

Table 2 Continued

Humans				Animals		
Chemical or industrial process	Main type of exposure ^a	Target organ	Main route of exposure ^b	Animal	Target organ	Route of exposure
13 Cyclophosphamide	Medicinal	Bladder	p.o., injection	Mouse Rat	Hemopoietic system, lung Various sites Bladder ^c Mammary gland Various sites	i.p. s.c. injection p.o. i.p. i.p. i.v.
14 Diethylstilbestrol	Medicinal	Uterus, vagina	p.o.	Mouse Mouse Rat Hamster Squirrel monkey	Mammary Mammary, lymphoreticular testis vagina Mammary, hypophysis ^d bladder Kidney Uterine serosa	p.o. s.c. injection s.c. implantation Local s.c. implantation s.c. injection, s.c. implantation s.c. implantation
15 Hematite mining (? radon)	Occupational	Lung	Inhalation	Mouse, hamster, guinea pig Rat	Negative Negative	Inhalation i.p. s.c. injection
16 Isopropyl oils	Occupational ^a	Nasal cavity, larynx	Inhalation	No adequate tests		
17 Melphalan	Medicinal	Hemopoietic system	p.o., injection	Mouse Rat	Initiator Lung, lymphosarcomas Local	Skin i.p. i.p.
18 Mustard gas	Occupational	Lung, larynx	Inhalation	Mouse	Lung Local mammary	Inhalation i.p. s.c. injection
19 2-Naphthylamine	Occupational ^a	Bladder	Inhalation, skin, p.o.	Hamster, monkey Mouse Rat, rabbit	dog, Bladder Liver, lung Inadequate	p.o. s.c. injection p.o.
20 Nickel (nickel refining)	Occupational	Nasal cavity, lung	Inhalation	Rat Mouse, rat Mouse, rat Mouse, rat	hamster Lung Local Local	Inhalation s.c. i.m. injection i.m. implantation
21 N,N-Bis(2-chloroethyl)-2-naphthylamine	Medicinal	Bladder	p.o.	Mouse Rat	Lung Local	i.p. s.c. injection
22 Oxymetholone	Medicinal	Liver	p.o.	No adequate tests		
23 Phenacetin	Medicinal	Kidney	p.o.	No adequate tests ^d		
24 Phenytoin	Medicinal	Lymphoreticular tissues	p.o., injection	Mouse	Lymphoreticular tissues	p.o., i.p.
25 Soot, tars, and oils	Occupational, environmental	Lung, skin (scrotum)	Inhalation, skin	Mouse rabbit	Skin	Topical
26 Vinyl chloride	Occupational	Liver, lung ^e	Inhalation, skin	Mouse, rat	Lung liver, blood vessels mammary, Zymbal gland, kidney	Inhalation

^a The main types of exposures mentioned are those by which the association has been demonstrated, exposures other than the mentioned may also occur

^b The main routes of exposure given may not be the only ones by which such effects could occur

^c Indicative evidence

^d The induction of tumors of the nasal cavities in rats given phenacetin has been reported recently (S. Odashima, personal communication, 1977)

nary tumors in rats. The causative agent in hemalite mining has not been identified, and the role of radon is suspected. Ferric oxide was not carcinogenic in mice, guinea pigs, or hamsters when given by inhalation or intratracheally.

Correlations between human and animal data should be somewhat easier for the remaining 21 compounds, because in most of these cases a single identified chemical is involved. One notable exception, however, is arsenic compounds. Occupational exposure or exposure of the general population to drinking water containing high levels of arsenic or to arsenic-containing drugs is associated with the occurrence of skin cancer, while lung cancer is also associated with occupational exposure. No definite carcinogenic effect has been observed in the available results from experimental studies.

For 5 of the remaining 20 compounds, namely, benzene, chloramphenicol, isopropyl oil, oxymetholone, and phenacetin, no adequate carcinogenicity tests were available for evaluation at the time that the monographs were prepared. However, recent unpublished results indicate that phenacetin is carcinogenic in rats (S. Odashima, personal communication, 1977). The other 15 compounds were carcinogenic in at least one, but mostly in more than one, animal species.

