information must so indicate on the first page of its application and submit two additional copies of its application from which the information which the applicant claims is confidential has been deleted, together with a statement specifying why any such information is privileged or confidential. Each application must indicate whether the applicant or any person acting on its instructions has filed or intends to file any other application or claim of whatever nature regarding the matters at issue in the underlying Amtel enforcement proceeding. Each application must also include the following statement: I swear (or affirm) that the information submitted is true and accurate to the best of my knowledge and belief. See 10 CFR 205.283(c); 18 U.S.C. 1001. In addition, the applicant should furnish us with the name, title, and telephone number of a person who may be contacted by the OHA for additional information concerning the application. All applications should be sent to: Amtel Consent Order Refund Proceeding, Office of Hearings and Appeals, Department of Energy, Washington, DC. 20585. All applications for refund received within the time limit specified will be processed pursuant to 10 CFR 205.284 and the procedures set forth in this Decision and Order.

In order to assist applicants in establishing eligibility for a portion of the consent order funds, the following subjects should be covered in each application:

A. Each applicant should establish its volume of purchases by calendar quarter for the period of time for which it is claiming it was injured by the alleged overcharges.

B. Each applicant should state whether it was a regular customer of Amtel or whether it made only spot purchases from Amtel.

C. Each applicant should specify how it used the Amtel products—i.e., whether it was a reseller (including retailers) or ultimate consumer.

D. If the applicant is a reseller who wishes to claim a refund in excess of \$2,500 per calendar year or a total refund in excess of \$15,000 under the volumetric methodology, it should also

(i) State whether it maintained banks of unrecouped produce cost increases from the date of the alleged violation until the product was decontrolled. It should furnish OHA with quarterly bank calculations.

(ii) State whether it or any of its affiliates have filed any other applications for refunds in which they have referred to their banks to demonstrate injury. (iii) Submit evidence to establish that it did not pass on the alleged injury to its customers. For example, a firm with multiple suppliers in addition to Amtel may submit market surveys to show that price increases to recover alleged overcharges were not feasible.

E. If the applicant is a reseller who wishes to claim a refund in excess of the per gallon volumetric amount, it must, in addition, to providing the information specified in paragraph D, submit evidence which proves that it was injured by alleged overcharges on a per gallon basis at a level greater than the volumetric amount.

F. The applicant should report whether it is or has been involved as a party in any DOE or private Section 210 enforcement actions. If these actions have terminated, the applicant should furnish a copy of any final order issued in the matter. If the action is ongoing, the applicant should briefly describe the action and its current status. Of course, the applicant is under a continuing obligation to keep the OHA informed of any change in status during the pending of its application for refund. See 10 CFR 205.9(d).

It Is Therefore Ordered That: (1) Applications for refunds from the funds remitted to the Department of Energy by Amtel, Inc. pursuant to the Consent Order executed on February 7, 1980, may not be filed.

(2) All applications must be filed no later than 90 days after publication of this Decision and Order in the Federal Register.

George B. Breznay,

Director, Office of Hearings and Appeals. Date: August 24, 1984.

Footnotes

(1) Amtel purchased South Central on March 29, 1974 and sold it on May 3, 1979. Amtel purchased Hauck Oil on January 1, 1973. Amtel changed Hauck Oil's name to Premier Automotive, Inc. in July 1976 and discontinued the Hauck Oil/Premier Automotive operations in November 1977. Hauck Trading was formed from Hauck Oil in April 1974. In April 1975, Amtel changed Hauck Trading's name to Ambur Oil and Trading Company, Inc. The Ambur Oil operations were sold by Amtel in May 1978.

(2) One commenter urges that, with the exception of end-users, refunds be made only to claimants with fully documented claims and no presumption of injury based upon small purchase volumes be allowed. We cannot accept this position. For the reasons stated in the text, we have consistently found that a presumption of injury for small claims is necessary to effect the purposes of Subpart V and is in accordance with the regulatory requirements. See 10 CFR 205.282[c].

(3) Additionally, we note that many multistation operations should be eligible for larger refunds as a result of our increasing the threshold to the \$15,000 level.

(4) In the event that successful claims exceed the amount in the consent order fund, we will reduce the refunds on a pro rata basis. We will, therefore, not disburse any refunds until the deadline for applications has passed and we have determined the aggregate amount of the refunds.

[FR Dec. 04-31319 Filed 11-20-04 8:45 am] BILLING CODE 6450-01-M

ENVIRONMENTAL PROTECTION AGENCY

[OPTS-41015; FRL-2725-7]

Fifteenth Report of the Interagency Testing Committee to the Administrator; Receipt of Report and Request for Comments Regarding Priority List of Chemicals

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: The Interagency Testing Committee (ITC), established under section 4(e) of the Toxic Substances Control Act (TSCA), transmitted its Fifteenth Report to the Administrator of EPA on November 6, 1984. This report, which revises and updates the Committee's priority list of chemicals, adds seven designated chemicals to the list for priority consideration by EPA in the promulgation of test rules under section 4(a) of the Act. The new designated chemicals are anthraquinone, 2-chloro-1,3-butadiene, cumene, mercaptobenzothiazole, octamethylcyclotetrasiloxane, pentabromoethylbenzene, and sodium N-methyl-N-oleoyltaurine. The Fifteenth Report is included in this notice.

The Agency invites interested persons to submit written comments on the Report, and to attend Focus Meetings to help narrow and focus the issues raised by the ITC's recommendations. Members of the public are also invited to inform EPA if they wish to be notified of subsequent public meetings on these chemicals. EPA also notes the removal of 5 chemicals from the priority list because EPA has responded to the ITC's previous recommendations for testing of the chemicals.

DATES: Written comments should be submitted by December 31, 1934. Focus Meetings will be held on December 19 and 20, 1934.

ADDRESSES: Send written submissions to: TSCA Public Information Office (TS– 793), Office of Pesticides and Toxic Substances, Environmental Protection Agency, Rm., E-108, 401 M St., SW., Washington, D.C. 20460.

Submissions should bear the document control number (OPTS-41015).

The public record supporting this action, including comments, is available for public inspection in Rm., E-107 at the address noted above from 8 a.m. to 4 p.m. Monday through Friday, except legal holidays. Focus Meetings will be held in Rm. 3906, EPA Headquarters, 401 M St., SW, Washington, D.C. Persons planning to attend any one of the Focus Meetings and/or seeking to be informed of subsequent public meetings on these chemicals, should notify the 'TSCA Assistance Office at the address listed below. To insure seating accommodations at the Focus Meeting, persons interested in attending are asked to notify EPA at least one week ahead of the scheduled dates:

FOR FURTHER INFORMATION CONTACT: Edward A. Klein, Director, TSCA Assistance Office (TS-799), Office of Toxic Substances, Environmental Protection Agency, 401 M St., SW., Washington, D.C. 20460, Toll Free: (800– 424–9065), In Washington, D.C.. (554– 1404, Outside the USA: (Operator-202– 554–1404).

SUPPLEMENTARY INFORMATION: EPA has received the Fifteenth Report of the TSCA Interagency Testing Committee to the Administrator.

I. Background

Section 4(a) of TSCA (Pub. L. 94–469, 90 Stat. 2003 et seq; 15 U.S.C. 2601 et seq.) authorizes the Administrator of EPA to promulgate regulations requiring testing of chemical substances and mixtures in order to develop data relevant to determining the risks that such chemical substances and mixures may present to health and the environment.

Section 4(e) of TSCA established an Interagency Testing Committee to make recommendations to the Administrator of EPA of chemical substances and mixtures to be given priority consideration in proposing test rules under section 4(a). Section 4(e) directs the Committee to revise its list of recommendations at least every 6 months as necessary. The ITC may "designate" up to 50 substances and mixtures at any one time for priority consideration by the Agency. For such designations, the Agency must within 12 months either initiate rulemaking or issue in the Federal Register its reasons for not doing so. The ITC's Fifteenth Report was received by the Administrator on November 6, 1984, and follows this Notice. The Report designates seven substances for priority

consideration and response by EPA within 12 months.

II. Written and Oral Comments and Public Meetings

EPA invites interested persons to submit detailed comments on the ITC's new recommendations. The Agency is interested in receiving information concerning additional or ongoing health and safety studies on the subject chemicals as well as information relating to the human and environmental exposure to these chemicals. A notice is published elsewhere in today's Federal Register adding the seven substances designated in the ITC's Fifteenth Report to the TSCA section 8(d) Health and Safety Data Reporting Rule (40 CFR Part 716). The section 8(d) rule requires the reporting of unpublished health and safety studies on the listed chemicals. These seven chemicals will also be added to the TSCA section 8(a) **Preliminary Assessment Information** Rule (40 CFR Part 712) published elsewhere in this issue. The section 8(a) rule requires the reporting of production volume, use, exposure, and release information on the listed chemicals.

Focus Meetings will be held to discuss relevant issues pertaining to the chemicals and to narrow the range of issues/effects which will be the focus of the Agency's subsequent activities in responding to the ITC recommendations. The Focus Meetings will be held December 19 and 20, 1984, in Rm. 3906, EPA Headquarters, 401 M St., SW, Washington, D.C. These meetings are intended to supplement and expand upon written comments submitted in response to this notice. The schedule for the Focus Meetings is as follows: December 19, 9:30 a.m.-2-Chloro-1,3butadiene; 11:00 a.m.-

Octamethylcyclotetrasiloxane; 1:30 p.m.—Pentabromoethylbenzene; 3:00 p.m.—Sodium N-methyl-N-oleoyltaurine; December 20, 9:30 a.m.—Anthraquinone; 11:00 a.m.—Mercaptobenzothiazole; 2:00 p.m.—Cumene.

Persons wishing to attend one or more of these meetings or subequent meetings on these chemicals should call the TSCA Assistance Office at the toll free number listed above at least one week in advance.

All written submissions should bear the identifying docket number (OPTS-41015).

III. Status of List

In addition to adding the seven designations to the priority list, the ITC's Fifteenth Report notes the removal of five chemicals from the list since the last ITC report because EPA has responded to the Committee's prior recommendations for testing of the chemicals. Subsequent to the ITC's preparation of its Fourteenth Report, EPA responded to the ITC's recommendations for five additional chemicals. The five chemicals removed and the dates of publication in the Federal Register of EPA's responses to the ITC for these chemicals are: calcium naphthenate, May 21, 1984 (49 FR 21411-21418); cobalt naphthenate, May 21, 1984 (49 FR 21411-21418); lead naphthenate, May 21, 1984 (49 FR 21411-21418); methylolurea, May 21, 1984 (49 FR 21371-21375); and 2-phenoxyethanol, May 21, 1984 (49 FR 21407-21411). The current list contains 16 designated substances or groups of substances and two recommended substances or groups of substances.

(Sec. 4, Pub. L. 94-469, 90 Stat. 2003; [15 U.S.C. 2601])

Dated: November 15, 1984.

John A. Moore,

Assistant Administrator for Pesticides and Toxic Substances.

Fifteenth Report of the TSCA Interagency Testing Committee to the Administrator, Environmental Protection Agency

Summary

Section 4 of the Toxic Substances Control Act of 1976 (TSCA, Pub. L. 94-469) provides for the testing of chemicals in commerce that may present an unreasonable risk of injury to health or the environment. It also provides for the establishment of a Committee, composed of representatives from eight designated Federal agencies, to recommend chemical substances and mixtures (chemicals) to which the Administrator of the U.S. Environmental Protection Agency (EPA) should give priority consideration for the promulgation of testing rules.

Section 4(e)(1)(A) of TSCA directs the Committee to recommend to the EPA Administrator chemicals to which the Administrator should give priority consideration for the promulgation of testing rules pursuant to section 4(a). The Committee is required to designate those chemicals, from among its recommendations, to which the Administrator should respond within 12 months by either initiating a rulemaking proceeding under section 4(a) or publishing the Administrator's reason for not initiating such a proceeding. Every 6 months, the Committee makes those revisions in the TSCA section 4(e) Priority List that it determines to be necessary and transmits them to the EPA Administrator.

46932

As a result of its deliberations, the Committee is revising the TSCA section 4(e) Priority List by the addition of seven chemicals and is noting the removal of five, as a result of responses by EPA.

The Priority List is divided into two parts: part A contains those recommended chemicals and groups designated for priority consideration and response by the EPA Administrator within 12 months, and part B contains chemicals and groups that have been recommended for priority consideration by EPA without being designated for response within 12 months. Although TSCA does not establish a deadline for EPA response to nondesignated chemicals and groups (part B of the Priority List), the Committee anticipates that the EPA Administrator will respond in a timely manner.

The chemicals being added to the Priority List are presented, together with the types of testing recommended, in the following Table 1.

TABLE 1.- ADDITIONS TO THE SECTION 4(E)

- Chemical/group	Recommended studies -
Designated for re- sponses within 12 months:	
Anthrequinone (CAS No. 84-66-1).	Chemical fata: Water solubility biodegradation. Ecological et
	fects: Acute toxicity to fish aquatic invertebrates, an algas; chronic toxicity to aquatic organisms (testin cenditional upon results o
2-Chloro-1,3-butadiene	ecute tests). Chemical fate: Water solubility
(CAS No. 128-99-8).	persistence. Ecological el fects: Acute loxicity to sensi tive life stages of fish, aquat ic invertebrates, and algae
Currens (CAS No. 98- 82-8).	Health effects: Short-term gen otoudcity; chronic effects in cluding oncogenicity; terato genicity and reproductive tos icity. Ecological effect Acute and chronic toxicity to sstuarine and freahwater fiel
Mercaptobenzothiazole	and invertebrates. Chemical fate: Dissociation
(CAS No. 149-30-4).	constant; persistence is retain and soit, leaching/m gration. Environmental el lecta: Acute and chronic too lotty to fish, equatic inverte traiplemb.
Octamenthylcyclotetrasi- loxane (CAS No. 556- 67-2).	Chemical fate: Water solubility octanol/water partition coeffi- clent; biodegradation. Ecolog loal effects: Acute toxicity to linh, aquatic invertabrater and algae (concentrations co- the chemical to be measure (luring, the course of th (toules); chronic toxicity to (quate organisme (testin
Pantabromoethyloan- zena (CAS No. 85-22- 3).	conditional upon results of recute tasts). Health effects: Two-year chron ic bioassay; teratogenicit study. Ecological effects /Acute and chronic toxicity a fait. equatic invertebrater und plants.

TABLE 1.- ADDITIONS TO THE SECTION 4(E) PRIORITY LIST-NOVEMBER 1984-Continued

Chemical/group	Recommended studies
Sodium Armethyl-Ar olecyttaurine (CAS No. 137-20-2).	Health effects: Short-term gen- otoxicity; sensitization; chron- le toxicity to include onco- genicity (testing conditional upon results of short-term
	tests).
 Recommended but not designated for response 	in the intervence of the

TSCA Interagency Testing Committee

Statutory Member Agencies and Their Representatives

- Council on Environmental Quality Thomas H. Magness, III, Member (1) George W. Schlossnagle, Alternate
- Department of Commerce Bernard Greifer, Member and Vice Chairperson
- Environmental Protection Agency Carl R. Morris, Member Arthur M. Stern, Alternate (2) National Cancer Institute Elizabeth K. Weisburger, Member and Chairperson
- Richard Adamson, Alternate National Institute of Environmental Health Sciences
- Dorothy Canter, Member National Institute for Occupational Safety and Health Rodger L. Tatken, Member Sanford S. Leffingwell, Alternate National Science Foundation
- Winston C. Nottingham, Member Occupational Safety and Health Administration
 - Ralph Yodaiken, Member
- Liaison Agencies and Their Representatives
- Consumer Product Safety Commission Lakshmi Mishra Arthur Gregory Department of Agriculture Homer E. Fairchild Richard M. Parry, Jr. Department of Defense Edmund Cummings (3) Patrick A. Truman Department of the Interior
- Vyto A. Adomaitis David R. Rosenberger
- Food and Drug Administration Arnold Borsetti Allen H. Heim
- National Toxicology Program Dorothy Canter

Committee Staff

Martin Grief, Executive Secretary (4) Norma Williams, ITC Coordinator Alan Carpien—Office of the General Counsel, EPA Stephen J. Ells—Office of Toxic Substances, EPA

Vera W. Hudson—National Library of Medicine

Notes

(1) Thomas Magness resigned from the Committee on May 1, 1984.

