National Advisory Committee (NAC)
for Acute Exposure Guideline Levels (AEGL) for Hazardous Substances
Final Meeting 1 Highlights

Green Room, 3rd Floor, Ariel Rios Building
1200 Pennsylvania Avenue, NW
Washington, D.C.
June 19-21, 1996

INTRODUCTION

The highlights of the meeting are outlined below, and the meeting agenda (Attachment 1) and attendee list (Attachment 2), and acronym list (Appendix) are attached.

Dr. Roger Garrett (U.S. EPA) provided an historical overview of the project including establishment of the Federal Advisory Committee Act (FACA) and the National Advisory Committee for Acute Exposure Guideline Levels (AEGLs) for Hazardous Substances under FACA; the genesis of AEGLs from and along with other inhalation guidelines; the process by which AEGLs are developed, reviewed, and published; and the role of the chemical manager in the AEGL evaluation process. Dr. Garrett also discussed the National Academy of Science's (NAS) "Guidelines for Developing Community Emergency Exposure Levels (CEELs) for Hazardous Substances," which is to be used as guidance for deriving AEGLs. He pointed out that CEELs and AEGLs are identical and that values were renamed AEGLs to reflect their more generic application. Chemical managers will serve as liaisons among committee members and attempt to resolve scientific issues, seek a consensus of the committee members, frame scientific issues for upcoming committee meetings, present the draft AEGL values and issues at the meeting, and engage in follow-up activities.

Dr. Garrett introduced Dr. Paul Tobin (EPA), the assigned "Designated Federal Officer" (DFO) for this FACA committee, and the chair of the AEGL committee, Dr. George Rusch (AlliedSignal). Dr. Tobin gave an orientation regarding guidance for AEGL development. The organizations that may participate include AAPCC, ACOEM, AFL-CIO, ATSDR, CDC, DOE, DOT, DoD, EPA, FEMA, ICEH, NFPA, NESCAUM, OSHA, STAPPA/ALAPCO, AlliedSignal, Exxon and state agencies. In addition, discussions continue with regard to participation by FDA and NIOSH. He emphasized the need for numbers by these and other participants (e.g., chemical companies, manufacturers, and the state of Pennsylvania for its incineration program). Without the development of these values, evacuation guidelines may be set by persons who are not scientifically trained. The AEGL values will also help eliminate some of the overlap among agencies currently developing guidelines.

Dr. Rusch gave a brief introduction to the committee and requested that the members be provided bylaws before the next meeting.

To provide AEGL members with a comprehensive background and the scientific principles involved in developing CEELs, Dr. John Doull (University of Kansas Medical Center, retired) reviewed the process presented in the "Guidelines for Developing Community Emergency Exposure Levels for Hazardous Substances."

Several questions were asked before the committee members began their review of the draft. (Answers were prepared after the meeting and provided by the EPA project officer, the DFO, the AEGL chair, and Oak Ridge National Laboratory [ORNL] staff.)

Q. Can more time be given to the committee members reviewing the drafts prior to the meeting?
A. Ideally, 6 weeks will be given between the committee members receiving the drafts and the meetings. However, it will require several meetings before this amount of time can be provided.

Q. How will the uncertainty factors be used?
A. A special task group will be reviewing this issue and will provide some information at the next meeting.

Q. Can the references be provided to the committee members?
A. The chemical manager will receive a full set of key references, and additional references can be provided by request. Committee members can request the ORNL staff to provide articles from the draft document's reference check list.

REVIEW OF DRAFT DOCUMENTS

Fluorine
CAS No. 7782-41-4

Chemical manager: Dr. Ernest Falke, EPA
Author: Dr. Sylvia Talmage, ORNL

Dr. Ernest Falke presented an overview of the draft technical support document and the revised AEGL values. (Attachment 3 is a copy of the slides used in that presentation.)