Comparison between target organs in humans and animals confirms that the carcinogens have, in some cases, the same organ specificity in humans as that in experimental animals. In most cases, the chemicals were tested in more than one animal species, but the similarity in organ specificity may be limited to only one of the animal species tested. The chances for similar target organs are apparently greater if similar routes of exposure are used (Table 2). However, chemicals that were found to be carcinogenic in both humans and animals were carcinogenic in the latter also when administered by routes different from the one by which humans are exposed. The target organs in animals are more often multiple than in humans.

The evaluation of the carcinogenic risk to humans of these 26 chemicals or processes was based on evidence provided by epidemiological studies or case reports, and experimental evidence of carcinogenicity was not used in the formulation of the evaluation. For several chemicals (aflatoxin, 4-aminobiphenyl, bis(chloromethyl) ether, diethylstilbestrol, melphalan, mustard gas, and vinyl chloride), experimental evidence of carcinogenicity preceded human evidence and a similar effect in humans could have been predicted. The case of bis(chloromethyl) ether is slightly different from the others. The first observations in humans, indicating an unusual clustering of lung cancer in occupationally exposed workers, were made as early as 1962 (45, 46). Evidence of a carcinogenic effect in animals was first published in 1968 and preceded the publication of the first 2 epidemiological studies, which appeared in 1973 (25).

Chemicals Carcinogenic to Experimental Animals. While in 1968 it was considered that lists of carcinogens should not be prepared without an in-depth evaluation of the available evidence, which differs both quantitatively and qualitatively from compound to compound, an attempt is now made to separate chemicals evaluated in the first 16 volumes of the IARC monographs into those for which some evidence of carcinogenicity in experimental animals

was found and those for which the data were judged inadequate for evaluation.

Of the 368 chemicals evaluated in Volumes 1 to 16 of the monographs, the 26 associated with cancer induction in humans have been referred to previously. For 221 of the remaining 342 (Table 3), some evidence of carcinogenicity in 1 or more animal species was found. It should be stressed that for these chemicals (marked with an asterisk) no attempt was made to distinguish between those for which "strong evidence" and "weak evidence" of carcinogenicity, following the criteria discussed in the last part of this paper, existed or those for which additional confirmatory carcinogenicity studies are needed. For the majority of these chemicals, evidence of human exposure exists but no evaluation of the carcinogenic risk to humans was made, either because no epidemiological studies or case reports were available (205 compounds) or because the results were inconclusive. (Some case reports were available for 7 compounds: BHC/lindane, carbon tetrachloride, chlorambucil, dimethyl sulfate, iron dextran, tris(aziridinyl)-p-benzoquinone and tris(1-aziridinyl)phosphine sulfide. Results of some epidemiological studies were available for 9 compounds: amitrole, beryllium compounds, DDT, dieldrin, dimethylcarbamoyl chloride, isoniazid, lead salts, phenobarbital, *N*-phenyl-2-naphthylamine.)

For the remaining 121 chemicals, the available studies were inadequate to make an evaluation of the presence or absence of a carcinogenic effect in animals or humans.

Conclusions and Discussion

For 26 chemicals (or industrial processes) of the 368 evaluated in the first 16 volumes of the IARC monographs, an evaluation was made of the positive association between exposure and occurrence of cancer in humans (Table 2). In these cases, the presence or absence of evidence of carcinogenicity in test animals was not used in the formulation of the evaluations as is shown, for instance, for benzene and arsenic compounds. One important lesson from these human carcinogens is that experimental evidence has in some cases preceded human evidence, and a similar effect in humans might have been predicted.

For 221 of the 368 chemicals evaluated in the 16 monograph volumes, some evidence of carcinogenicity was found in at least 1 experimental animal species (Table 3). Humans are or can be exposed to the majority of these chemicals. In some instances, results of epidemiological studies or case reports were available but provided insufficient evidence on which to base an evaluation. In most instances, however, epidemiological studies or case reports were not available (the general scarcity of epidemiological studies noted throughout the monograph program must be stressed). In no case was an attempt made to interpret animal data directly in terms of human risk or in terms of the degree of carcinogenicity evidence provided by the experimental data. However, a large number of food additives, pesticides, and herbicides have been reviewed for their toxicity by national and international agencies, and for a number of them the experimental evidence of carcinogenicity was considered sufficient to issue specific regulations or recommendations (39, 47). Therefore, insofar as

Table 3

List of chemicals for which there is some evidence of carcinogenicity in experimental animals only or for which the data were inadequate for evaluation of the presence or absence of carcinogenicity (IARC monographs, Volumes 1 to 16)