(2) Arthur Stern was appointed Acting Executive Secretary on September 4, 1984.
(3) Edmund Cummings was appointed on lune 18, 1984.

(4) Martin Grief died on August 8, 1984. He served 4 years of distinguished and faithful service as the ITC Executive Secretary. The Committee deeply regrets his passing. His dedication and outstanding contributions to the goals of the Committee will long be remembered.

The Committee acknowledges and is grateful for the assistance and support given to it by the staffs of CRCS, Inc., and Dynamac Corporation (technical support prime and subcontractors) and personnel of the EPA Office of Toxic Substances.

Chapter 1—Introduction

1.1 Background. The TSCA Interagency Testing Committee (Committee) was established under 50 section 4(e) of the Toxic Substances Control Act of 1976 (TSCA, Pub. L. 94-.11.715 469). The specific mandate of the Committee is to recommend to the Administrator of the U.S. Environmental Protection Agency (EPA) chemical substances and mixtures in commerce - : that should be given priority consideration for the promulgation of testing rules to determine their potential hazard to human health and/or the environment. TSCA specifies that the Committee's recommendations shall be in the form of a Priority List, which is to be published in the Federal Register. The Committee is directed by section 4(e)(1)(A) of TSCA to designate those chemicals on the Priority List to which the EPA Administrator should respond within 12 months by either initiating a rulemaking proceeding under section 4(a) or publishing the Administrator's reason for not initiating such a proceeding. There is no statutory time limit for EPA response regarding chemicals that ITC has recommended, but not designated for response within 12 months.

Every 6 months, the Committee makes those revisions in the section 4(a) Priority List that it determines to be necessary and transmits then to the EPA Administrator.

The Committee is comprised of representatives from eight statutory member agencies, five liaison agencies, and one national program. The specific representatives and their affiliations are

named in the front of this report. The Committee's chemical review procedures and prior recommendations are described in previous reports (Refs. 1 through 14).

1.2 Committee's previous reports. Fourteen previous reports to the EPA Administrator have been issued by the Committee and published in the Federal Register (Refs. 1 through 14). Seventynine entries (chemicals and groups of chemicals) were recommended for priority consideration by the EPA Administrator and designated for response within 12 months. In addition, two groups were recommended without being so designated. Removal of 65 entries was noted in the previous reports.

1.3 Committee's activities during this reporting period. Between April 1, 1984, and September 30, 1984, the Committee continued to review chemicals from its fourth and fifth and scoring exercises.

The Committee contacted chemical manufacturers and trade associations to request information that would be of value in its deliberations. Most of those contacted provided unpublished information on current production, exposure, uses, and effects of chemicals under study by the Committee.

During this reporting period, the Committee examined 97 chemicals for priority consideration. Seven chemicals were added to the section 4[e) Priority List, and 16 were deferred indefinitely. The remaining chemicals are still under study.

1.4 The TSCA section 4(e) Priority List. Section 4(e)(1)(B) of TSCA directs the Committee to: ' make such revisions in the [priority] list as it determines to be necessary transmit them to the and Administrator together with the Committee's reasons for the revisions." Under this authority, the Committee is revising the Priority List by adding seven chemicals: Anthraquinone; 2chloro-1,3-butadiene; cumene; mercaptobenzothiazole; and sodium Nmethyl-N-oleoyltaurine. All of these chemicals are designated for response within 12 months. The testing recommended for these chemicals and the rationales for the recommendations are presented in Chapter 2 of this report.

Five chemicals are being removed from the Priority List because the EPA Administrator has responded to the Committee's prior recommendations for testing them. They are: Calcium naphthenate Cobalt naphthenate Lead naphthenate Methylolurea 2-Phenoxyethanol

With the seven recommendations and five removals noted in this report, 18 entries now appear on the section 4(e) Priority List. The Priority List is divided in the following Table 2 into two parts; namely, Table 2A, Chemicals and Groups of Chemicals Designated for Response Within 12 Months, and Table 2B, Other Recommended Chemicals and Groups.

Table 2.—The TSCA Section 4(e) Priority List, November 1984

2A. CHEMICALS AND GROUPS OF CHEMICALS DESIGNATED FOR RESPONSE WITHIN 12 MO

Entry	Date of designation		
1. Anthraquinone			
3. 2-(2-Butoxyethoxy) ethyl acetale	November 1984.		
 Cumene	Do. May 1984.		
7. Diisopropyl biphenyl			
9. 2-Ethylhexanoic acid 10. 1, 2, 3, 4, 7, 7-Hexachloronorborna- diene.	May 1984. November 1983.		
11. Isopropyl biphenyl	Novomber 1984.		
Octamethylcyclotetrasiloxane Oloylantine Sentabromoethylbenzene Sodium Armethyl-Moleoyltaurine	November 1983. November 1984.		

2B. OTHER RECOMMENDED CHEMICALS AND GROUPS OF CHEMICALS

Entry	Date of recommendation	
1. Carboluran Intermodiates	November 1982.	
2. 3,4-Dichorobenzotrifluoride	May 1984.	

To date, 70 chemicals and groups of chemicals have been removed from the Priority List. The cumulative list is presented in the following Table 3.

TABLE 3.- CUMULATIVE REMOVALS FROM THE TSCA SECTION 4(E) PRIORITY LIST-NOVEMBER 1984

	EPA responses to committee	recommendations
Chemical/group	Federal Regist	ler
	Citation	Publication Date
I. Acetonitrile	47 FB 58020-58023	Dec. 20, 198
2. Acrylamide		July 31, 198
3. Alkyl epoxidos		Jan. 4, 198
4. Alkyl phthalates		Oct. 30, 198
5. Alkyltin compounds		'Fob. 5, 190
3. Aniline and bromo- chloro-, and/or nitroanilines		
7. Antimony metal		
3. Antimony sulfide		
9. Antimony tnoxide		
10. Aryl phosphates	48 FR 57452-57460	
11. Benzidine-based dyes		
12. Benzyl butyl phthalate		
13. Biphenyl		
14. Bis(2-ethylhexyl) tereohthalate		
15. Butyl glycolyl butyl phthalate		
16. Calcum naphthenate	49 FR 21411-21418	
17. Chlorendic acid		
18. Chlorinated benzenes, mono- and di-		
19. Chlorinated benzenes, tn., tetra., and penta-	49 FB 1760-1770	Jan. 13, 198
20. Chlonnated naphthalenes		
21. Chlorinated paraffins		
22. 4-Chlorobenzotrifluoride		
23. Chloromethane		
24. 2-Chlorotoluene		
25. Cobalt naphthenate		
26. Cresols		
27. Cyclohexanone		
28. o-Dianiskine-based dyes		Nov. 5, 198
29. Dibutyltin bis(isooctyl maleate)		
30. Dibutyltin bis(isooctyl mercaptoacetate)		
31. Dibutyllin bis(lauryl mercaptide)		
32. Dibutyltin dilaurate		
33. Dichloromethane		

		a se la ser a s	EPA responses to committee recommendations		
Chemical/group		a serie de la s	Federal Register		
		Citation	Publication Date		
34. 1,2-Dichloropropane			49 FR 899-908		
35. Diethylenetrienene		and the second state of th	47 FR 18386-18391		
38. Dimethyltin bieliscochi merceptosostate	a)		46 FR 51361-51366		
37. 1,3-Dioxolane			49 FR 32113-32114	and a state of a state	
38. Ethyltoluene			46 FR 23066-23095		
39. Fluorosikense	Courses and and the second property of the local second		46 FR 53704-53708	Mey 23, 198	
40. Formamide		Contraction to diversify the second			
1. Glycidal and its derivatives.		and the grant part of the Deliver Syndrom program in the same state the Specific Conference in the same state of the same	48 FR 23098-23102	May 23, 198	
12. Halogenated alkyl epoxiden			48 FR 57686-57700	Dec. 30, 198	
13. Hexachloro-1,3-butadiene			40 FH 5/000-5//00		
14. Hexechlorocyclopentadiene	The second s		47 FR 58029-58031		
15. Hexachioroethana	A REAL PROPERTY AND A REAL		4/ FT 30023-36023	Dec. 29, 198 Apr. 28, 198	
16. Hydroquinone			47 FR 18175-18178	Apr. 28, 198	
7. Isophorone		and setting a de a set begin time as and as a set of or a setting setting and it is taking	49 FR 438-449		
8. Lead mephthenate	A REAL PROPERTY OF LODIES AND ADDRESS OF A REAL PROPERTY OF A REAL PRO	nandeli badre Gangel ant ann de port och bord be diraditegenare, en bar nam er diller, en a so	48 FR 727-730	Jan. 6, 198	
9. Meetyl cuide			49 111 21411-21418	Jan. 6, 198 May 21, 198	
0. 4,4'-Methylenecianiline			48 FR 30699-30706		
1. Methyl athyl katone	The second s		40 FR 58025-58029		
2. Methyl isobutyl ketone	-		47 FR 58025-58029		
3. Methylotures			49 FR 21371-21375		
4. Monobutyitin triellacoctyl murcaptoaceta		an an a far a ga anna a tha a tha a tha an anna an a tha anna ann	48 FR 51361-51366		
5. Monomethyltin tris(leooctyl mercaptoace			48 FR 51361-51366		
8. Nitrobenzene			48 FR 30300-30320	Nov. 6, 196	
7. 2-Phenoxyethanol		a a ben bin die 1998 de alle de Calender van ander die de Antoine de Antoine de Antoine de Antoine de Antoine d	49 FR 21407-21411		
8. Phenylanediamines			47 FR 973-983		
9. Potychiorinated terphenetis			46 FR 54482-54483		
0. Pyridne			46 FR 56031-58035		
1. Quinone		A CONTRACTOR CONTRACTOR OF A DESCRIPTION OF	47 FR 58031-58035		
2 4-(1,1,2,3-Tetramethybuty) phanol					
3. c-Tolidine-based dyes			49 FR 29449-29450		
4. Tokiene	Called Big 1987 is a surface international properties and provide a big of a distance of a state of a state of a		48 FR 55004-65008		
5. 1.2.4,-Trimethylberzens			47 FR 56391-56392		
8. Trimethylbertzenes			48 FR 23068-23095	May 23, 198	
7 1 1 1 Trichlamathana			48 FR 23089-23095		
7. 1,1,1-Trichloroethene	· · ·	an a	46 FR 30300-30320		
9. Trie(2-ethylinexy) primelikate	1		47 FR 49488-49487	Nov. 1, 196	
O. INDEC-CONTRACTOR STORINGED	Contraction of the second s		48 FR 51842-51845	Nov. 1, 196	
0. Xylenes.			47 FR 56392-56394		

TABLE 3 --- CUMULATIVE REMOVALS FROM THE TSCA SECTION 4(E) PRIORITY LIST-NOVEMBER 1984-Continued

* Removed by the Committee for reconsideration. Seven individual group stambers ware subsequently designated in the 11th (TC Report (Ref. 11)) for priority consideration

References

(1) Initial Report to the Administrator, Environmental Protection Agency, TSCA Interagency Testing Committee, October 1, 1977. Published in the Federal Register of Wednesday, October 12, 1977, 42 FR 55020-55080. Corrections published in the Federal Register of November 11, 1977, 42 FR 58777-58778. The report and supporting dossiers were also published by the Environmental Protection Agency, EPA 580-10-78/001, January 1978.

(2) Second Report of the TSCA Interagency Testing Committee to the Administrator. Environmental Protection Agency: TSCA Interagency Testing Committee, April 1978. Published in the Federal Register of Wednesday, April 19, 1178, 43 FR 18684-18688. The report and supporting dossiers were also published by the Environmental Protection Agency, EPA 580-10-78/002, July 1978.

(3) Third Report of the TSCA Interagency Testing Committee to the Administrator. Environmental Protection Agency. TSCA Interagency Testing Committee. October 1978. Published in the Fuderal Register of Monday, October 10, 1978, 43 FR 50630-50635. The report and supporting dossiers were also published by the Environmental Protection Agency. EPA 560-10-79/001, January 1979.

(4) Fourth Report of the TSCA Interagency Testing Committee to the Administrator. Environmental Protection Agency. TSCA Interagency Testing Committee, April 1979. Published in the Federal Register of Friday. June 1, 1979, 44 FR 31861-31893. (5) Fifth Report of the TSCA Interagency Testing Committee to the Administrator, Environmental Protection Agency. TSCA Interagency Testing Committee, November 1979. Published in the Federal Register of Friday, December 7, 1979, 44 FR 70664-70674.

(8) Sixth Report of the TSCA Intersectory Testing Committee to the Administrator. Environmental Protection Agency. TSCA Interagency Testing Committee, April 1980. Published in the Federal Register of Wednesday, May 28, 1980, 45 FR 35897-35910.

(7) Seventh Report of the TSCA Interagency Testing Committee to the Administrator, Environmental Protection Agency, TSCA Interagency Testing Committee, October 1980, Published in the Federal Register of Tuesday, November 25, 1980, 45 FR 78432-78446.

(8) Eighth Report of the TSCA Interagency Testing Committee to the Administrator, Environmental Protection Agency. TSCA Interagency Testing Committee, April 1981. Published in the Federal Register of Friday, May 22, 1981, 48 FR 28138-28144.

(9) Ninth Report of the TSCA Intersectory Testing Committee to the Administrator, Environmental Protection Agency. TSCA Intersectory Testing Committee, October 1981. Published in the Federal Register of Friday, February 5, 1982, 47 FR 5458-5483.

(10) Tenth Report of the TSCA Interagency Testing Committee to the Administrator, Environmental Protection Agency. TSCA Interagency Testing Committee, April 1982. Published in the Federal Register of Tuesday; May 25, 1982, 47 FR 22585-22596. (11) Eleventh Report of the TSCA Interagency Testing Committee to the Administrator, Environmental Protection Agency. TSCA Interagency Testing Committee, October 1982, Published in the Federal Register of Friday, December 3, 1982, 47 FR 54025-54044.

(12) Twelfth Report of the TSCA. Interagency Testing Committee to the Administrator, Environmental Protection Agency. TSCA Interagency Testing Committee, May 1983. Published in the Federal Register of Wednesday, June 1, 1983; 48 FR 24443-24452.

(13) Thirteenth Report of the TSCA Interagency Testing Committee to the Administrator, Environmental Protection Agency. TSCA Interagency Testing Committee, November 1983. Published in the Federal Register of Wednesday, December 14, 1983, 48 FR 55674-55684.

(14) Fourteenth Report of the TSCA Interagency Testing Committee to the Administrator, Environmental Protection Agency, TSCA Interagency Testing Committee, May 1984. Published in the Federal Register of Tuesday, May 29, 1964. 49 FR 22369-22407.

Chapter 2—Recommendations of the Committee

2.1 Chemicals recommended for priority consideration by the EPA Administrator. As provided by section 4(e)(1)(B) of TSCA, the Committee is adding the following seven chemical substances to the section 4(e) Priority List: Anthraquinone; 2-chloro-1,3butadiene; cumene; mercaptobenzothiazole; octamethylcyclotetrasiloxane; pentabromoethylbenzene; and sodium N-methyl-N-oleoyltaurine. The recommendation of these chemicals is being made after considering the factors identified in section 4(e)(1)(A) and other available relevant information, as well as the professional judgment of Committee members.

The seven recommendations designated for response by the EPA Administrator within 12 months and supporting rationales are presented in section 2.2 of this report.