He emphasized the similarity in response (particularly the LC_{50} values but also the irritant effects) to fluorine among four tested species -- rat, mouse, guinea pig, and rabbit -- and the steepness of the dose-response curve. The mouse data for mild and severe lung congestion were used to derive the AEGL-2 and AEGL-3 values, respectively. These data are 67 ppm for 30 minutes and 30 ppm for 60 minutes (very mild lung congestion) and 75 ppm for 60 minutes (severe lung congestion). Because the irritant and LC_{50} concentrations among species were nearly identical, indicating that irritation and lethality are a function of the concentration of fluorine in the air, no scaling factor among species was applied. The data were divided by a factor of 3 for differences in human sensitivity and by a factor of 2 to account for the fact that the data set was from one laboratory and not confirmed elsewhere. At the suggestion of a committee member, the AEGL-2 values will be compared with values derived from a human exposure to 25 ppm for 5 minutes that resulted in slight irritation of the eyes. Also, at the suggestion of a committee member, the revised AEGL-1 values, initially based on a slight irritant effect to humans at an intermittent exposure to 10 ppm for a total of 30 minutes, were recalculated based on no effects during continuous exposure to 10 ppm for 15 minutes. The resultant values were divided by 3 to account for differences in human sensitivity. All values were scaled from the test time periods to other time periods by the formula derived from the animal test data: $C^n \times t = k$, where C is the concentration, n is approximately 2, t is time in minutes, and k is a constant. The values accepted by the majority of the committee members are summarized in the following table. Two committee members concurred with the AEGL values developed by NAC but with comments. These comments will be prepared and become an integral part of the technical support document.

SUMMARY TABLE OF AEGL VALUES					
Classification	**30 Minutes**	**1 Hour**	**4 Hours**	**8 Hours**	**Endpoint (Reference)**
AEGL-1	2 ppm (4 mg/m^3)	2 ppm (3 mg/m^3)	1 ppm (1 mg/m^3)	1 ppm (1 mg/m^3)	no effect in humans (Keplinger and Suissa, 1968)
AEGL-2*	11 ppm (17 mg/m^3)	5 ppm (8 mg/m^3)	2 ppm (4 mg/m^3)	2 ppm (3 mg/m^3)	mild lung congestion-mice (Keplinger and Suissa, 1968)
AEGL-3	19 ppm (29 mg/m^3)	13 ppm (19 mg/m^3)	6 ppm (9 mg/m^3)	4 ppm (6 mg/m^3)	severe lung congestion-mice (Keplinger and Suissa, 1968)

*AEGL-2 values for 30 and 60 minutes were based on separate data points.

Methyl Mercaptan
CAS No. 74-93-1

Chemical manager: Dr. Doan Hansen, BNL
Author: Dr. James C. Norris, ORNL

Dr. Hansen presented an overview of the draft. Attachment 4 is a copy of the slides used in that presentation.

After discussion of the draft completion of the following actions was determined to be needed before the document could be forwarded.

1. Compare the results from the ten Berge and the Wilson equations.
2. Obtain a translation of the Horiguchi (1960) paper for more details.
3. Obtain a translation of the Pickler (1918) paper.
   How was methyl mercaptan analyzed?
   Was the methodology valid?
   Were additional analog chemicals tested?
4. Determine if there are definitive reasons for "dismissing" the results of Seluzhitsky (1972) other than the low values.
5. Can the subchronic results of Tansy et al. (1981) be incorporated for setting the AEGL-2 value?
6. For the scaling of AEGL-3 values, use 400 ppm instead of 600 ppm from the Tansy et al. (1981) paper.
7. The nausea and vomiting for ethyl mercaptan should be used to set AEGL-2 values and not AEGL-1 values.
8. What are the IDLH values for structural related chemicals?
9. Should an uncertainty factor of 10 be used instead of 3?
10. Mail a copy of the Tansy et al. (1981) paper to George Alexeeff.

11. Determine the AEGL values from the benchmark methodology. (Dr. Daniel Guth, EPA, committed to perform these calculations).

