CoC	= Colon cancer
Cor	= Coronene
CpdefPh	= 4H-Cyclopenta(*def*)phenanthrene
CPecdP	= Cyclopenteno(*cd*)pyrene
CT	= Chemotherapy
Cyclic AMP	= Adenosine 3′,5′-cyclic monophosphoric acid
Cyclic GMP	= Guanosine 3′,5′-cyclic monophosphoric acid

DAAB	= 4-Dimethylaminoazobenzene
DAB	= p-Dimethylaminobenzaldehyde
DAC	= *p*-Dimethylaminocinnamaldehyde
DBacA	= Dibenz(*a,c*)anthracene
DBacAcr	= Dibenz(*a,c*)acridine
DBahA	= Dibenzo(*a,h*)anthracene
DBahAcr	= Dibenz(*a,h*)acridine
DBajA	= Dibenz(*a,j*)anthracene
DBajAcr	= Dibenz(*a,j*)acridine
DBchAcr	= Dibenz(*c,h*)acridine
DBCP	= 1,2-Dibromo-3-chloropropane
DBdef-pC	= Dibenzo(*def-p*)chrysene
DBf	= Dibenzofuran
DBN	= Di-*n*-butylnitrosamine
DBt	= Dibenzothiophene
DD	= Dibenzo-*p*-dioxin
p,p'-DDD	= 1,1-Dichloro-2,2-bis(*p*-chlorophenyl)ethane
DDE	= 1,1-Dichloro-2,2-bis(*p*-chlorophenyl)ethylene
DDP	= *cis*-dichlorodiammineplatinum (II)
o,p'-DDT	= 1,1,1-Trichloro-2-(*p*-chlorophenyl)-2- (*p*-chlorophenyl)ethane
p,p'-DDT	= 1,1,1-Trichloro-2,2-bis(*p*-chlorophenyl)ethane
DENA	= Diethynitrosamine or *N*-Nitrosodiethylamine
DEO	= 1,2,7,8-Diepoxyoctane
DES	= Diethylstilbestrol
DGA	= *N*-Diazoacetylglycine amide
DHNT	= 3-Di(hydroxymethyl)amino-6-(5-nitro-2-furylethenyl)-1,2,4-triazine
7,12-DiMeBaA	= DMBA = 7,12-Dimethylbenz(*a*)anthracene
DM	= Daunamycin
DMB	= *p*-Dimethylaminobenzaldehyde
DMBA	= 7,12-DiMeBaA = 7,12-Dimethylbenz(*a*)anthracene
DMC	= p-Dimethylaminocinnamaldehyde
1,2-DMH	= 1,2-Dimethylhydrazine
DMNA	= Dimethylnitrosoamine or *N*-Nitrosodimethylamine
D/M/Y	= Deaths per million per year
DNA	= Deoxyribonucleic acid
DNPP	= Di(*N*-nitroso)perhydropyrimidine
DOC	= Dissolved organic carbon
DON	= 6-Diazo-5-oxo-L-norleucine
L-Dopa	= L-3,4-Dihydroxyphenylalanine
DS	= Down's syndrome
dThd	= Thymidine
EBV	= Epstein-Barr virus
ec	= Electron capture
EDB	= Ethylene dibromide
EH	= Epoxide hydrase
EL	= Erythroleukemia
EMS	= Ethyl methanesulfonate
EMTD	= Estimated maximum tolerated dose
EPA	= Environmental Protection Agency
F	= Fluorene
FA	= Fanconi's anemia

FANFT	= *N*-[4-(5-Nitro-2-furyl)-2-thiazolyl]formamide
FCPS	= Familial colorectal polyposis syndrome
FDA	= Federal Drug Administration
FID	= Flame ionization detector
FPC	= Familial polyposis of colon
Ft	= Fluoranthene
FUdR	= 5-Fluorodeoxyuridine
G	= Guanine
GC	= Gas chromatography
GC-MS	= Gas chromatography coupled to mass spectrometry
GC-MS-COMP	= Computerized gas chromatography-mass spectrometry
GLC	= Gas liquid chromatography
GPC	= Gel permeation chromatography
G6PD	= Glucose-6-phosphate dehydrogenase
GSC	= Gas solid chromatography
GST	= Glutathione *S*-transferase
Hb	= Hemoglobin
HBV	= Hepatitis B virus
HCB	= Hexachlorobenzene
$(^3H)dT$	= Tritium-labeled thymidine
HFBA	= Heptafluorobutyric anhydride
HGPRT	= Hypoxanthine-guanine phosphoribosyltransferase
HN2	= Methyl-bis-(2-chloroethyl)amine
HPLC	= High performance liquid chromatography
HR	= 4-Hexylresorcinol
HSV-2	= Herpes simplex virus type 2
$(^3H)TdR$	= Tritiated thymidine
I	= Indole
i	= Isochromosome
IC	= Ion chromatography
ICR-191	= 2-Methoxy-6-chloro-9-[3-(2-chloroethyl)aminopropylamino]-acridine·2HCl
i.m.	= Intramuscular
IND1,2-bQ	= 11H-Indeno(1,2-*b*)quinoline
Ind1,2,3-cdFt	= Indeno(1,2,3-*cd*)fluoranthene
Ind1,2,3-cdP	= Indeno(1,2,3-*cd*)pyrene
Ind1,2,3-ijIq	= Indeno(1,2,3-*ij*)isoquinoline
INH	= Isonicotinyl hydrazide
i.p.	= Intraperitoneal
IUdR	= 5-Iododeoxyuridine
IVCT	= In vitro cell transformation
LC	= Liquid chromatography
LD	= Lethal dose
LD_{50}	= Median lethal dose
LET	= Linear energy transfer
LSD-25	= Lysergic acid diethylamide
LTSPF	= Low temperature spectrophotofluorescence
m	= Meter
MAKA	= Major karyotypic abnormalities
MAM	= Methylazoxymethanol
MBC	= Methyl 2-benzimidzolylcarbamate
MBTH	= 3-Methyl-2-benzothiazolinone hydrazone