2.2 Chemicals designated for response within 12 months with supporting rationales.

2.2a Anthraguinone

Summary of recommended studies. It is recommended that anthraquinone be tested for the following:

A. Chemical Fate:

Water solubility

Biodegradation

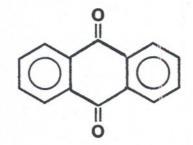
B. Ecological Effects:

Acute toxicity to fish, aquatic invertebrates, and algae

Chronic toxicity to aquatic oranisms (testing conditional upon results of acute tests)

Physical and Chemical Information

CAS Number: 84–65–1. Synonyms: 9,10-Anthracenedione; 9,10-Anthraquinone; 9,10-Dioxoanthracene. Structural Formula:



Empirical Formula: C₁₄H₈O₂ Molecular Weight: 208 Melting Point: 283.5–285 °C. Boiling Point: 379–381 °C. Vapor Pressure: 1mmHg at 190 °C. Specific Gravity: 1.419–1.438 (20/4). Solubility in Water: 0.05mg/L (Ref. 7, C–I–L, 1984).

Log Octanol/Water Partition Coefficient: 2.16 (estimated; Ref. 17, Lyman et al., 1982).

Description of Chemical: Light-yellow needles.

Rationale for Recommendations

I. Exposure information—A. Production/use/disposal. No publicly available data were found on the current production volume of anthraquinone; however, it is known to be produced in the United States. In 1979, 3.7 million pounds of anthraquinone were imported (Ref. 24, USITC, 1980). In 1982, 364,358 pounds were imported (Ref. 25, USITC, 1983).

The major uses for anthraquinone are as an intermediate in the manufacture of dyes and dyestuff intermediates and as a kraft pulping additive to aid in the delignification of wood pulp (Refs. 12, 15, and 4, Kirk-Othmer, 1978, 1982; CEH, 1983). It is also used as a catalyst in the isomerization of vegetable oils and in the polymerization of drying oils, as an accelerator in nickel electroplating, and as an aid in improving the adhesion and heat stability of tire cords (Refs. 12, 13, and 14, Kirk-Othmer, 1978, 1979, 1980). In Europe, it is also used as a bird repellent, applied to growing crops and seeds (Ref. 15, Kirk-Othmer, 1982).

B. Evidence for exposure. Games and Hites (Ref. 9, 1977) found anthraquinone in six raw wastewater samples from a dye manufacturing plant at

concentrations ranging from 49 to 110 ppb but did not detect anthraquinone in the effluent from the plant's wastewater treatment facility. According to Voss (Ref. 29, 1981), increased use of anthraquinone in wood pulping "may show up environmental effects which are, as yet, not obvious." Typical addition levels in the paper mills are 0.025-0.1 percent anthraquinone on bone-dry wood.

Anthraquinone has been found in the Waal River in the Netherlands (Ref. 19, Meijers and Van der Leer, 1976) and in the Baltic Sea (Ref. 8, Ehrhardt et al., 1982). Akiyama et al. (Ref. 1, 1980) found 5.2 ng/L of anthraquinone in samples of Japanese tapwater and detected it in sediments. In a study of drinking water contaminants in 12 Great Lakes municipalities, anthraquinone was found in all 12 locations (Ref. 30, Williams et al., 1982). The concentrations ranged from 0.3 to 72 ng/

L.

The compound has been found in atmospheric samples taken in Toronto, Canada (Ref. 21, Pierce and Katz, 1976); Antwerp, Belgium (Ref. 3, Cautreels et al., 1977); and in southern Norway (Ref. 16, Lunde, 1976).

Bullhead catfish sampled from the Black River, Ohio, contained 42 ppb of anthraquinone (Ref. 26, Vassilaros et al., 1982). Anthraquinone also has been found in the surface wax of a perennial grass and in the heartwood of Quebrachia lorentzii (Ref. 2, Allebone et al., 1971), in the leaflets and pods of *Cassia angustifolia* (Ref. 23, Singh and Rao, 1982), and in cell suspension cultures of *Morinda citrifolia* (Ref. 32, Zenk et al., 1975). Ehrhardt et al. (Ref. 8, 1982) postulated that anthraquinone is formed in the atmosphere by the natural photooxidation of anthracene.

II. Chemical fate information—A. Persistence. Several environmental fate tests were performed to obtain information on the biodegradability of anthraquinone (Ref. 7, C-I-L, 1984). The reported results are summarized below.

1. BOD test. There was complete degradation of a 500 mg/L test solution in 24 days. The 5-day BOD showed 61 and 45 percent degradation using acclimated and unacclimated tests, respectively.

2. CO_2 evolution test. Test concentrations of 20 and 30 mg/L were used. After 38 days, the amount of CO_2 was equivalent to 83–96 percent of the theoretical CO_2 anticipated from the biodegradation of anthraquinone.

3. End-product test. At the end of the CO_2 evolution test, there were no discernible end products other than residual anthraquinone. More than 99 percent of the anthraquinone had degraded.

B. Rationale for chemical fate recommendations. All of the environmental fate tests were performed using test solutions in which the concentration of anthraquinone exceeded the reported solubility limit of anthraquinone. Therefore, the test data cannot be interpreted reliably. Based on expected releases of anthragumone to the aquatic environment, biodegradation tests should be performed at test concentrations not exceeding its water solubility limit. Prior to fate and effects testing, the water solubility of anthraquinone must be accurately quantified to properly design and conduct these tests.

III. Biological effects of concern to human health. Anthraquinone has been tested for health effects and is not being recommended for further testing at this time.

Rats fed anthraquinone in the diet for 4 days excreted untransformed anthraquinone in urine (Ref. 22, Sims, 1964).

The intraperitoneal LD_{50} of anthraquinone in the rat is 3,500 mg/kg (Ref. 27, Volodchenko, 1977). Anthraquinone fed to rats daily for 7 days inhibited the absorptive and excretory functions of the liver (Ref. 20, Pidemskii and Masenko, 1970). Repeated enteral injections of anthraquinone at one-fifth LD_{50} in experimental animals caused damage to the liver, kidneys, and peripheral blood (Ref. 28, Volodchenko and Labunskii, 1972). Anthraquinone administered orally (1,206 ppm in diet) for 17 months, or subcutaneously (single dose of 1,000 mg/kg), failed to elevate the incidence of tumors in two hybrid strains of mice (Ref. 11, Innes et al., 1969).

Anthragunone has been well tested in the Salmonella/microsome test system. Only one of nine studies showed any evidence of induction of mutagenicity. Positive results were obtained in the absence of metabolic activation in strains TA1537, TA1538, and TA98, indicating a frameshift type of mutagenesis. Yamaguchi (Ref. 31, 1982) found that anthragumone markedly decreased the mutagenicity of some known mutagens. Cesarone et al. (Ref. 5, 1982) observed an increased in vivo production of single-strand DNA breaks in the liver and kidneys of mice injected intraperitoneally with anthraquinone.

IV. Ecological effects of concern-A. Short-term effects. Static acute toxicity tests have been performed with several aquatic organisms (Ref. 7, C-I-L, 1984). The LC50s for Daphnia pulex and the fathead minnow were 46 and 2,650 mg/ L, respectively. Both levels greatly exceed the reported solubility limit. The report stated that the fish died from clogging of the gills with undissolved anthraquinone. Giddings (Ref. 10, 1979) reported that 1,4-anthraquinone was "essentially nontoxic to Selenastrum capricornutum." The researcher attributed this to its insolubility. The test concentration was reported as 100 percent saturation.

Chillingworth (Ref. 6, 1974) determined the toxicity of anthraquinone to fathead minnows and *S. capricornutum.* No effects on the fish were observed at 180 mg/L or on the algae at 10 mg/L. In screening studies using three species of fish, all fish died in less than 13 hours after exposure to an anthraquinone concentration of 10 ppm (Ref. 18, MacPhee and Ruelle, 1969).

In a seed germination test with the radish, *Raphanus sp.*, the 48-hour ED₅₀ was calculated to be 428,000 ppm. A 500 mg/L solution of anthraquinone applied to seedling wheat and soybean plants had no effect on shoot height and biomass, root biomass, and growth pathology (Ref. 7, C-I-L, 1984). Several laboratory tests were

Several laboratory tests were performed to investigate the potential ecological effects of using anthraquinone as a pulping additive (Ref. 7, C-I-L, 1984). Several tests were performed with bluegill and *Daphnia magna* using an effluent containing anthraquinone. Due to the high mortality in the control groups, the observed problems with solubility, and the fact that the anthraquinone concentrations were not measured, the data from these studies cannot be used to reliably access the toxic effect levels of anthraquinone.

B. Long-term effects. No information was found.

C. Bioconcentration. C-I-L (Ref. 7, 1984) reported the results of a 28-day bioconcentration test with the fathead minnow. Within 16 days of exposure to anthraquinone in water at a mean concentration of approximately 0.4 mg/ L, a steady-state concentration in the fish was reached. The bioconcentration factor (BCF) was 24. Eleven days after transfer to clean water, 66 percent of the accumulated residues had been eliminated from the fish. After 16 days, the residues were below detectable limits. In a 30-day bioconcentration study with bluegill and a 5-day study with D. magna, the resulting BCFs were approximately 50 and 100, respectively (Ref. 7, C-I-L, 1984).

D. Rationale for ecological effects recommendations. The toxicity tests that were reviewed were performed at test concentrations that exceed the reported water solubility level of anthraquinone. These data are inadequate to quantify the acute and potential chronic toxicity to aquatic organisms. These data indicate, however, that anthragumone may be very toxic to aquatic organisms. Since organisms were killed in the toxicity tests, anthraquinone may be toxic at its reported solubility limit of 0.05 mg/L. Flowthrough toxicity tests at concentrations not exceeding the measured solubility limit of anthraquinone should be performed. If these tests indicate toxicity at the solubility limit, chronic tests chould also be performed. The bioconcentration tests indicate that bioconcentration of anthraquinone is not expected to be significant.

References

(1) Akayama T, Koga M, Shinehara R, Kido A, Etoh S. 1989. Detection and identification of trace organic substances in the aquatic environment. J. UOEH 2(3):205–299.

(2) Allebone JE, Hamilton RJ, Bryce TA, Kelly W. 1971. Anthraquinone in plant surface waxes. Experientia 27(1):13–14.

(3) Cautreels W. Van Cauwenberghe K. Guzman LA. 1977. Companson between the organic fraction of suspended matter at a background and an urban station. Sci. Total Environ. 8(1):79–88.

(4) CEH. 1983. Chemical Economica Handbook. Section 300.70035. Stanford Research Institute, Menlo Park, CA: SRI International.

(5) Cesarone CF. Bolognesi C. Santi L. 1932. Evaluation of damage to DNA after in vivo exposure to different classes of chemicals. Arch. Toxicol. 5 (Suppl.):355–359. (6) Chillingworth MA. 1974. The toxicity of

(6) Chillingworth MA. 1974. The toxicity of aminoanthraquinone dyes to fish and algae. In: Dyea aild Environment. American Dye Manufacturers Institute. Inc.

(7) C-I-L, Inc. 1934. Unpublished information on the fate and effects of anthraquinone submitted by H.H. Holten, C-I-L, Inc. March 22, 1934.

(6) Ehrhardt M, Bouchertall F, Hopf HP.
1932. Aromatic ketones concentrated from
Boltic Sea water. Mar. Chem. 11:449–461.
(9) Games LM, Hites RA. 1977.

Composition, treatment efficiency, and environmental agailticance of dye menufacturary plant effluents. Anal. Chem. 49(9):1433-1449.

(10) Giddingo J.M. 1979. Acnte toxicity to Salanastrum capricomutum of aromatic compounds from cool conversion. Bull. Environ. Contam. Toxicol. 23:350–354.

(11) Inn25 JRM, Ulland BM, Valeno MG, Patrucelli L, Fishbein L, Hart ER, Pallotta AJ, Bates RR, Folk HL, Gart JJ, Klein M, Mitchell J, Paters J. 1959. Bioassay of pesticides and industrial chemicals for tumorizenicity in mice: A preliminary note. J. Natl. Cancer Inst. 42(0):1101–1114.

(12) Kirk-Othmer. 1978. Encyclopedia of Chemical Technology. 3rd ed. Vol. 2. New York: John Wiley & Sons. Inc. pp. 760–768.

(13) Kirk-Othmer. 1979. Encyclopedia of Chemical Technology, 3rd ed. Vol. 8. New York: John Wiley & Sons, Inc. pp. 138, 170– 171, 177–178.

(14) Kirk-Othmer. 1930. Encyclopedia of Chemical Technology, 3rd ed. Vol. 11. New York: John Wiley & Sons, Inc. p. 700.

(15) Kirk-Othmer. 1932. Encyclopedia of Chemical Technology, 3rd ed. Vol. 19. New York: John Wiley & Sons, Inc. pp. 317–318. 409, 497.

(10) Lunde G. 1978. Long-ronge certal transmission of organic micropollutants. Ambio 5(5-8):207-203.

(17) Lyman WJ, Reehl WF, Rosenblatt DH. 1932. Handbook of Chemical Property Estimation Methods, Chapter 1. New York: McGraw-Hill Book Co.

(18) MacFhee C, Ruelle R. 1969. Lethal effects of 1829 chemicals upon four species of fish from western North America. Forest, Wildlife and Range Experiment Station. Moscow, ID: University of Idaho.

(19) Meijers AP. Van der Leer RC. 1976. The occurrence of organic micropollutants in the nver Rhine and the nver Maas in 1974. Sci. Res. 10:557–674.

(20) Fidemskii EL, Masenko VP. 1970. Effect of some anti-inflammatory preparations on the absorption and excretory function of a normal liver and one affected by hepatitis. Tr Perm. Gos. Mcd. Inst. 93:325–328.

(21) Pierce RC, Katz M. 1978. Chromatographic isolation and spectral analysis of polycyclic quinones: Application of air pollution analysis. Environ. Sci. Technol. 10(1):45-51.

(22) Sims P. 1934. Metabolism of polycyclic compounds: 25. The metabolism of anthracene and some related compounds in rats. Biochem. J. 92:621-631.

(23) Singh P. Rao MM. 1932. Optimum stage of harvest of leaflets and pods of Senna

Cassia Angustifolia Vahl, in relation to yield of crude drug and anthraquinone. Indian J. Pharm. Sci. 44(1):12–13.

(24) USITC. 1980. U.S. International Trade Commission. Imports of Benzenoid Chemicals and Products, 1979. USITC Publ. No. 1083. Washington, DC: U.S. Govt. Printing Office.

(25) USITC. 1983. U.S. International Trade
Commission. Imports of Benzenoid Chemicals
and Products, 1982. USITC Publ. No. 1401.
Washington, DC: U.S. Govt. Printing Office.
(26) Vassilaros DL, Stoker PW, Eooth GM,
Lee ML. 1982. Capillary gas chromatographic

(26) Vassilaros DL, Stoker PW, Booth GM, Lee ML. 1982. Capillary gas chromatographic determination of polycyclic aromatic compounds in vertebrate fish tissue. Anal. Chem. 54:106–112.

(27) Volodchenko VA. 1977 Results of and iuture prospects for studying the toxicological characteristics of anthraquinone derivatives. Sig. Tr. Prob. Zabol. 21(12) 27–30.

(28) Volodchenko VA, Labunskii VV. 1972. Biological characteristics of the liaminoanthraquinone and anthraquinone somers viewed in a comparative aspect. Gig. Fr. Prof. Zabol. 16(11):44–45.

(29) Voss GP 1981. 9,10-Anthraquinone as in additive in chemical pulping. Paper Tech. nd. 22(4):125-130.

(30) Williams DT, Nestmann ER, LeBel GL, lenoit FM, Otson R. 1982. Determination of nutagenic potential and organic ontaminants of Great Lakes drinking water. hemosphere 11(3):263–276.
(31) Yamaguchi T. 1982. Reduction of

(31) Yamaguchi T. 1982. Reduction of nduced morality with biologically active unones through inhibition of metabolic ctivation. Agric. Biol. Chem. 46(9):2373-2375.

(32) Zenk MH, El-Shagi H, Schulte U. 1975. Inthraquinone production by cell suspension ultures of *Morinda citrifolia*. Planta Med. Suppl.):79–101.