For the 26 compounds evaluated as carcinogenic to humans, see Table 2

1. Acetamide*	49. γ -Butyrolactone	toluene*	138. Disulfiram	189. Lead carbonate
2. Acridine orange	50. Cadmium acetate	97. 2,5-Diaminotoluene (sulfate)	139. Dithranol*	190. Lead chromate
3. Acrilavinium chloride	51. Cadmium chloride*	98. Diazepam	140. Dulcin	191. Lead phosphate*
4. Actinomycins*	52. Cadmium powder*	99. Diazomethane*	141. Endrin	192. Lead subacetate*
5. Adrenalin	53. Cadmium sulfate*	100. Dibenz(a,h)acridine*	142. Eosin (disodium salt)	193. Ledate
6. Aldrin	54. Cadmium sulfide*	101. Dibenz(a,j)acridine*	143. Epichlorohydrin*	194. Light green SF*
7. Amaranth	55. Calcium arsenate	102. Dibenz(a,h)anthracene*	144. 1-Epoxyethyl-3,4-epoxycyclohexane*	195. Lindane*
8. 5-Aminoacronaphthene	56. Calcium chromate*	103. Dibenzo(c,g)carbazole*	145. 3,4-Epoxy-6-methylcyclohexylmethyl-3,4-epoxy-6-methyl carboxylate*	196. Luteoskyrin*
9. p-Aminoazobenzene*	57. Cantharidin*	104. Dibenzo(h,rst)pentaphene*	146. cis-9,10-Epoxy-stearic acid	197. Magenta*
10. o-Aminoazobenzene*	58. Carbaryl	105. Dibenzo(a,e)pyrene*	147. Estradiol mustard*	198. Maleic hydrazide*
11. p-Aminobenzoic acid	59. Carbon tetrachloride*	106. Dibenz(a,h)pyrene*	148. Ethinylestradiol*	199. Maneb
12. 2-Amino-5-[5-nitro-2-furyl]-1,3,4-thiadiazole*	60. Carmoisine	107. Dibenz(a,i)pyrene*	149. Ethionamide*	200. Mannomustine (hydrochloride)*
13. 4-Amino-2-nitrophenol	61. Catechol	108. Dibenz(a,l)pyrene*	150. Ethylene dibromide*	201. Medphalan
14. Amitrole*	62. Chlorambucil*	109. 1,2-Dibromo-3-chloropropane*	151. Ethylene oxide	202. Medroxyprogesterone acetate*
15. Aniline	63. Chlorinated dibenzodioxins	110. Dibutyltinrosamine*	152. Ethylene sulfide*	203. Merphalan*
16. Anthranilic acid	64. Chloromadinone acetate*	111. o-Dichlorobenzene	153. Ethylenethiourea*	204. Mestranol*
17. Apholate	65. Chlorobenzilate*	112. p-Dichlorobenzene	154. Ethyl methanesulfonate*	205. Methoxychlor
18. Aramite*	66. Chloroform	113. 3,3'-Dichlorobenzidine*	155. Ethyl Selenac	206. 2-Methylaziridine
19. Arsenic trioxide	67. Chloropropham	114. trans-Dichlorobutene	156. Ethyl Tellurac	207. Methylazoxymethanol acetate*
20. Aurothioglucose*	68. Chloroquine	115. 3,3'-Dichloro-4,4'-diamino-diphenyl ether*	157. Ethynodiol diacetate*	208. Methyl carbamate
21. Azaserine*	69. p-Chloro-o-toluidine (hydrochloride)	116. Dieldrin*	158. Evans blue*	209. N-Methyl-N,4-dimethoxyaniline*
22. Aziridine*	70. Cholesterol	117. Diepoxybutane*	159. Fast green FCF*	210. 4,4'-Methylenebis(chloroaniline)*
23. 2-(1-Aziridinyl)ethanol*	71. Chromic chromate*	118. 1,2-Diethylhydrazine*	160. Ferbam	211. 4,4'-Methylenebis(methylaniline)*
24. Aziridyl benzoquinone*	72. Chromium acetate	119. Diethylnitrosamine*	161. 2-(2-Formylhydrazino)-4-(5-nitro-2-furyl)thiazole*	212. 4,4'-Methylenedianiline
25. Azobenzene*	73. Chrysene*	120. Diethyl sulfate*	162. Fusarenon-X	213. Methyl iodide*