The draft document for methyl mercaptan will be reconsidered at the next meeting to fully evaluate comments from outside participants.

Hydrazine
CAS No. 302-01-2

Chemical manager: Dr. Richard Thomas, ICEH
Author: Dr. Robert A. Young, ORNL

Dr. Richard Thomas presented an overview of the draft. Attachment 5 is a copy of the slides used in that presentation.

After discussion of the draft, completion of the following actions was determined to be needed before the document could be forwarded.

1. Review 2 or 3 epidemiological studies mentioned by Dr. Richard Thomas.
2. Incorporate maternity toxicity for AEGL-2 and embryonic toxicity for AEGL-3.
3. Perform cancer calculations.
4. Incorporate the vapor density value.
5. Perform C^n x $t = k$, where $n = 2$.
6. Obtain additional information on acute exposures in animal studies and human experience.

Ammonia
CAS No. 7664-41-7

Chemical manager: Mr. Larry A. Gephart, Exxon Biomedical Sciences
Author: Dr. Kowetha A. Davidson, ORNL

Mr. Larry Gephart presented an overview of the draft. Attachment 6 is a copy of the slides used in that presentation.

Dr. Daniel Guth analyzed the ammonia data using categorical regression and presented his results.

Dr. George Alexeeff analyzed the ammonia data using a benchmark approach and presented his results.

Dr. Robert A. Michael (RAM TRAC Corp.) presented an overview of the report "Acute Inhalation Risks Potentially Posed by Anhydrous Ammonia," dated May 31, 1996 (Attachment 7).

The AEGLs agreed upon by the committee are listed below.

SUMMARY TABLE OF AEGL VALUES FOR AMMONIA

Classification	30 Minutes	1 Hour	4 Hours	8 Hours	Endpoint (Reference)
AEGL-1	25 ppm (17 mg/m^3)	25 ppm (17 mg/m^3)	25 ppm (17 mg/m^3)	25 ppm (17mg/m^3)	odor (no reference)
AEGL-2*					
AEGL-3*					

*To be determined.

Committee recommendations included recalculating the HEC values and describing the different approaches used for deriving AEGL values for ammonia at the next meeting.

CLOSING COMMENTS

Dr. George Rusch requested comments regarding the format and results of the meeting. Listed below are those comments:

1. A wide range of technical issues were discussed.
2. The quality of ORNL's documents was excellent, and ORNL was responsive to the chemical managers' needs.
3. A good exchange of ideas and information took place.
4. The interaction between committee members and document authors is a critical step in the AEGL developmental process.
5. Having different perceptions from the committee members was helpful.
6. The diversity of backgrounds, interests, and disciplines of the committee members facilitated the committee's task.
7. In a short time period, a number of values were generated.
8. AEGL values should be based on "good" science.
9. The chemical managers provided needed support.
10. Voting was a valuable part of the process.
11. The selection of the first four chemicals provided a diverse number of problems.
12. The Chair did an exceptional job.
13. The DFO's support was excellent.
14. The cooperation of all the committee members was appreciated in dealing with governmental delays.
15. The efforts of Dr. Roger Garrett were appreciated.
16. It was great not to have any telephones.
17. The leadership of Drs. Garrett, Tobin, and Rusch was appreciated.
18. Broad coverage of issues aided in understanding.
19. The committee was supportive to all speakers.

ACTION ITEMS

- Issues on the use of uncertainty factors (such as intraspecies differences). ORNL will coordinate with Drs. Alexeeff, Borak, Gephart, and Guth on a progress report to be presented at the next meeting.
- Definitions of AEGLs are to be reviewed. ORNL will work with Dr. Thomas for clarified

definitions.

- EPA will be responsible for distributing bylaws to the committee members.

NAC/AEGL FUTURE MEETINGS

- NAC AEGL Meeting 2: August 5, 6, and 7 in Washington, D.C.
- NAC AEGL Meeting 3: September 17, 18, and 19 in Washington, D.C.
- All chemicals scheduled for review should be distributed to the committee.
- The documents need to be distributed earlier.