2.2.b 2-Chloro-1,3-Butadiene Chloroprene).

Summary of recommended studies. It recommended that chloroprene be ssted for the following:

A. Chemical Fate:

Vater solubility

ersistence

B. Ecological Effects:

cute toxicity to sensitive life stages of fish, aquatic invertebrates, and algae

hysical and Chemical Information

CAS Number: 126-99-8. Synonym: Chloroprene. Structural Formula:

$CH_2 = C - CH = CH_2$

Empirical Formula: C_4H_5Cl , Molecular Weight: 88.5. Melting Point: -130 °C (Ref. 15, erschueren, 1983). Boiling Point: 58.4 °C (Ref. 15; erschueren, 1983). Vapor Pressure: 188 mmHg at 20 °C ef. 9, NIOSH, 1977). Specific Gravity: 0.9583 (Ref. 7, Irish, 63). Log Octanol/Water Partition Coefficient: 1.73 (estimated; Ref. 8, Lyman et al., 1982).

Description of Chemical: Colorless liquid.

Rationale for Recommendations

I. Exposure information—A. Production/use/disposal. No data were found on the current production volume of chloroprene. However, the total volume can be estimated from its use in the production of polychloroprene (neoprene) elastomers, which is the only significant use of chloroprene reported (Ref. 2, CEH, 1982). In 1983, approximately 254 million pounds of polychloroprene were produced in the United States (Ref. 6, Greek, 1984); the amount of chloroprene produced is expected to be similar. Approximately 33 percent of the production volume of polychloroprene is used in automotive belts and wire coverings; 25 percent, in nonautomotive wire coverings; and 25 percent, in the manufacture of adhesives.

B. Evidence for exposure. Preliminary data from the National Occupational Exposure Survey conducted during 1980–83 estimated that 6,405 workers were exposed to chloroprene in the workplace in 1980 (Ref. 10, NIOSH, 1984). It has been reported (Ref. 2, CEH, 1982) that chloroprene is shipped from the site of manufacture to a different plant for polymerization; thus, the potential exists for its release during handling and transport as well as during manufacturing and processing.

II. Chemical fate information—A. Transport. No information was found. B. Persistence. No information was found.

C. Rationale for chemical fate recommendations. On the basis of its vapor pressure, the compound is expected to partition to the atmosphere. However, water solubility data are needed to confirm this conclusion and to provide information on the rate and extent of its partitioning to the atmosphere and other environmental media. This information is also needed to determine what types of persistence testing (biological, chemical, or photochemical) are most pertinent to an assessment of environmental exposure.

III. Biological effects of concern to human health. Although chloroprene is a high production chemical that is structurally related to the carcinogens 1,3-butadiene and vinyl chloride, sufficient testing to elucidate its potential health effects of concern to humans either has been conducted, is underway, or is planned. Thus, further testing for health effects is not being recommended at this time. The acute oral LD₅₀ for chloroprene was found to be 260 mg/kg body weight in mice and 251 mg/kg body weight in rats. In an inhalation study, the approximate lethal concentration of the chemical in rats following a 4-hour exposure was 2,300 ppm (Ref. 3, Clary, 1977).

Chloroprene has been shown to be genotoxic in a number of test systems, including the *Salmonella* microsomal assay (Ref. 1, Bartsch et al., 1979) and the *Drosophila* sex-linked recessive lethal mutation system (Ref. 16, Vogel, 1979). It was shown to induce chromosomal aberrations in human cells (Refs. 13 and 17, Sanotskii, 1976; Zhurkov et al., 1977). It was negative in V79 Chinese hamster cells for inducing resistence to 8-azaguanine or ouabain (Ref. 4, Drevon and Kuroki, 1979).

No published studies on the metabolism or toxicokinetics of chloroprene were found.

Several carcinogenicity studies have been published in the literature (Refs. 18, 20, 19, and 12, Zil'fyan and Fichidzhyan, 1972; Zil'fyan et al., 1975; Zil'fyan et al., 1977; Ponomarkov and Tomatis, 1980); however, they were of insufficient duration to evaluate chloroprene's carcinogenic potential. The Joint Industry Group on Chloroprene sponsored a chronic inhalation study of the chemical in Wistar rats and Syrian golden hamsters (Ref. 14, Trochimowicz, 1984). Exposure levels were targeted at 0, 10, and 50 ppm chloroprene for 6 hours/day, 5 days/ week. The rats were exposed for 24 months; the hamsters, for 18 months. No evidence of chloroprene-induced carcinogenicity was found in either species.

As part of its testing initiative on 1,3butadiene, an animal carcinogen, the National Toxicology Program is testing chloroprene for a number of toxicological endpoints. The chemcial has been selected for an indepth toxicological evaluation in 14- and 90day studies. The 90-day studies will include sperm morphology and vaginal cytology evaluation, while the 14-day studies will include micronuclei evaluations and in vivo cytogenetics testing. Chloroprene will also be tested for carcinogenicity by inhalation. Toxicokinetic and metabolism studies are also planned using intravenous injections over a wide range of doses. Finally, inhalation teratology studies of the chemical in rats and mice are planned, as are fertility assessment studies of chloroprene in mice (Ref. 11, NTP, 1984.

IV Ecological effects of concern—A. Short-term effects. The 96-hour LC₅₀ of chloroprene with bluegill was 245 ppm (Ref. 5, Dupont, 1984). The test was a flowthrough test with the LC_{50} based on nominal test concentrations. The 7-day EC_{50} with the alga, *Navicula seminulum* was 3,800 ppm. This EC_{50} was also based on nominal test concentrations.

B. *Long-term effects*. No information was found.

C. *Bioconcentration*. Based on an estimated low log octanol/water partition coefficient of 1.73, substantial bioconcentration is not expected.

D. Rationale for ecological effects recommendations. Based on its estimated large production volume, environmental releases of chloroprene are likely. The available data on its acute toxicity to fish and algae are not sufficient to reliably assess the acute and chronic effect levels of chloroprene to aquatic organisms. The chloroprene concentrations in the test solutions from the reported tests were not measures; the LC50s were based on nominal concentrations. The high vapor pressure of chloroprene suggests that the actual concentrations of choroprene in the test solutions may be considerably less than the nominal concentrations reported. Chloroprene may be more toxic than these data indicate. To reliably estimate the effects of the compound, acute toxicity tests in which the test concentrations are measured should be performed with sensitive life stages of fish, aquatic invertebrates, and algae.

References

(1) Bartsch HC, Malaveille C, Barbin A, Planche G. 1979. Mutagenic and alkylating metabolites of haloethylenes, chlorobutadienes, and dichlorobutenes produced by rodent or human liver tissues: Evidence for oxirane formation by P450linked microsomal mono-oxygenases. Arch. Toxicol. 41:249-277.

(2) CEH. 1982. Chemical Economics Handbook. Standford Research Institutes. Menlo Park, CA: SRI International. Sections 300.5802 J, K.

(3) Clary JJ. 1977. Toxicity of chloroprene, 1,
3-dichlorobutene-2, and 1, 4-dichlorobutene-2.
Environ. Health Perspect. 21:269–274.
(4) Drevon C, Kuroki T. 1979. Mutagenicity

(4) Drevon C, Kuroki T. 1979. Mutagenicity of vinyl chloride, vinylidene chloride and chloroprene in V79 Chinese hamster cells. Mutat. Res. 67:173–182.

(5) DuPont. 1984. Unpublished information on 2-chloro-1, 3-butadiene submitted by M. H. Cristman, E. I. du Pont. De Nemours & Co. October 17, 1984.

(6) Greek BF. 1984. Elastomers finally recover growth. Chem. Eng. News, April 30, 1984, pp. 35–56.

(7) Îrish DD. 1963. Halogenated Hydrocarbons: I. Aliphatic. In: Patty, F. A., ed. Industrial Hygiene and Toxicology, 2nd rev. ed. New York: Interscience Publishers, Vol. II. pp. 1319–1321.

(8) Lyman WJ, Reehl WF, Rosenblatt DH. 1982. Handbook of Chemical Property Estimation Methods. New York: McGraw-Hill Book Co.

(9) NIOSH. 1977. Criteria for a recommended standard * * * Occupational exposure to chloroprene. U.S. Dept. of Health, Education, and Welfare. National Institute for Occupational Safety and Health. DHEW (NIOSH) Publ. No. 79–210. 176 pp.

(10) NIOSH. 1984. National Occupational Exposure Survey, 1980–83. Cincinnati, OH: National Institute for Occupational Safety and Health.

(11) NTP. 1984. National Toxicology Program. Minutes of the NTP Toxicology Design Committee Meeting. May 15, 1984.

(12) Ponomarkov V, Tomatis L. 1930. Longterm testing of vinylidene chloride and chloroprene for carcinogenicity in rats. Oncology 37:138–141.

(13) Sanotskii IV. 1976. Aspects of the toxicology of chloroprene: Immediate and long-term effects. Environ. Health Perspect. 17:85–93.

(14) Trochimowicz H. 1934. Personal communication from H. Trochimowicz, E. I. du Pont de Nemours & Co. September 28, 1984.

(15) Verschueren K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. New York: Van Nostrand Reinhold Co. p. 384.

 (16) Vogel E. 1979. Mutagenicity of chloroprene, 1-chloro-1,3-trans-butadiene, 1, 4-dichlorobutene-2, and 1,4-dichloro-2, 3epoxybutane in *Drosophila melanegaster*. Mutata. Res. 67:377–381.

Mutata. Res. 67:377–381. (17) Zhurkov VS, Fichidzhyan BS, Batikyan GG, Arutyunyan RM, Zil'fyan VN. 1977. Cytogenetic examination of persons in contact with chloroprene udner industrial conditions. Tsitol. Genet. 11:210–212. (18) Zil'fyan VN, Fichidzhyan BS. 1972.

(18) Zil'fyan VN, Fichidzhyan BS. 1972. Effect of chloroprene on development of Crocker's murne sarcoma. In: Proceedings of a Scientific Conference Devoted to the 59 Years of the Universities of USSR and to the 25 Years of the Armenian Institute of Roentgenology and Oncology, 1972. Yerevan, USSR. pp. 105–106 (Chem. Abst. 61:22033).

(19) Zil'Fyan VN, Fichidzhyan BS,
Garibyan DK, Pogosova AM. 1977.
Experimental study of chloroprene for carcinogenicity. Vopr. Onkol. 23:61–65.
(20) Zil'fyan VN, Fichidzhyan BS, Pogosova

(20) Zil'fyan VN, Fichidzhyan BS, Pogosova AM. 1975. Results of testing chloroprene for carcinogenicity. Zh. Eksp. Klin. Med. 15:54– 57.

2.2.c Cumene.

Summary of recommended studies. It is recommended that cumene be tested for the following:

A. Health Effects:

Short-term genotoxicity Chronic effects including oncogenicity Teratogenicity and reproductive toxicity B. Ecological Effects:

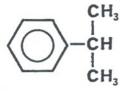
Acute and chronic toxicity to estuarine and freshwater fish and invertebrates

Physical and Chemical Information

CAS Number: 98–82–8. Synonyms: Isopropylbenzene: 2-Phenylpropane: Benzene, (1-

methylethyl)- (9 CI).

Structural Formula:



Empirical Formula: C₈H₁₂. Molecular Weight: 120.19. Melting Point: -96.0 °C. Boiling Point: 152.7 °C.

Vapor Pressure: 3.2 mmHg at 20 °C. (Ref. 49, Verschueren, 1977).

Specific Gravity: 0.862 at 20 °C. Solubility in Water: 50 mg/L at 20 °C. (Ref. 18, Hutchinson et al., 1980).

Log Octanol/Water Partition Coefficient: 3.51 (Ref. 18, Hutchinson et al., 1980); 3.66 (Ref. 35, Rogerson et al., 1983).

Description of Chemical: Colorless, mobile liquid with a sharp, penetrating, aromatic odor.

Rationale for Recommendations

I. Exposure information—A. Production. The total annual production capacity of the 11 domestic producers of cumene as of January 1, 1983, was estimated to be 4.7 billion pounds (Ref. 41, SRI, 1933). The 1983 U.S. production of cumene was reported to be 3.30 billion pounds. On this basis, cumene was ranked 31 of the top 50 chemical products for 1983 (Ref. 6, C&EN, 1984). Domestic production of cumene in 1932 was reported to be 2.74 billion pounds, down from the 3.92 billion pounds produced in 1979 (Ref. 46, USITC, 1983). The public portion of the TSCA Inventory listed 21 companies at 25 sites with a 1977 aggregate production/ importation range of 1.5 to 6.6 billion pounds (Ref. 10, EPA, 1984).

Importation of cumene has declined in recent years but is expected to remain at the 1980–81 level of about 300 million pounds over the next 5 years. Cumene exports in 1980 amounted to 63 million pounds (Ref. 7, CEH, 1982).

Cumene is present in a number of crude oils and in refinery streams, but all commercial (high-purity) cumene is produced by the alkylation of benzene with chemical-grade propylene under elevated temperature and pressure, most commonly in the presence of a solid phosphoric acid catalyst (Refs. 25 and 21, Lowenheim and Moran, 1975; Kirk-Othmer, 1979). Cumene also has been reported to be produced by distillation from coal-tar naphtha fractions or from petroleum (Ref. 17, Hawley, 1977).

B. Use. More than 98 percent of domestically produced cumene is used

in the manufacture of phenol and acetone; most of the remaining 2 percent is exported. Although more than half of the cumene utilized in the manufacture of phenol and acetone is used captively, a substantial portion is sold to other domestic producers of the two compounds (Ref. 7, CEH, 1982). More than 98 percent of domestically manufactured phenol and approximately 70 percent of domestically synthesized acetone are produced from cumene (Refs. 21 and 7, Kirk-Othmer, 1979, 1982; CEH, 1982).

A small amount of cumene is used in the production of alpha-methylstyrene (Refs. 7 and 21, CEH, 1982; Kirk/Othmer, 1979), which is used as a copolymer in acrylonitrile-butadiene-styrene plastics and which is also used in musk oil fragrances and shoe soles (Ref. 7, CEH, 1982). Minor uses of cumene are as a solvent (Refs. 7 and 17, CEH, 1982; Hawley, 1977) and as a high-octane component in aviation gasolines (Ref. 21, Kirk-Othmer, 1979).

C. Evidence for exposure. The OSHA 8-hour time-weighted average (TWA) permissible exposure limit for cumene in the workplace is 50 ppm (245 mg/m³) (Ref. 33, OSHA, 1978); the ACCIH 8-hour TWA threshold limit value is the same (Ref. 1, ACGIH, 1983), and was selected to prevent induction of narcosis. The ACGIH 15-minute short-term exposure limit for the skin is 75 ppm (365 mg/m³) (Ref. 1, ACGIH, 1983).

The National Occupational Hazard Survey, conducted by NIOSH during the years 1972-74, estimated that 863 workers were exposed to cumene in the workplace during that time period (Ref. 30, NIOSH, 1984.

One manufacturer of cumene recently reported that 20 workers are potentially exposed to the chemical. The cumene concentration in 102 8-hour TWA breathing zone samples ranged from less than 1 to 3 ppm; the concentration in the great majority of the samples was less than 1 ppm (Ref. 44, Texaco, 1984).

A second manufacturer reported that 12 of its process personnel are involved in the production of cumene. The manufacturing process is a closed system; the sewer system is closed and vented to a flare stack. A closed system also is used for sampling. Cumene vapors at levels of less than or equal to 0.5 ppm have been detected through area sampling. Breathing zone samples of process and maintenance personnel have been no higher than 0.14 ppm cumene vapor (Ref. 23, Koch, 1984).

A third manufacturer reported that 8– 10 workers are involved in the production of cumene. The cumene units are closed systems without any waste streams, except spent catalyst. The cumene concentration in 730 12- or 8hour TWA personal samples ranged from below the detection limit to 20 ppm. The average for the samples was below 0.08 ppm (Ref. 16, Gulf, 1984).