26. Barium chromate	74. Chrysoidine*	121. Diglycidyl resorcinol ether	163. Glycidaldehyde*	214. Methyl methanesulfonate*
27. Benz(a)acridine*	75. C.I. Disperse Yellow 3	122. Dihydrosafrole*	164. Glycidyl oleate	215. N-Methyl-N'-nitro-N-nitrosoguanidine*
28. Benz(c)acridine*	76. Cinnamyl anthranilate	123. Dimethisterone	165. Glycidyl stearate	216. Methyl red
29. Benzo(b)fluoranthene*	77. Citrus red No. 2*	124. Dimethoxane*	166. Griseofulvin*	217. Methyl Selenac
30. Benzo(f)fluoranthene*	78. Copper 8-hydroxyquinoline	125. 3,3'-Dimethoxybenzidine*	167. Guinea green B*	218. Methylthiourea*
31. Benzo(a)pyrene*	79. Coumarin*	126. p-Dimethylaminoazobenzene*	168. Heptachlor	219. Metronidazole*
32. Benzo(e)pyrene*	80. Cycasin*	127. p-Dimethylamino-benzenediazo-sodium sulfonate	169. Hexamethylphosphoramide*	220. Mirex*
33. Benzyl chloride*	81. Cyclochlorotine*	128. trans-2-[(Dimethylamino)methylamino]-5-[2-(5-nitro-2-furyl)vinyl]-1,3,4-oxadiazole*	170. Hycanthone (mesylate)*	221. Mitomycin C*
34. Benzyl violet 4B*	82. 2,4-D and esters	129. 3,3'-Dimethylbenzidine*	171. Hydrazine*	222. Monocrotaline*
35. Beryllium*	83. Daunomycin*	130. Dimethylcarbamoyl chloride*	172. Hydroquinone	223. Monuron*
36. Beryllium oxide*	84. D & C Red No. 9	131. 1,1-Dimethylhydrazine*	173. 4-Hydroxyazobenzene	224. 5-(Morpholino-methyl)-3-[(5-nitrofurfuryl)deno]-amino]-2-oxazolidinone
37. Beryllium phosphate*	85. Dichlorodiphenyldichloroethane (DDD)	132. 1,2-Dimethylhydrazine*	174. 8-Hydroxyquinoline	225. 1-Naphthylamine
38. Beryllium sulfate*	86. 1,1-Dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE)	133. Dimethylnitrosamine*	175. Hydroxysenikirkine	226. Native carrageenans*
39. Beryl ore*	87. DDT*	134. Dimethyl sulfate*	176. Indeno(1,2,3-cd)pyrene*	227. Nickel carbonyl
40. BHC (technical grades)*	88. Diacetylaminoazobenzene	135. Dinisopentamethylenetetramine	177. Iron dextran*	228. Nickelocene*
41. Bis(1-aziridinyl)-morpholinophosphine sulfide*	89. N,N-Diacetylbenzidine*	136. 1,4-Dioxane*	178. Iron dextrin*	229. Nickel oxide*
42. Bis(chloroethyl) ether*	90. Diallate*	137. 2,4'-Diphenyldiamine	179. Iron oxide	230. Nickel powder*
43. 1,2-Bis(chloromethoxy)ethane*	91. 2,4-Diaminoanisole (sulfate)		180. Iron-sorbitol-citric acid complex	231. Nickel subsulfide
44. 1,4-Bis(chloromethoxymethyl)benzene*	92. 4,4'-Diaminodiphenyl ether*		181. Isatinidine*	232. Nirdazole*
45. Blue VRS*	93. 1,2-Diamino-4-nitrobenzene		182. Isonicotinic acid hydrazide*	233. 5-Nitroacenaphthene*
46. Brilliant blue FCF*	94. 1,4-Diamino-2-nitrobenzene		183. Isopropyl alcohol	234. 4-Nitrobiphenyl
47. 1,4-Butanediol dimethanesulfonate (Myleran)*	95. 2,6-Diamino-3-(phenylazo)-pyridine (hydrochloride)		184. Isosafrole*	235. Nitrofuralddehyde semicarbazone
48. β -Butyrolactone*	96. 2,4-Diamino-		185. Jacobine	236. 1-[(5-Nitrofurfur-dene)amino]-2-imidazolinone
			186. Lasiocarpine*	237. N-[4-(5-Nitro-2-furyl)-2-thiazolyl]-acetamide*
			187. Lead acetate*	
			188. Lead arsenate	