Cumene has been detected in ambient air samples of the vulcanization area of a shoe-sole factory at concentrations of $60-250 \text{ g/m}^3$ It also has been measured in the vulcanization and extrusion areas of the tire retreading factory at concentrations of 2–200 g/m³ and 0–10 g/m³, respectively (Ref. 8, Cocheo et al., 1983).

Cumene has been detected in aqueous effluents from a petroleum refinery and from a textile finishing and dyeing plant (Refs. 40 and 15, Snider and Manning, 1982; Gordon and Gordon, 1981). It has been detected at part-per-billion (ppb) levels in groundwater samples taken at progressive distances downgradient from an aviation fuel spill (Ref. 43, Tester and Harker, 1981]. The Chemical also has been found at ppb levels in groundwater samples taken near two underground gasification sites in northeastern Wyoming (Ref. 42, Stuermer et al., 1982). Cumene has been measured at ppb levels in the ambient air samples from a number of localities (Refs. 2, 5, 27, 24, and 45, Arnts and Meeks, 1981; Bos et al., 1977; Miller and Alkezweeny, 1980; Lonneman et al., 1968; Tsanı-Bazaca et al., 1982).

II. Chemical fate information. Cumene is expected to partition among soil, sediment, water, and air. Cumene in natural waters exposed to light of wavelength greater than 290 nm for 5 days was oxidized to the extent of 1.2– 9.2 percent, depending on the water source and initial cumene concentration (Ref. 26, Mill et al., 1978). On the basis of its reactivity with atmospheric hydroxyl radicals, cumene was estimated to have a half-life of 2.4–24 hours in air (Ref. 9, Darnall et al., 1976).

Cumene is expected to biodegrade readily. The biological oxidation demand of cumene in sewage-seeded freshwater was 40, 62, 63, and 70 percent in 5, 10, 15, and 20 days, respectively (Ref. 31, OHMTADS). A number of strains of the Pseudomonas genus assimilate cumene (Ref. 32, Omori et al., 1975), and Pseudomonas aeruginosa has been shown to hydroxylate one of the chemical's methyl groups (Ref. 48, Van der Linden and Van Ravenswaay Claasen, 1971). At 10 °C in clean groundwater, cumene was completely degraded by microbes within 11 days (Ref. 20, Kappeler and Wuhrmann, 1978).

On the basis of its moderately high log P, cumene is expected to have a moderate potential to bioconcentrate. Actual bioconcentration will depend, however, on the rate of metabolism of the compound and the duration of exposure.

III. Biological effects of concern to human health—A. Acute toxicity. The acute toxicity of cumene is summarized in the following Table 4. Cumene was found to be a primary skin and eye irritant in rabbits (Ref. 51, Wolf et al., 1956).

B. Metabolism and toxicokinetic studies. Cumene has been observed to be absorbed via several routes. Ninety percent of an oral dose of cumene in rabbits was recovered as metabolites in the urine (Ref. 34, Robinson et al., 1955). Approximately 50 percent of cumene vapors inhaled by human volunteers was retained following exposure to the chemical at levels of 240–720 mg/m³ (Ref. 36, Senczuk and Litewka, 1976). Cumene was absorbed through the shaved skin of rats (Ref. 47, Valette and Cavier, 1954).

In rats exposed for 2 months by inhalation to cumene at levels of 25 mg/ L, cumene was detected in a number of tissues 24 hours following cessation of the last exposure. The highest levels were observed in the thyroid and adrenal glands. Rats were exposed by inhalation to the same concentration of cumene for 6 months. Seventy-two hours after the last exposure, cumene was found to distribute mainly to endocrine organs, central nervous system components, bone marrow, spleen, and liver (Ref. 16, Fabre et al., 1955.) BILLING CODE 6560-50-M Table 4 -- Acute Toxicity of Cumene in Laboratory Animals

Anımal	Route of Admin.	Test Group (No.)	Dose	Effects	Peference
Mouse	Inh	8M, 8F for each concentration	LC ₅₀ . 10 mg/L (about 2,000 ppm) for 7 hr	Slight incoordination to deep narcoois (including analgeoia, unconsciousness, complete relaxation, and loss of reflexes) and death	Warner et al. (Faf. 50, 1944)
		4	2,490 pph for up to 30 min exposure	50% decrease in respiratory rate due to sensory irritation of upper respiratory tract	Melsen and Alarie (Fof. 29, 1982)
		Unspecified	20 tg/L; duration of exposure unspecified	Minimal concentration to cause prostration	Lohmann and Flury (1943, cited in Ref. 13, Gerarde, 1960)
			25 Eg/L; duration of exposure unspecified	Ninical concentration to cause loss of reflexes	•
Rat	Oral	5 for each concentration	LD ₅₀ : 2.91 g/i:g	-	Smyth et al. (Raf. 39, 1951)
		Unspecified	LD ₅₀ . 1.4 g/kg	Some irritation to the stomach and intestines	Wolf et al. (Ref. 51, 1956)
		10	5 ml/kg	6 Died	Gerarde (Eef. 13, 1960)
		5 for each concentration	LD ₅₀ . 2,700 mg/kg	Weight loss, weakness, orular discharge, collagse, and death; hemorrhagic lungs, liver discoloration, and GI tract inflammation in decedents	Monsanto (Ref. 28, 1934)
	Inh	Unspecified	"Saturated vapor"	The maximum duration of exposure for no deaths was 1 hour.	Smyth et al. (Ref. 39 1951)
		6	8,000 ppm, for 4 hr	4 Diel	
Rabbit	Skin	5 for each concentration	LD ₅₀ . 12.3 nl/kg	-	
		Unspecified	LD ₅₀ . >3,160 ⊡g <i>/i</i> :g	Weight loss, weakness, collagse, and death; hemorrhagic lungs, liver, discoloration, enlarged gallbladder, darkened kidneys and spleen, and GI tract inflammation in decedents	Moncanto (Ref. 23, 1934)

BILLING CODE 6560-50-C

Following intravenous administration of cumene to rats, the highest concentrations were found in adipose tissues, the brain, adrenal glands, heart, and lungs. Two to three hours after oral administration of the chemical to rats, maximum concentrations were measured in adipose tissues with levels 15–20 times higher than those seen in the adrenal glands and liver (Ref. 14, Gorban et al., 1978).

In biotransformation studies of cumene in rabbits, the animals were administered 2 ml of the chemical orally and 24-hour urine samples were collected for analysis of metabolites. About 40 percent of the dose was excreted as the glucuronide of 2-phenyl-2-propanol, 25 percent as the glucuronide of 2-phenyl-1-1-propanol, and 25 percent as the ester-glucuronide of 2-phenylpropionic acid (Ref. 34, Robinson et al., 1955).

In a biotransformation study in rats, the animals received a single oral dose of cumene (100 mg/kg) and 48-hour urine samples were collected following dosing. 2-Phenyl-2-propanol and 2phenyl-1-propanol were both observed in the urine at unspecified levels (Ref. 3, Bakke and Scheline, 1970).

2-Phenyl-2-propanol also was excreted in the urine by human subjects exposed to cumene by inhalation. The levels of the metabolite in the urine were proportional to the atmospheric concentration of cumene during exposure (Ref. 36, Senczuk and Litewka, 1976).

C. Teratogenicity/embryotoxicity. Following inhalation exposure of female rats for 4 months to maximum permissible concentrations of cumene, embryonal mortality increased from 7.5 to 39.3 percent; the frequency of teratogenic effects increased from 3.0 to 11.0 percent (Ref. 37, Serebrennikov and Ogleznew, 1978). No other information on these studies was found.

D. Genotoxicity. When studied by several investigators, cumene was negative in the Salmonella microsomal assay using both the spot test and the plate incorporation technique (Refs. 12, 28, and 38, Florin et al., 1980); Monsanto, 1984; Simmon et al., 1977). In the Saccharomyces cerevisiae D3 test system, the chemical also was not mutagenic (Ref. 38, Simmon et al., 1977).

E. Subchronic toxicity. Cumene was administered in olive oil to female rats by gavage daily, 5 days a week for 6 months. At a dose of 154 mg/kg, no ill effects were observed on the basis of gross appearance, final body and organ weights, periodic blood counts, blood urea nitrogen, histopathologic examinations, and bone marrow counts. An increase in kidney weight was seen at a dose of 462 mg/kg/day (Ref. 51, Wolf et al., 1956).

No significant changes were noted in the lungs, liver, and kidneys of rats exposed to 500 ppm cumene for 8 hours/ day, 6 days/week for 5 months (Ref. 11, Fabre et al., 1955).

Rats, guinea pigs, monkeys, and dogs were exposed to cumene vapors either repeatedly for 8 hours a day, 5 days a week, for a total of 30 exposures over a 6-week penod, or continuously for 90– 127 days. There was no difference in body weight and hematologic data between the treated animals and controls, and histopathologic examinations of the heart, liver, lungs, spleen, and kidneys were essentially negative (Ref. 19, Jenkins et al., 1970).

F Chronic toxicity/carcinogenicity. No studies of the chronic toxicity of carcinogenicity of cumene were found.

G. Rationale for health effects recommendations. The potential exists for occupational and environmental exposure to cumene. Cumene has been detected in the ambient air of several localities; it also has been found in effluents from a petroleum refinery and an industrial plant and in groundwater samples. Although cumene was not mutagenci in either the Salmonella microsomal assay or the S. cerevisiae D3 test system, a more complete evaluation of its genotoxic potential should be performed. Data are lacking on chronic effects including oncogenicity. A limited productive toxicity study in female rats indicated an icrease in teratogenic effects after inhalation exposure to cumene. Accordingly, a battery of short-term genotoxicity assays (excluding the Salmonella assay), a chronic effects study including oncogenicity, and teratogenicity and reproductive toxicity studies are recommended for cumene.

IV Ecological effects of concern. In a study on the effects of cumene on the green algae Chlamydomonas angulosa and Chlorella vulgaris, a 50 percent reduction in photosynthetic activity was noted at concentrations of 8.8 and 21.2 ppm, respectively (Ref. 18, Hutchinson et al., 1980). In 18- to 24- hour tests with the freshwater ciliates Colpidium colpoda and Tetrahymena elliotti, acute toxicity thresholds of 12 ppb and 3 ppm, respectively, were determined (Ref. 35, Rogerson et al., 1983). Bobra et al. (Ref. 4, 1983) reported that the 48-hour LC 50 for the water flea Daphnia magna was 0.6 ppm.

V Rationale for ecological effects recommendations. Cumene is one the top 50 chemicals produced in the United States and is released to the environment from a number of sources, as evidenced by detection of the compound in ambient air and water samples. This exposure potential, coupled with the demonstrated toxicity of the compound to invertebrates at low levels and the lack of test data on fish, indicate that further investigation of the toxicity of the compound to estuarine and freshwater fish and invertebrates should be made.

References

(1) ACGIH. 1983 TLV's—Threshold Limit Values for Chemical Substances and Physical Agents in the Work Environment with Intended Changes for 1983–84. Cincinnati, OH: American Conference of Governmental Industrial Hygienists. p. 10.

(2) Arnts RR, Meeks SA. 1981. Biogenic hydrocarbon contribution to the ambient air of selected areas. Atmos. Environ. 15(9): 1643–1651.

(3) Bakke OM, Scheline RR. 1970. Hydroxylation of aromatic hydrocarbons in the rat. Toxicol. Appl. Pharmacol. 18:691-700.

(4) Bobra AM, Shin WY, Mackay D. 1983. A predictive correlation for the acute toxicity of hydrocarbons and chlorinated hydrocarbons to the water flea (*Daphnia magna*). Chemosphere 12(9/10):1121-1129.

(5) Bos R, Guicherit R, Hoogeveen A. 1977 Distribution of some hydrocarbons in ambient air near Elft and the influence on the formation of secondary air pollutants. Sci. Total Environ. 7(3):269–281.

(6) C&EN. 1984. C&EN's top 50 chemical products & producers. Chem. Eng. News. May 7, 1984. pp. 7–10.

(7) CEH. 1982. Chemical Economics Handbook. Stanford Research Institute, Menlo Park, CA: SRI International. Sections 638.5030A-.5031B.

(8) Cocheo V, Bellomo ML, Bombi GG. 1983. Rubber manufacture: Sampling and identification of volatile pollutants. Am. Ind. Hyg. Assoc. J. 44(7):521–527

(9) Darnall KR, Lloyd AC, Winer AM, Pitts JN Jr. 1976. Reactivity scale for atmospheric hydrocarbons based on reaction with hydroxyl radical. Environ. Sci. Technol. 10(7):692–696.

(10) EPA. 1984. TSCA Chemical Substances Inventory (public portion). Washington, DC: Environmental Protection Agency.

(11) Fabre R, Truhaut R, Bernuchon J, Louisillier F. 1955. Toxicological studies on solvent substitutes for benzene. III. Studies on isopropylbenzene or cumene. Arch. Mal. Prof. 16:285–299.

(12) Florin I, Rutberg L, Curvall M, Enzell CR. 1980. Screening of tobacco smoke constituents for mutagenicity using the Ames test. Toxicology 15:219–232.

(13) Gerarde HW. 1960. Toxicology and Biochemistry of Aromatic Hydrocarbons. Elsevier Monographs. Amsterdam, The Netherlands: Elsevier Publishing Co.

(14) Gorban GM, Solomin GI, Bizin YP, Sopikov NF Tikhonova GP, Zinozev VM, Pilipyuk ZI, Gorshunova AI, Chukhno EI. 1978. Study of the toxicity and determination of the threshold of the acute inhalation effect of isopropyl benzene. [In Russian]. Gig. Saint. 10:113. (Toxline Abstract). (15) Gordon AW, Gordon M. 1981. Analysis of volatile organic compounds in a textile finishing plant effluent. Trans. Ky. Acad. Sci. 42(3-4):149-157.

(16) Gulf. 1984. Unpublished information on cumene submitted by J.F. Dey, Gulf Oil Products Co. July 2, 1984.

(17) Hawley GG. 1977. The Condensed Chemical Dictionary. 9th ed. New York: Van Nostrand Reinhold Co. p. 241.

(18) Hutchinson TC, Hellebust JA, Tam D, Mackay D, Mascarenhas, RA, Shiu WY. 1980. The correlation of the toxicity to algae of hydrocarbons and halogenated hydrocarbons with their physical-chemical properties. Environ. Sci. Res. 16:577–586.

(19) Jenkins LJ Jr., Jones R.A, Siegel J. 1970. Long-term inhalation screening studies of benzene, toluene, o-xylene, and cumene on experimental animals. Toxicol. Appl. Pharmacol. 16(3):818–823.

(20) Kappeler TH, Wuhrmann K. 1978. Microbial degradation of the water-soluble fraction of gas oil. II. Bioassays with pure strains. Water Res. 12:335–342.

(21) Kirk-Othmer, 1979. Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed. Vol. 7. New York: John Wiley & Sons. pp. 286–290

(22) Kirk-Othmer. 1982. Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed. Vol. 17. New York: John Wiley & Sons. pp. 374–375.

(23) Koch. 1984. Unpublished information on cumene submitted by D.F. Janes, Koch Refining Co. April 6, 1984.

(24) Lonnema WA. Beller TA, Altshuller AP. 1968. Aromatic hydrocarbons in the atmosphere of the Los Angeles Basin. Environ. Sci. Technol. 2(11):1017-1020.

(25) Lowenheim FA, Moran MK. 1975. Faith, Keyes and Clark's Industrial Chemicals. 4th ed. New York: John Wiley & Sons. pp. 294–297.

(26) Mill T. Richardson H. Hendry DG. 1978. Oxidation of organic compounds in aquatic systems: The free radical oxidation of cumene. Aquatic Pollutants: Transformation and Biological Effects. Elmsford, NY: Pergamon Press. pp. 223–236. (Abstract).

(27) Miller DF, Alkezweeny AJ. 1980. Aerosol formation in urban plumes over Lake

Michigan. Ann. N.Y. Acad. Sci. 338:219–232. (28) Monsanto. 1984. Unpublished information on cumene submitted by A.W.

Thompson, Monsanto Co. April 27, 1984. (29) Nielsen GD, Alarie Y. 1982. Sensory

irritation, pulmonary irritation, and respiratory stimulation by airborne benzene and alkylbenzenes: Prediction of safe industrial levels and correlation with their thermodynamic properties. Toxicol. Appl. Pharmacol. 65:459–477.

(30) NIOSH. 1984. National Occupational Hazard Survey. Cincinnati, OH: National Institute for Occupational Safety and Health.

(31) OHMTADS. Oil and Hazardous Materials Technical Assistance Data System. Accession No. 7216655.

(32) Omori T, Jigami Y, Minoda Y. 1975. Isolation, identification and substrate assimilation specificity of some aromatic hydrocarbon utilizing bacteria. Agric. Biol. Chem. 39(9):1775–1779.

(33) OSHA. 1978. OSHA Safety and Health Standards for General Industry. 29 CFR 1910. Subpart Z. U.S. Department of Labor. Occupational Safety and Health Administration. Washington, DC: U.S. Govt. Printing Office. OSHA Publ. No. 2260. p. 541.

 (34) Robinson D, Smith JN, Williams RT.
 1935. Studies in detoxication. 69. The metabolism of alkylbenzenes: Isopropylbenzene (cumene) and derivatives of hydratropic acid. Biochem. J. 59:153–159.
 (35) Rogerson A, Shiu WY, Huang GL,

 (35) Rogerson A, Shiu V/Y, Huang GL,
 Mackay D, Berger J. 1903. Determination and interpretation of hydrocarbon toxicity to ciliated protozoa. Aquat. Texicol. 3:215–220,
 (36) Senczuk W, Litewha B. 1970.

Absorption of cumene through the respiratory tract and excretion of dimethylphenylcarbinol in urine, Brit, J. Ind. Med. 33:100–105.

(37) Serebrennikov OA, Ogleznev GA. 1978. Developmental anomalies in the mother-fetus system following exposure to petrochemical products. Deposited Doc; ISS VINITI. 2007– 78; 151–1. (Abstract).

(38) Simmon VF, Kauhanen K, Tardiff RG. 1977. Mutagenic activity of chemicala identified in drinking water. Dev. Toxicol. Environ. Sci. 2:249–258.

(39) Smyth HF, Carpenter CP, Weil CS. 1951. Range-finding toxicity data: List IV. AMA Arch. Ind. Hyg. Occup. Med. 4:119–122.

(40) Snider EH, Manning FS. 1892. A survey of pollutant emission levels in wastewaters and residues from the petroleum refining industry. Environ. Internat. 7:037-203.

(41) SRL 1933. Directory of Chemical Producers, USA, 1983. Stanford Recearch Institute, Menlo Park, CA: SRI International. pp. 518–519.

(42) Stuermer DH, Ng DJ, Merris CJ. 1932. Organic contaminants in groundwater near an underground coal gasification site in northeastern Wyoming. Environ. Sci. Technol. 16:582–537.

(43) Tester DJ, Harker RJ. 1931. Groundwater pollution investigations in the Great Ouse basin. Water Follut. Control 80(5):614–631.

(44) Texaco. 1984. Published and unpublished information on cumene submitted by R.T. Richardo, Texaco, Inc. May 14, 1984.

(45) Tsani-Bazaca E, McIntyra A, Lester J, Perry R. 1982. Ambient concentrations and correlations of hydrocarbons and halocarbons in the vicinity of an airport. Chemosphere 11(1):11–23.

(46) USITC. 1983. U.S. International Trade Commission. Synthetic Organic Chemicale, U.S. Production and Sales, 1932. Publ. No. 1422. Washington, DC: U.S. Govt. Printing Office. pp. 27, 37.

(47) Valette G, Cavier R. 1934. Percutaneous absorption and chemical structure. The case of hydrocarbons, of alcohols and of esters. [In French]. Arch. Int. Pharmacol. Ther. 97:232–240.

(48) Van der Linden AC, Van Ravenswaay Claasen JC. 1971. Hydrophobic enzymes in hydrocarben degradation. Lipids 6(7):437–443.

(49) Verschueren K. 1977. Handbook of Environmental Data on Organic Chemicala-New York: Van Nostrand Reinhold Co. pp. 410-411.

(50) Werner HW, Dunn RC, Von Oatlingen WF. 1944. The acute effects of cumene vapors in mice. J. Ind. Hyg. Toxicol. 26:264-253. (51) Welf MA, Rowe VK, McCollister DD, Hollingworth RL, Olyen F. 1955. Toxicological studics of certain allvylated banzenes and banzene. AMA Arch. Ind. Health 14:337–333.

2.2.d Mercaptobenzothiazole. Summary of recommended studies. It

is recommended that

mercaptobenzothazole be tested for the following:

A. Chemical Fate:

Dissociation constant

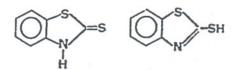
Persistence in water and soil Leaching/migration

B. Environmental Effects:

Acute and chronic toxicity to fish, aquatic invertebrates and plants, and terrectrial plants

Physical and Chemical Information

CAS Number: 149–30–4. Synonyms: MBT; 2(3H)-Benzothiazolethione; Thiotax². Structural Formula:



Empirical Formula: C7H2NS2.

Molecular Weight: 167.

Melting Point: 180.2–181.7 °C (REF. 10, Merck, 1976).

Vapor Pressure: $<1.9 \times 10^{-6}$ torr at 25 °C. (Ref. 13, Monsanto, 1934).

Specific Gravity: 1.42 (Ref. 10, Merck, 1976).

Water Solubility: 51 ppm at pH 5. (25 °C): 118 ppm at pH 7, (25 °C); 909 ppm at pH 9, (25 °C) (Ref. 13, Monsanto, 1934).

Log Octanol/Water Partition

Coefficient: 2.42 (Ref. 13, Monsanto, 1984).

Description of Chemical: Yellow, monoclinic needles or leaflets.

Rationale for Recommendations

I. Exposure information—A. Production/use/disposal. The 1931 production volume of mercaptobenzothiazole (MBT) was reported to be 2,323,000 pounds (Ref. 19, USITC, 1932). Its major use is as a rubber vulcanization accelerator and antioxidant. It is also used as a corrosion inhibitor in cutting oils, an additive in greases, a fungicide, and as a chemical intermediate in the manufacture of other rubber processing chemicals (Refs. 17, 10, 7, and 16, Uniroyal, 1984; Merck, 1976; Hawley, 1931; Santodonato et al., 1976).

The production volume of the sodium salt of mercaptobenzothiazole (NaMBT) was estimated to be 40 million pounds: 2 million pounds in the manufacture of ubber vulcanization accelerators, 4 nillion pounds as corrosion inhibitors in queous cooling systems; and 4 million ounds in the manufacture of MBT (Ref. 1, Monsanto, 1982).

The 1972 production volume of the inc salt of MTB was estimated to be 4 illion pounds (Ref. 18, USITC, 1974). It i used as a rubber accelerator and in ingicidal formulations.

B. Evidence for exposure. MBT has een found at a concentration of 30 μ g/L 1 the wastewater from a tire lanufacturing plant (Ref. 9, Jungclaus et I., 1976) and was detected in the achate from a waste dump (Ref. 4, ox, 1976). In addition to the releases om several manufacturing and rocessing plants, MBT may be released) the environment from the disposal of res and rubber products. Substantial leases occur when MBT and the Na nd Zn salts leach from the estimated 12 illion pounds of vulcanization celerators that are deposited annually i tire dust along highways (Ref. 16, antodonato, 1976). At pH <7, MBT and e Na and Zn salts of MBT are pected to dissociate, yielding the onic form of MBT. According to ktulga (Refs. 1 and 2, 1971a, 1971b), BT can be leached out of rubber; the thor reported that the MBT was a gradation product of

nzothiazyldisulfide, an ingredient of e rubber sample.

Releases may also occur as a result of sposal of waste radiator coolants from tomobiles and industrial cooling stems.

II. Chemical fate information—A. ansport. The occurrence of MBT in the ichate from a waste dump (Ref. 4, ix, 1976) indicates that it has some ibility.

B. Persistence. In tests with Thiotax[®] trade name for 2-

rcaptobenzothiazole), a 1.1 ppm lution had a photolysis half-life of 3.7 urs at midday in August. In the dark, : half-life was approximately 100 urs (Ref. 13, Monsanto, 1984). n an 8-week biodegradation study inducted in the dark) using buffered er water and a 1 ppm solution of iotax[®], there was no biodegradation Thiotax[®] Some chemical degradation s observed (Ref. 13, Monsanto, 1984). 3. Rationale for chemical fate ommendations. Because of the sected releases into the aquatic and restrial environments, further prmation on the persistence of MBT 1 of any degradation product 18 ded. To estimate the amount of MBT t may leach from landfills, testing to ermine leaching and migration rates ecommended.

III. Biological effects of concern to human health. Because of the extensive toxicological testing of MBT already completed and presently underway, no additional health effects testing is being recommended at this time.

MBT fed to two hybrid strains of mice at a daily dose of 100 mg/kg for 18 months failed to cause a significant increase in tumors (Ref. 8, Innes et al., 1969). MBT is presently in the histopathology phase of a National Toxicology Program (NTP) gavage carcinogencity study in rats and mice (Ref. 14, NTP, 1984a).

Analysis of urinary metabolites from rats does intraperitoneally with ³⁵S labeled MBT indicated that the compound underwent conjugation with glutathione and with glucuronic acid. Radiolabeled sulfate was also identified in the urine. Similar results were seen in rabbits and dogs (Ref. Colucci and Buyske, 1965).

NaMBT and MBT were both reported to be negative in *Salmonella* assays in two independent studies (Refs. 13 and 5, Monsanto, 1982; Godek et al., 1984a). In addition, MBT and

mercaptobenzothiazole disulfide were negative in the mouse lymphoma assay conducted by this company (Ref. 11, Monsanto, 1982). In addition, MBT was found to be negative in CHO-/HGPRT mammalian cell forward gene mutation assay (Ref. 6, Godek et al., 1984b) and in micronucleus assay (Sorg et al., 1984). MBT was tested by NTP in the Salmonella assay at two different laboratories. At one laboratory, it was negative in strains TA98, TA100, TA1535, and TA1537 with and without metabolic activation. At the other laboratory, MBT yielded equivocal results in TA98 with activation; it was negative in TA98 without activation as well as in the other strains mentioned above with and without activation. The chemical is presently being tested for its ability to induce chromosomal aberrations and sister-chromatid exchanges in Chinese hamster ovary cells (Ref. 15, NTP, 1984b).

No toxic or teratogenic effects of MBT were noted in studies in which pregnant rats were injected intraperitoneally with MBT on days 1–15 of gestation (Ref. 5, Hardin et al., 1981).

IV Ecological effects of concern—A. Short-term effects. Static, acute toxicity tests have been performed with MBT and NaMBT (Refs. 11, 12, and 13, Monsanto, 1982, 1983, 1984). The following Table 5 summarizes these data; the data demonstrate that MBT and its sodium salt can be highly toxic to some aquatic organisms.

B. Long-term effects. No information was found.

C. Other effects. No information was found.

D. *Bioconcentration.* The log P of 2.42 for MBT suggests that significant bioconcentration in animals is unlikely. In a 72-hour feeding study with carp, elimination of ¹⁴C-MBT was rapid (Ref. 6, Hashimoto et al., 1978).

E. Rationale for ecological effects recommendations. The available data demonstrate that MBT and its sodium salt exhibit high acute toxicity to aquatic organisms. Flowthrough tests in which the test concentrations are measured should be performed to quantify the acute toxicity values. The decreases in LC50s over time in many of the acute tests indicate that chronic effects on these species may occur at considerably lower concentrations. Early life-stage tests with fish and lifecycle tests with daphnids should also be performed to estimate the chronic effect levels. Because of the expected widespread terrestrial exposure along roadways, tests to determine the effects on terrestrial plants should also be performed. Because of differences in dissociation rates at different pHs, acute testing at various pHs should be considered.

TABLE 5.—ACUTE TOXICITY OF MERCAPTOBEN-ZOTHIAZOLE (MBT) AND SODIUM MERCAPTO-BENZOTHIAZOLE (NAMBT) TO AQUATIC OR-GANISMS

	LC ₆₀ (mg/L)					
Spocies	1	MBT		NaMBT*		
	24 h	48 h	96 h	24 h	48 h	96 h
Daphnia	7.0	4.1			19	
magna Rainbow trout	0.92	0.75	0.75	2.0	1.8	1.8
Bluegill Fathead	3.4	2.1	1.5	5.7	4.5	3.8
- minnow Selsnastrum	18	13	115	-	-	-
capncomu- tum	-	-	230	2	1	0.4

^a Tested as a 50% aqueous solution; il calculatod on a 100% active ingredient basis, the LC₅₀s would be 50% lower ^b A yellow preciptate was observed in all test solutions.

References

(1) Aktulga A. 1971a. Identification of the leached ingredients from rubber closures by spectrophotometric analysis. Eczacilik Bul, 13(4):49–56.

(2) Aktulga A. 1971b. Identification of the leached ingredients from rubber closures with gel filtration and quantitative estimation of 2-mercaptobenzothiazole. Eczacilik Bul. 13(5):74–80.

(3) Colucci DF, Buyske DA. 1965. The biotransformation of a sulfonamide to a mercaptan and to mercapturic acid and glucuronide conjugates. Biochem. Pharmacol. 14:457–466.

(4) Cox GB. 1976. Determination of 2mercaptobenzothiazole in waste dump effluent by high pressure liquid

chromatography. J. Chromatogr. 116:244-247. (5) Godek EG, Naismith RW, and Matthews RJ. 1984a. Ames Salmonella/microsome plate test: mercaptobenzothiazole, PH 301-CMA-001-83, PH 301-CMA-001-83A. Submitted by Pharmakon Research International, Inc., Waverly, PA to Chemical Manufacturers Association, Washington, DC.

(6) Godek EG, Naismith RW, and Matthews RJ. 1984b. CHO/HGPRT. Mammalian cell forward gene mutation assay: mercaptobenzothiazole. PH 314-CMA-001-83. Submitted by Pharmakon Research International, Inc., Waverly, PA to Chemical Manufacturers Association, Washington, DC.

(7) Hardin BD, Bond GP, Sikov MR, Andrew FD, Beliles RP, Niemeier RW. 1981. Testing of selected workplace chemicals for teratogenic potential. Scand. J. Work Environ. Health 7 (Suppl. 4):66-75.

(8) Hashimoto K, Sasaki Y, Ohara Y, Matsuzana T. 1978. Absorption, distribution and excretion of 2-mercaptobenzothiazole in carp. Bull. Japanese Soc. Sci. Fisheries 44(6):623-629.

(9) Hawley GG. 1981. The Condensed Chemical Dictionary. 10th ed. New York: Van Nostrand Reinhold Co. p. 652.

(10) Innes JRM, Ulland BM, Valerio MG, Petrucelli L, Fishbein L, Hart ER, Pallotta AJ Bates RR, Falk HL, Gart JJ, Klein M, Mitchell II, Peters J. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice: A preliminary note. J. Natl. Cancer Inst. 42[6]:1101-1114.

(11) Jungclaus GA, Games I.M, Hites RA. 1976. Identification of trace organic compounds in tire manufacturing plant wastewaters. Anal. Chem. 48[13]:1894-1896.

(12) Merck. 1976. The Merck Index. 9th ed. Rahway, NJ: Merck and Co., Inc. p. 762.

(13) Monsanto. 1982. Unpublished information on the production, uses, toxicity, environmental releases, and aquatic toxicity of MBT and sodium MBT submitted by J. R. Condray, Monsanto Co. July 18, 1982. [14] Monsanto. 1983. Unpublished aquatic

toxicity test data for sodium MBT submitted by B. J. Hill. August 12, 1983.

(15) Monsanto. 1984. Unpublished information submitted by B. J. Hill, Monsanto Co. March 29, 1984.