Table 3-Continued

238 Nitrogen mustard (hydrochloride)*	261 Phenoxybenzamine*	279 Propylthiouracil*	304 Succinic anhydride*	326 Tri(1-aziridinyl)-phosphine oxide
239 Nitrogen mustard N-oxide (hydrochloride)*	262 Phenylbutazone	280 Pyrimethamine*	305 Sudan I*	327 Tri(1-aziridinyl)-phosphine sulfide*
240 Nitrosoethylurea*	263 m-Phenylenediamine (hydrochloride)	281 p-Quinone	306 Sudan II*	328 2,4,6-Tris(1-aziridinyl)-s-triazine*
241 Nitrosomethylurea*	264 p-Phenylenediamine (hydrochloride)	282 Quinazolinone*	307 Sudan III	329 1,2,3-Tris(chloromethoxy)-propane*
242 N-Nitroso-N-methylurethane*	265 N-Phenyl-2-naphthylamine*	283 Reserpine	308 Sudan brown RR	330 Tri(2-methyl-1-aziridinyl)phosphine oxide
243 Norethisterone*	266 Polychlorinated biphenyls*	284 Resorcinol	309 Sudan red 7B	331 Trypan blue*
244 Norethisterone acetate*	267 Ponceau MX*	285 Refrorsine*	310 Sunset yellow FCF	332 Uracil mustard*
245 Norethynodrel*	268 Ponceau 3R*	286 Rhodamine B*	311 2,4,5-T and esters	333 Urethan*
246 Norgestrel	269 Ponceau SX	287 Rhodamine 6G*	312 Tannic acid*	334 Vinyl cyclohexane
247 Ochratoxin A	270 Potassium arsenite	288 Riddelline	313 Terpene polychlorinated nates*	335 2,4-Xylydine (hydrochloride)
248 17 β -Oestradiol*	271 Potassium bis(2-hydroxyethyl)-di-thiocarbamate*	289 Saccharated iron*	314 Testosterone*	336 2,5-Xylydine (hydrochloride)
249 Oestrin	272 Progesterone*	290 Salfrole*	315 Tetraethyl & tetramethyl lead	337 Yellow AB
250 Oestrone*	273 Pronetolol hydrochloride*	291 Scarlet red	316 Thioacetamide*	338 Yellow OB*
251 Oil orange SS*	274 1,3-Propanesultone*	292 Selenium compounds	317 4,4'-Thioaniline*	339 Zeclran
252 Orange I*	275 Proptham	293 Semicarbazide (hydrochloride)*	318 Thiouracil*	340 Zinc chromate hydroxide*
253 Orange G	276 β -Propiolactone*	294 Seneciophylline	319 Thiourea*	341 Zineb
254 Oxazepam*	277 n-Propyl carbamate*	295 Senkirkine	320 Thiram	342 Ziram
255 Oxyphenbutazone	278 Propylene oxide*	296 Sodium arsenate	321 o-Toluidine (hydrochloride)	
256 Parasorbic acid*		297 Sodium arsenite	322 Trichloroethylene*	
257 Patulin*		298 Sodium dichromate	323 Trichloroethylamine hydrochloride	
258 Penicillic acid*		299 Sodium diethyldithiocarbamate	324 Triethylglycol diglycidyl ether*	
259 Phenicarbazide*		300 Sterigmatocystin*	325 Tri(aziridinyl)-p-	
260 Phenobarbital sodium*		301 Streptozotocin*		
		302 Strontium chromate*		
		303 Styrene oxide		

* Asterisk, chemicals for which there is some evidence of carcinogenicity in experimental animals only (see also text, p. 881).

food additives or chemical residues in food are concerned, the value of experimental results to predict a similar effect in humans has been accepted as valid.

The criteria used in the preparation of the draft monographs, in judging the adequacy of the data and in evaluating the carcinogenic risk of chemicals to humans, were first established in 1971 and, with minor modifications, have been adopted by all the working groups whose deliberations have resulted in the first 16 volumes of the IARC monograph series. Adherence to this basic uniform background and maintenance of a certain rigidity in observing the rules set out in the preamble to the monographs permitted a relative uniformity of the evaluations formulated by the working groups.