(16) NTP. 1984a. National Toxicology Program. Toxicology Research and Testing Program Management Status Report, p. 24. Available from Florence Jordan, Mail Drop 18-01, NIEHS, POB 12233, Research Triangle Park, NC 27709.

(17) NTP. 1984b. National Toxicology Program. Annual Plan for Fiscal Year 1984. p. 77

(18) Santodonato J. Davis LN, Howard PH. Saxena J. 1976. Investigation of selected potential environmental contaminants: Mercaptobenzothiazoles. Syracuse, NY: Syracuse Research Corp. EPA-560/2-76-006.

(19) Sorg RM, Naismith RW, and Matthews RJ. 1984. Genetic Toxicology. Micronucleus test: mercaptobenzothiazole. PH 309A-CMA-001-83. Submitted by Pharmakon Research International, Inc., Waverly, PA to Chemical Manufacturers Association, Washington, DC.

(20) Uniroyal. 1984. Unpublished information submitted by R.J. Dowling, Uniroyal, Inc. May 4, 1984.

(21) USITC. 1974. U.S. Tarilf Commission, Synthetic Organic Chemicals, U.S Production and Sales, 1972, TC Publ. No. C31.

Washington, DC: U.S. Govt. Printing Office. (22) USITC. 1982. U.S. International Trade Commission, Synthetic Organic Chemicals, U.S. Production and Sales, 1931. USITC Publ. No. 1292. Washington, DC: U.S. Govt. Frinting Office.

2.2.e **Octamethylcyclotetrasiloxane** (9 CI).

Summary of recommended studies. It is recommended that

octamethylcyclotetrasiloxane be tested for the following:

A. Chemical Fate:

Water solubility

Octanol/water partition coefficient Biodegradation

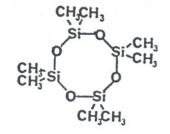
B. Ecological Effects:

- Acute toxicity to fish, aquatic invertebrates, and algae (concentrations of the chemical to be measured during the course of the studies)
- Chronic toxicity to aquatic organisms (testing conditional upon results of acute tests)

Physical and Chemical Information

CAS Number: 556-67-2.

Synonyms: SF 1173 (General Electric); Dow Corning[®] 344 Fluid (95"); Organosilicone Fluid VS-7207 (Union Carbide); Silicone 4-7207 (Union Carbide); OMCTS. Structural Formula:



Empirical Formula: CoH26O4Sis. Molecular Weight: 296.62 Melting Point: 17.4-17.6 °C [Ref. 1,

Alpha, 1982).

Boiling Point: 175 °C (Ref. 1, Alpha. 1982).

Vapor Pressure: 1 mm Hg at ambient temperature (Ref. 26, Scarbel, 1982).

Specific Gravity: 0.9558 at 20 °C (Ref. 1, Alpha, 1982).

Water Solubility: Less than 1 percent (Ref. 26 Scarbel, 1932); less than 500 ppm (Ref. 15, Isquith and Annelin, 1976).

Octanol/Water Partition Coefficient: No information was found.

Description of Chemical: Clear,

colorless liquid (Ref. 11, Griffiths and Parent, 1979).

Rationale for Recommendations

L Exposure information-A. Production/use/disposal. Between 20

and 25 million pounds of octamethylcyclotetrasiloxane (OMCTS) were produced in 1932 by one major manufacturer (Ref. 14, Hobbs, 1932). About 80 percent of this production is used internally as a chemical intermediate, apparently in the manufacture of poly(dimethylsiloxane) and related polymers; the remainder is used in a variety of applications (e.g., as constituents of cosmetics, cleaners, antifiatulents, antacids, and antispasmodics) as mixtures from 3 to 93 percent OMCTS. Disposal of liquid manufacturing wastes and liquid wastes from consumer uses is expected to be via waste treatment facilities. Solid wastes are expected to be disposed of in landfills or by incineration.

B. Evidence for exposure. OMCTS is manufactured in large volume. Its multiple uses indicate that it may be released over wide geographical areas: therefore, many species or organisms may be exposed to this chemical. Furthermore, polyorganosiloxanes (silicones) have been found in effluents from waste treatment plants (4.8 ppb) and in sediment cores taken from Delaware Bay (1.2 ppm) (Ref. 22, Pellenbarg, 1979). Silicones have also been found in river water (2.0-54.2 µg/ L), sediment (0.3 and 5.8 μ g/g), and fish (0.36-0.69 µg/g) samples taken from tributaries of the Nagara River, Japan (Ref. 28, Watanaba et al., 1924).

II. Chemical fate information—A. Transport. OMCTS is volatile and not particularly water-soluble (precise data not available), so it is expected that a good deal of the released chemical may partition to the atmosphere and into soils and sediments. Silicone polymers are less volatile and therefore are expected to partition into soils and sediments only.

B. Persistence. Estimations of the halflife (T½= 13 days) of OMCTS, using the method of Atlanson (Ref. 4, 1930), indicate that the compound is destroyed in the troposphere and, therefore, is not expected to persist following atmospheric release. Isquith and Annelin (Ref. 15, 1976) attempted to measure the biodegradation rate of OMCTS. The compound persisted: however, there was a problem associated with the chemical's solubility and no attempt was made to determine the level of chemical that was actually solubilized during the course of the test.

C. Rationale for chemical fate recommendations. Tests for determining the water solubility, octanol/water partition coefficient, and biodegradation of OMCTS are being recommended for several reasons: Water solubility data are critical to the interpretation of

ecological effects (and biodegradation) data. The octanol/water partition coefficient data are necessary to assist in determining whether or not OMCTS will partition into biota and organic matter in soils and sediments. Confirmation of actual biodegradation is required to determine whether OMCTS can be expected to accumulate in the environment with time. The biodegradation test that has been performed is difficult to interpret, since there is no way of determining the concentrations of the chemical that were actually subjected to such testing.

III. Biological effects of concern to human health. Commercial products containing OMCTS have been tested extensively in laboratory animals with no adverse effects. Further testing for health effects is not being recommended at this time.

Acute and subchronic tests with OMCTS administered via the oral, dermal, and inhalation routes showed in most cases no untoward effects (Refs. 20, 29, 30, 21, 11, 24, and 19, McHard, 1961; Wolf, 1956; Weil et al., 1972; Myers et al., 1982; Griffiths and Parent, 1979; Rowe et al., 1948; Janssen, 1974). OMCTS was essentially nonirritating in skin and eye irritation tests (Refs. 9, 29, and 12, Clayton, 1972; Wolf, 1956; Groh, 1978). Chronic studies using Antifoam (5 percent OMCTS) also did not produce any significant effects (Refs. 6, 25, and 8, Carson, 1965, 1966; Rowe et al., 1950; Child et al., 1951). A three-generation itudy in rats using Antifoam A revealed 10 treatment-related effects on eproduction, hermatology, renal unction, and histopathology, and no hange in morbidity or tumor incidence Ref. 27, Statt and Bennett, 1974). **DMCTS** administered to rats to assess eproductive effects gave no evidence or ither inducing chromosomal damage in erminal tissue or resulting in any strogenic effects (Refs. 16, and 13, squith et al., 1982; Hayden and Barlow, 972). Antifoam A was tested for eratogenic potential in rats with no etectable embryotoxic or teratogenic ffects observed in fetuses (Ref. 10, longwer et al., 1970). Genotoxicity tests icluding the Ames Salmonella/ ucrosome plate test, the dominant thal assay, and the mouse lymphoma ell assay using OMCTS did not produce ny treatment-related effects (Refs. 18, 7 and 16, Jagannath and Brusick, 1978; quith and Whaley, 1979; Isquith et al., **)82)**.

IV Ecological effects of concern—A. cute toxicity. Acute toxicity tests have sen performed with several types of ganisms exposed to nominal incentrations of OMCTS. In a 72-hour test with *Daphnia magna*, the LC_{10} , LC_{50} , and LC_{50} were 1.85, 23.4, and 298 ppm, respectively (Ref. 2, Annelin, 1976a). In a test with algae, up to 2,000 ppm (far in excess of the expected water solubility) of OMCTS did not inhibit growth, although there was an unexplained variation in filament weights (Ref. 3, Annelin, 1976b).

In addition, 4-day static bioassays were performed with rainbow trout, bluegill, mummichog, shore crab, and grass shrimp. No effects were observed up to 100 ppm (Ref. 23, Rausina et al., 1976); however, at 1,000 ppm all tests animals were quiescent.

In test performed with the house fly, southern armyworm, Mexican bean beetle, pea aphid, and strawberry spider mite, no toxic effects were observed (Ref. 7, Cerro, 1976).

B. Long-term toxicity. No information was found.

C. *Bioconcentration*. No information was found.

D. Rationale for ecological effects recommendations. The ecological test results did not indicate a high level of toxicity; however, nominal concentrations or OMCTS were used. In view of the volatile nature of the compound and uncertainty regarding its solubility, the interpretation and validity of these results should be questioned. Retesting should be performed in which concentration levels are determined at several intervals during the testing period.

References

(1) Alpha Catalog. 1982. Ventron Division of Thiokol Corp., Danvers, MA.

(2) Annelin RB. 1976a. Acute toxicity test with Daphnia magna-

octamethylcyclotetrasiloxane. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.

(3) Annelin RB. 1976b. Algal assay-bottle test: Octamethylcyclotetrasiloxane. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.

(4) Atkinson R. 1980. Comments on "predicting gas phase organic molecule reaction rates using linear free energy correlations. I. O(P) and OH addition and abstraction reactions." J. Chem. Kinet. 12:761– 765.

(5) Carson S. 1965. Chronic (3-month) feeding studies with Antifoam A in rabbits.
Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.
(6) Carson S. 1966. Chronic (1-year) feeding

(6) Carson S. 1966. Chronic (1-year) feeding studies with Antifoam A in rats. Upublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.

(7) Cerro JE. 1976. Primary insecticidal and miticidal screening of octamethylcyclotetrasiloxane. Unpublished

octamethylcyclotetrasiloxane. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.

(8) Child GP. Paquin HP Deichmann WB. 1951. Chronic toxicity of the methylpolysiloxane "Dow Corning Antifoam A" in dogs. AMA Arch. Ind. Hyg. 3:479–482.

(9) Clayton TE. 1982. Silicone Y-7207, range finding toxicity study. Unpublished raport submitted by D.L. Heywood, Union Carbide Corp. July 22, 1982.

(10) Gongwer LE, Saatman RA, Hubben K. 1970. Dow Corning Antifoam A (medical grade): A teratogenic potential study in rats. Unpublished report. Submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.

(11) Griffiths JT, Parent RA. 1979. Dermal acute study in rabbits. Unpublished roport submitted by M.P. Scarbel, General Electric Silicone Products. July 15, 1982.

(12) Gorh CL. 1978. Investigation of the concentration of a silicone vapor necessary to elicit threshold reactions in the human eye. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.

(13) Hayden JF, Barlow SA. 1972. Structureactivity relationships of organosiloxanes and the female reproductive system. Toxicol. Appl. Pharmacol 21:68–79.

(14) Hobbs EJ. 1982. Letter with attachments submitted by Dow Corning Corp. December 18, 1982.

(15) Isquith AJ. Annelin RB. 1976. Biochemical oxygen demand of octamethylcyclotetrasiloxane. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.

(16) Isquith AJ, Siddiqui WH, Miller BJ, Stanton E. 1982. Evaluation of

octamethylocyclotetrasiloxane in the rodent dominant lethal assay. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.

(17) Isquith AJ, Whaley DP. 1979. Mutagenicity evaluation of Dow Corning 344 Fluid in the Ames bacterial assay. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 10, 1982.

(18) Jagannath DR and Brusick DJ. 1978. Mutagenicity evaluation of octamethylcyclotetrasiloxane Part I microbial assays, Part II cell culture assays. Unpublished report submitted by R.C.

Gerlach, Industrial Health Foundation, Inc. July 6, 1982.

(19) Janssen L. 1974. 90-Day Subacute Aerosol Inhalation Toxicity Study with TX-1015, TS-997 and TX-1013 in Cynomolgus Monkeys. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 10, 1982.

(20) McHard J. 1961. Results of range finding toxicological tests on dimethyl cyclic tetramer. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 10, 1982.

(21) Myers RC, DePass LR, Frank FR. 1982. Organosilicone fluid VS-7207, acute inhalation toxicity studies. Unpublished report submitted by D.L. Heywood, Union Carbide Corp. July 22, 1982.

(22) Pellenbarg R. 1979. Environmental poly(organosiloxanes) (silicones). Environ. Sci. Technol. 13;505–573.

(23) Rausina G. 1976. Four-day static aquatic toxicity with TX-1283 in freshwater and saltwater species. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982. (24) Rowe VK, Spencer HC, Bass SL. 1948. Toxicological Studies on certain commerical silicones and hydrolyzable silane intermediates. J. Indust. Hyg. Toxicol. 30(6):332–353.

(25) Rowe VK, Spencer HC, Bass SL. 1950. Toxicological Studies on certain commercial silicones. II. Two year dietary feeding of "DC Antifoam A" to rats. Arch. Ind. Hyg. Occup. Med. 1:539–544.

(26) Scarbel MP. 1982. Letter with attachments submitted by General Electric Co. July 15, 1982.

(27) Statt WH, Bennett DR. 1974. Pharmacokinetic and metabolic studies on Dow Corning© Antifoams A and M in mice, monkeys and humans. Appendix I. Polydimethylsiloxane—13th Report of FAO/ WHO Food Additives Committee. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.

(28) Watanabe N, Nakamura T, Watanabe E, Sato E, Ose Y. 1984. Distribution of organosiloxanes (silicones) in water, sediments and fish from the Nagara River watershed. Sci. Total Environ. 35:91-97.

(29) Wolf MA. 1956. The results of range finding toxicological tests on octamethylcyclotetrasiloxane. Unpublished report submitted by E.J. Hobbs, Dow Corning Corp. December 16, 1982.

[30] Weil CS, Condra NI, Geary DL, Jr., DeMary LJ. 1972. Silicone Y-7207 range finding toxicity studies. Unpublished report submitted by D.L. Heywood, Union Carbide Corp. July 22, 1982.

2.2.f Pentabromoethylbenzene. Summary of recommended studies. It is recommended that pentabromoethylbenzene be tested for the following: A. Health Effects:

Two-year chronic bioassay

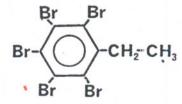
Teratogenicity study

B. Ecological Effecats:

Acute and chronic toxicity to fish, aquatic invertebates, and plants

Physical and Chemical Information

CAS Number: 85–22–3. Structural Formula:



Empirical Formula: C₈H₅Br₅. Molecular Weight: 501.

Melting Point: 135–138 °C (Ref. 3, Great Lakes, 1984a).

Boiling Point: 371 °C (estimated; Ref. 5,

Lyman et al., 1982). Vapor Pressure: $2-4 \times 10^{-8}$ mmHg at 25

°C (estimated; Ref. 5, Lyman et al., 1982).

Specific Gravity: Approximately 2.7 at 25 °C.

Log Octanol/Water Partition Coefficient: 7.3 (estimated; Ref. 5, Lyman et al., 1982).

Water Solubility: 8 ppb (estimated; Ref. 5, Lyman et al., 1982).

Decription of Chemical: White/offwhite powder

Rationale for Recommendations

I. Exposure information—A. Production/use/disposal. The TSCA Inventory (public portion) reported the 1977 production of pentabromoethylbenzene (PEB) to be between 100,000 and 1,000,000 pounds (Ref. 1, EPA, 1977). Current production data are not publicly available; however, two manufacturers reported publicly that they are currently producing the compound.

PEB is an additive-type flame retardant. Suggested applications include thermoset polyester resins for circuit boards, textiles, adhesives, wire and cable coatings, polyurethanes, and thermoplastic resins (Refs. 2, 3, and 4, Ethyl, 1984; Great Lakes, 1984a, 1984b). A typical concentration level in thermoplastic resins is 12 parts of PEB by weight per 100 parts of resin (Ref. 3, Great Lakes, 1984a). One manufacturer recently reported that production process wastes are disposed of offsite in secure landfills (Ref. 2, Ethyl, 1984).

B. Evidence for exposure. Two manufacturers reported limited opportunity for worker exposure because most of the manufacturing process is enclosed; however, they reported that a small number of workers may be exposed to PEB dust during packaging and shipping operations. Consequently, they recommend or require protective equipment for these workers (Refs. 4 and 2, Great Lakes, 1984a; Ethyl, 1984). Consumer exposure to low levels of PEB is possible through direct contact with finished products (e.g., treated textiles) or environmental media contaminated with the compound.

II. Chemical fate information—Å. Transport. PEB's high estimated octanol/water partition coefficient and low water solubility suggest that the compound partitions from water into sediments and has a strong tendency to bioaccumulate. Its low volatility suggests no partitioning into air except as dust particles.

B. *Persistence*. PEB's structure suggests that it is highly resistant to biodegradation and hydrolysis.

III. Biological effects of concern to human health—A. Toxicolanetics and metabolism. No information was found.

B. *Genotoxicity*. PEB was negative when tested for mutagenicity in *Salmonella* strains TA98, TA100, TA1535, and TA1537 with and without activation (Ref. 8, NTP, 1984).

C. Short-term effects. In primary skin irritation and sensitization studies, PEB was not a primary skin irritant or sensitizer (Refs. 3 and 2, Great Lakes, 1984a; Ethyl, 1984). In an inhalation study, rats exposed to PEB for 1 hour at a 2 mg/L concentration in air exhibited increased and then decreased respiration, prostration, salivation, lacrimation, erythema, and decreased motor activities. At 200 mg/L, dyspnea was also observed; no deaths occurred at either concentration (Ref. 3, Great Lakes, 1984a). The oral LD₅₀ in unspecified animals has been reported to be greater than 5,300 mg/kg; the inhalation LCco. greater than 200 mg/L, and the dermal LD₁₀, greater than 8,000 mg/l:g (Ref. 2, Ethyl, 1984). In a 28-day feeding study, no compound-related effects (except for slightly reduced weight gain) were observed in rats fed 1,000 and 100 ppm of PEB (Ref. 3, Great Lakes, 1984a).

D. Long-term (chronic) effects. No information was found.

E. Reproductive effects and teratogenicity. No information was found.

F. Rationale for health effects recommendations. There is concern for worker exposure to PEB dust during manufacture of the compound, and especially during processing of products treated with PEB. Also, consumers and downstream workers may be dermally exposed to the compound via migration from treated polymeric products and textiles. The extent of these exposures, however, cannot be assessed due to the lack of current publicly available production information. Because of the lack of information on the chronic effects of PEB, and the known toxic effects observed in compounds having a polyhalogenated aromatic morety, a 2year chronic bioassay and a teratogenicity study are recommended. The chronic bioassay is recommended rather than short-term tests because the latter do not, in general show a positive association with carcinogenicity for polyhalogenated compounds (Refs. 9, 7, and 6, Rinkus and Legator, 1979; McCann et al., 1975; McCann and Ames, 1976). In addition, teratogenicity testing is recommended because of lack of information.

IV. Ecological effects of concern—A. Short-term (acute effects). No information was found.

B. Long-term effects. No information was found.

C. *Bioconcentration*. PEB's high estimated log P suggests a strong tendency to bioaccumulate. A

structurally related compound, pentabromomethylbenzene (PMB), in a study with juvenile Atlantic salmon (Salmo salar) [Ref. 10, Zitko and Carson, 1977), exhibited a fairly low uptake from water (96 hours) and from food (14, 28, and 42 days). Depuration half-lives were 32 and 83 days for uptake from water and food, respectively. It should be noted that 96 hours is a fairly short time for evaluating chemical uptake from water, and that an extended period of testing may have resulted in much higher accumulation. The relatively long depuration half-lives create some concern for the potential for chronic effects.

D. Rationale for ecological effects recommendations. Acute and chronic toxicity to aquatic organisms and plants are recommended for the following reasons:

1. The purported and potential uses of PEB are evidence of its probable wide distribution.

2. Data on a structurally related compound pentabromomethylbenzene) indicate that, although low levels of PEB are likely taken up by aquatic organisms, its residence time in the body may be relatively long. This provides presumptive evidence that PEB may have the potential to produce chronic effects.

3. PEB is structurally similar to many halogenated compounds that have appreciable toxicity.

4. The chemical is expected to partition into soils, sediments, and biota after release.

References

(1) EPA. 1977. TSCA Chemical Substances Inventory (public portion). Washington, DC: Environmental Protection Agency.

(2) Ethyl. 1984. Unpublished information on PMB submitted by R.L. Smith, Ethyl Corp. February 1, 1984.

(3) Great Lakes. 1984a. Unpublished information on PEB and PMB submitted by D.L. McFadden, Great Lakes Chemical Corp. January 11, 1984.

(4) Great Lakes. 1984b. Unpublished information on PEB and PMB submitted by D.L. McFadden, Great Lakes Chemical Corp. February 14, 1984.

(5) Lyman WJ, Reehl WF, Rosenblatt DH. 1982. Handbook of Chemical Property Estimation Methods. New York: McGraw-Hill Book Co. Chapters 1, 2, 12, and 14.

(6) McCann J, Ames B. 1976. Detection of carcinogens as mutagens in the Salmonella microsome test: Assay of 300 chemicals: Discussion. Proc. Nat. Acad. Sci. 73:950–954.

(7) McCann J, Choi E, Yamasaki E, Ames B. 1975. Detection of carcinogens as mutagens in the *Salmonella*/microsome test: Assay of 300 chemicals. Pros. Nat. Acad. Sci. 72:5135-5139.

(8) NTP. 1984. National Toxicology Program. NTP testing status on 18 compounds submitted by V. Fung. January 1, 1984. (9) Rinkus SJ, Legator MS. 1979. Chemical characterization of 465 known or suspected carcinogens and their correlation with mutagenic activity in the *Salmonella typhimurium* system. Cancer Res. 39:3289– 3318.

(10) Zitko V, Carson WG. 1977 Uptake and excretion of chlorinated diphenyl ethers and brominated toluenes by fish. Chemosphere 6(6):293–301.

2.2.g. Sodium N-Methyl-N-

Oleoyltaurine.

Summary of recommended studies. It is recommended that sodium N-methyl-N-oleoyltaurine be tested for the following:

A. Health Effects:

Short-term genotoxicity

- Sensitization
- Chronic toxicity to include oncogenicity (testing conditional upon results of short-term tests)

Physical and Chemical Information

CAS Number: 137-20-2. Synonyms: Sodium oleoylmethyltauride; Ethanesulfonic acid, 2-[methyl[l-oxo-9octadecenyl]amino]-, sodium salt, (Z) [9 CI].

Structural Formula:

Empirical Formula: C₂₁H₄₀NO₄S·Na. Molecular Weight: 426.

Melting Point: No information was found.

Boiling Point: No information was found.

Vapor Pressure: No information was found.

Specific Gravity: No information was found.

Solubility in Water: No information was found.

Description of Chemical: Fine, white powder (Ref. 5, Hawley, 1981).

Rationale for Recommendations

I. Exposure information—A. Production/use. It was reported that in 1983 there were nine manufacturers of sodium N-methyl-N-oleoyltaurine (Ref. 7, SRI 1983). In 1977, the production/ importation volume listed in the public portion of the TSCA Inventory was 300,000 to 3.1 million pounds (Ref. 3, EPA, 1984). Current production volumes are not publicly available.

The compound is an anionic surfactant that is widely used as a detergent and wetting agent in pesticidal formulations (Refs. 4, and 5, GAF, 1984; Hawley, 1981). It is also used in textile applications to wash prints and fabrics (Ref. 1, CNC Chemical, 1984), in rug shampoos, and in laundry soaps (Ref. 4, GAF, 1984).

B. Evidence for exposure. There is a potential for worker exposure in the textile and pesticide formulation industries. Consumers may be exposed to the salts via the compound's use in products such as household detergents, rug shampoos, laundry soaps, and surface coatings. Consumer exposure is expected to be principally via dermal contact.

II. Chemical fate information. Chemical fate testing of sodium Nmethyl-N-oleoyltaurine is not being recommended at this time, since the petinent chemical fate data allowing decisions to be made with regard to ecological effects testing are known. The compound partitions to the surface of aquatic bodies where it is rapidly biodegraded. In one of several studies, the chemical was found to be degraded by 75 pecent in Chesapeake Bay water within 1-4 days [Ref. 2, Cook and Goldman, 1974].

III. Biological effects of concern to human health—A. Toxicological information. In mice, the acute LD₅₀ via the intravenous route was reported to be 350 mg/kg. At a concentration of 1 pecent in water, the compound caused "severe irritation" when applied to the eyes of rabbits (Ref. 6, Hopper et. al., 1949). No other toxicological information was found.

B. Rationale for recommendations. The use of sodium N-methyl-Noleoyltaurine in household detegents, rug shampoos, and laundry soaps (Refs. 1 and 4, CNC Chemical, 1984; GAF, 1984) indicates the possibility of widespread, repeated exposure of consumer to the compound. Industrial releases to the atmosphere as fugitive dust from manufacturing and pesticide formulation may also be important from the standpoint of worker exposure. Due to the absence of health data on the compound and the potential for worker and consumer exposure. it is recommended that the compound be tested for short-term genotoxicity, sensitization in appropriate test systems, and chronic toxicity/ oncogenicity (conditional upon the results of the short-term tests).

IV Ecological effects. No information was found. The compound is expected to be released to surface waters, where it will partition to air/water, soil/water, and sediment/water interfaces. However, since rapid biodegradation of the compound is expected, testing for ecological effects is not being recommended at this time.

References

 CNC Chemical. 1984. Unpublished information of the production and use of N/ methyl-N-oleoyltaurine submitted by P. O'Neil, CNC Chemical Co. May 1, 1984.

(2) Cook TM, Goldman CK. 1974. Degradation of anionic detergents in Chesapeake Bay. Chesapeake Sci. 15(1) 52– 55.

(3) EPA. 1984. TSCA Chemical Substances Inventory (public portion). Washington, DC: Environmental Protection Agency.

(4) GAF. 1984. Unpublished information on the production and use of N-methyl-Noleoyltaurine submitted by M. Rybka and H. Feigenbaum, GAF Corp. May 3, 1984.
(5) Hawley GG. 1981. The Condensed

(5) Hawley GG. 1981. The Condensed Chemcial Dictionary, 10th ed. New York: Van Nostrand Reinhold Co. p. 945.

(6) Hopper SS, Hulpieu HR, Cole VV. 1949. Some toxicological properties of surfaceactive agents. J. Am. Pharm. Assoc. 38:428– 432.

(7) SRI. 1983. Directory of Chemical Producers, USA, 1983. Stanford Research Institute, Menlo Park; CA: SRI International p. 907

[FR Doc. 84-31162 Filed 11-28-84; 8:45 am] BILLING CODE 6550-50-M

[OPTS-53065; TSH-FRL 2694-1]

Premanufacture Notices; Monthly Status Report for August 1984

Correction

In FR Doc. 84–27139 beginning on page 40200 in the issue of Monday, October 15, 1984, make the following corrections:

1. On page 40201, first column, ADDRESS, third line "[OPI'S-5305]" should read "[OPTS-53065]"

2. On the same page "Table 1. "should read "Table I"

3. On the same page Table I, PMN No. 84–1050, the third column should read "49 FR 33718 (33720) (8–24–84); and PMN No. 84–1065, third column, "do" should read "49 FR 33718 (33722) (8–24–84)" 4. On page 40202, Table II, PMN No.

84-905, second column, insert "resin" after "phenolic"; PMN No. 84-907, second column, insert "resin." after "phenolic"; PMN No. 84-913, second column, "thiazoline" should read "thiasoline"; PMN No. 84-916, second column, "substitued" should read "substituted"; PMN No. 84-927, second column "Do" should be removed; PMN No. 84-929, second column, "mono-Methyl" should read "mono-methyl"; PMN No. 84–936, second column, "Ketoester" should read "keto-ester": PMN No. 84–941, fourth column, "Do" should read "October 8, 1984"; PMN No. 84-952, fourth column, "Do" should read "October 13, 1984"; and PMN No. 84-958, fourth column, "Do" should read "October 14, 1984"

5. On page 40203, Table II, PMN No. 84–968, second column, "alkyl" should read "Alkyl"; PMN No. 84–986, second column, first line, "naphthylazo)" should read "naphthylazo]]"; PMN No. 84–933, second column, "salt-" should read "salt="; PMN No. 84–990, second column, second line, "(0, 0'(5)]" should read" (0, 0')[5-]]"; and PMN No. 84–990, second column, "Polyalkyl" should read "Poly alkyl"

6. On the same page, Table III, PMN No. 84–274, "[(1–OXO-2-propenyl) OXY]" should read "[(1-OXO-2propenyl) OXY]-"

7 On page 40205, Table IV, PMN No. 82–259, fourth column, "1984" should be removed.

8. On page 40206, Table V, PMN No. 84–376, second column, "Do" should read "Generic name: Aryl esters of alkyl dithiocarbamates."; and PMN No. 84– 558, third column, "(4803)" should read "(14803)"

BILLING CODE 1555-01-M

[FRL-2727-7]

Notice of Public Meeting on 1984 RCRA Amendments

The Environmental Protection Agency (EPA) will hold a public briefing on December 11, 1984, which will be in the form of a video teleconference to discuss and receive comments on the 1984 amendments to the Resource Conservation and Recovery Act (RCRA). The videoconference will originate in Washington, D.C. and be simultaneously televised to 10 regional satellite sites around the country, as well as a Washington, D.C. site.

A panel of EPA experts in Washington will discuss the major provisions of the bill and provide an opportunity for questions from the audiences. The videoconference will originate from the Biznet Studio in Washington, D.C.

The new law is even more extensive than the original RCRA statute. Among other provisions, it will for the first time, add many small businesses to those which must meet Federal guidelines for managing hazardous wastes.

EPA is inviting national. State and regional environmental officials, together with representatives of industry, environmental interest groups, and the general public to take a closer look at how the new bill will work and how it will affect the management of hazardous waste in this country.

In addition to summarizing the major provisions of the bill, the panel will describe a rule that EPA plans to publish in December. That rule codifies in regulations those requirements specified in the sections which, by their own term, take effect immediately following or shortly after enactment of the 1934 Act. The following provisions covered by the Codification Rule will be discussed:

 The ban on placement of bulk liquid hazardous waste and non-hazardous liquids in landfills;

2. The permitting and interim status requirements for double-liners and leachate collection systems at surface impoundments and landfills;

3. The re-definition of "regulated unit" for purposes of the ground-water* monitoring and response program;

 The obligation to institute corrective action for solid waste management units at permitted facilities;

5. The requirement to take corrective action beyond a facility's property boundary where needed;

 The elimination of the double-liner variance from the ground-water monitoring and response program allowed for landfills, surface impoundments and waste piles;

7. The variance from ground-water monitoring allowed for certain engineered structures;

 The ban on disposal in certain salt dome formations, caves and underground mines;

 The ban on use of materials mixed with dioxin or other hazardous waste for dust suppressions;

10. The interim measures (i.e., manifest and destination requirements) for small quantity generators producing between 100 and 1000 kilograms of waste per month;

11. The preconstruction ban with the variance for PCB facilities having EPA approvals under TSCA:

12. The restrictions on a facility s permit life;

13. The authority to add conditions to a permit beyond those provided for in regulations;

14. The extension of interim status to facilities that become subject to permitting requirements because of new regulatory requirements;

15. The loss of interim status for facilities failing to submit Part B applications within specific deadlines;

 The ban on the placement of hazardous wastes in certain cement kilns;

17. The requirement to label hazardous waste fuels;

18. The exclusion for certain wastes burned at resource recovery facilities;

19. The requirements for submission of exposure information about