The growing success and overall acceptance of this program have certainly not prevented suggestions and criticisms. Criticisms mostly refer to the fact that, except for the few instances in which an association between exposure to a chemical and occurrence of cancer in humans has been clearly confirmed, the monographs do not give a clear indication as to whether exposure to a chemical does or does not represent a hazard to humans because no attempt was made to evaluate the potential human risk. In most cases, in fact, the monographs only evaluate the carcinogenic risk of chemicals to experimental animals without making any attempt to interpret the results in terms of a possible risk for humans.

It was for these reasons that an *ad hoc* working group, jointly called by IARC/WHO, was convened in October 1977 in Lyon to update and revise the criteria on which the carcinogenicity of chemicals to humans and/or experimental animals is assessed and on which the evaluation of the

possible carcinogenic risk that they may represent for humans is made.

The report resulting from this meeting will appear as the "Preamble" of Volume 17 of the IARC monograph series which is presently in preparation (38) and will replace the introductory section called "Background and Purpose of the IARC Program on the Evaluation of the Carcinogenic Risk of Chemicals to Man," which appeared since 1971 with only minor modifications in all the 16 published monograph volumes. We would like to mention briefly here only the more significant changes that will henceforth influence the formulation of the evaluations, and the reader is referred to IARC Monograph, Volume 17, for the text in its entirety.

While it was recognized that no adequate criteria are presently available to interpret experimental data on carcinogenicity directly in terms of their carcinogenic potential to humans, it was also noted that extrapolations to a possible human risk can be reasonably approximated by utilizing data from appropriate animal tests. These data, however, may represent different degrees of evidence, this drawback being mainly due to our present insufficient knowledge of the mechanisms of carcinogenesis. "Strong evidence" of carcinogenicity is indicated by the unquestionable production of malignant tumors, while "weak evidence" of carcinogenicity reflects the qualitative and/or quantitative limitations of the experimental results of which a particular case could be, for instance, the induction solely from benign tumors in mice. In the presence of appropriate experimental carcinogenicity data and in the absence of adequate human data, it is reasonable to regard chemicals for which there is "strong evidence" of carcinogenicity as if they were carcinogenic to humans. Chemicals

for which there is weak evidence of carcinogenicity in experimental animals will, in general, require more experimental and epidemiological investigation.

It was recognized that there are no acceptable methods for quantifying the possible errors in making an approximate quantitative evaluation of human risk at some given exposure level on the basis of data that provide strong evidence of carcinogenicity in experimental animals. The limited data available, however, suggest that such a relationship may exist, at least within certain classes of carcinogenic chemicals, provided that account is taken of the nature of the chemical concerned and of the possible physiological, pharmacological, and toxicological differences between the test animals and humans.

More consideration than in the past is also given to indirect tests, most of which do not have the production of neoplasms in animals as the end point and are generally referred to as "short-term" tests. These tests are proposed to be used: (a) for the prediction of potential carcinogenicity when other carcinogenicity data are absent, (b) to contribute to the setting of priorities for chemicals to be tested in animals, (c) for the identification of active fractions from complex mixtures containing carcinogens, (d) for the identification of active metabolites of carcinogens in human body fluids, and (e) to help elucidate mechanisms of chemical carcinogenesis.

The last section of the monographs, which was originally headed "Comments on Data Reported and Evaluation," and comprised 2 subheadings, 1 for experimental data and 1 for human data, will now be called "Summary of Data Reported and Evaluation"; it will have 3 sections, i.e., a summary of experimental data, a summary of the human data, and the evaluation. The evaluation section will take into consideration all of the data in the monograph, particularly the summarized information on animals and humans.

The revised criteria for the evaluation of the carcinogenic risk of chemicals to humans have already been applied by the IARC working group that finalized the monographs scheduled to appear as Volume 17, which considers some *N*-nitroso compounds (38).

By applying the new criteria for the evaluation of the carcinogenicity of chemicals as they are described in the new preamble to the monographs, as well as acquiring further carcinogenicity and related data on chemicals and new knowledge on the mechanisms of carcinogenicity as it may become available, we hope that in the absence of epidemiological data a more clear distinction can be made between chemicals for which there is or is not evidence of a possible carcinogenic effect in humans.

We hope that in this way the critical analysis of the experimental and human data and the ensuing evaluations of environmental chemicals will make future IARC monographs more useful in assisting national and international authorities in formulating decisions concerning preventive measures.

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