Dr. Po-Yung Lu (ORNL) will coordinate the hotel and room reservations and will notify the committee members.

The meeting was adjourned at 1:00 pm.

The minutes of the meeting were prepared by Dr. Po-Yung Lu , ORNL.

ACRONYMS

AAPCO	Association of American Pesticide Control Officials
ACOEM/ACEP	American College of Occupational Environmental Medicine/ American College of Energy Physicians
AFL-CIO	American Federation of Labor & Congress of Industrial Organizations
ATSDR	Agency for Toxic Substances & Disease Registry
BNL	Brookhaven National Laboratory
CDC	Center for Diseases Control
DoD	Department of Defense
DOE	Department of Energy
DOT	Department of Transportation
EPA	Environmental Protection Agency
FEMA	Federal Emergency Management Agency
FDA	Food and Drug Administration
ICEH	International Center for the Environment and Health
NESCAUM	North Eastern States for Coordinated Air Use Management
NFPA	National Fire Protection Association
NIOSH	National Institute of Safety and Health
ORNL	Oak Ridge National Laboratory
OSHA	Occupational Safety and Health Administration
STAPPA/ALAPCO	State and Territorial Air Pollution Program Administrators/ Association of Local Air Pollution Control Officials

LIST OF ATTACHMENTS

The attachments were distributed during the meeting and will be filed in the EPA Docket Office.

Attachment 1. NAC meeting 1 agenda
Attachment 2. Attendee list
Attachment 3. Data analysis for Fluorine
Attachment 4. Data analysis for Methylmercaptan
Attachment 5. Data analysis for Hydrazine
Attachment 6. Data analysis for Ammonia
Attachment 7. Public comment on Ammonia by RAM TRAC Corporation

Attachment 1

National Advisory Committee For Acure Exposure Guidelines Levels (AEGLs) for Hazardous Substances

NAC/AEGL -1

Agenda

Wednesday, June 19, 1996

9:00AM	Informal gathering to get acquainted (refreshments)
10:00	Welcome and Introductions (R.L. Garrett and G. Rush)
10:30	Orientation - history, objectives, function, process and procedures of the committee (R.L. Garrett)
11:15	Orientation - Application of the AEGLs in the public and private sectors (P. Tobin)
12:00	Lunch
1:15	Orientation - NAS guidance for developing AEGLs (J. Doull)
2:15	Presentation and discussion - fluorine (E. Falke)
3:15	Discussion of fluorine contiunued and further discussion of Methodology and development of AEGLs
5:00	Adjourn for the day
5:30	Social hour
Evening-	Optional informal discussions on developing AEGL values

Thursday, June 20

8:30	Review of Wednesday deliberations
9:00	Presentation and discussion of methyl mercaptan (D. Hansen)
10:30	Break
10:45	Discussion of methyl mercaptan continued and further discussion of methodology and development of AEGLs
12:00	Lunch
1:15	Presentation and discussion of hydrazine (R. Thomas)
3:00	Break
3:15	Discussion od hydrazine continued and further discussion of methodology and development of AEGLs
5:00	Adjourn for the day
5:30	Social hour
Evening	Optional informal discussions on developing AEGL values

Friday, June 21

8:30	Housekeeping issues - future meetings, future chemicals and chemical manager assignments
9:00	Presentation and discussion of ammonia (L. Gephart)
10:30	Break
10:45	Discussion of ammonia continued and further discussion of methodology and development of AEGLs
12:00	Review of deliberations on all four chemicals and expections for next meeting
1:00	Adjourn

6/19/96

Attachment 2

Name	Phone	Fax
Paul Tobin	(202) 260-1736	(202) 260-0981
Richard Thomas	(703) 527-6500	(703) 243-0013
Larry Gephart	908 873-6319	908 873-6009
Dodi Hansen	(516) 344-7535	(516) 344-3284
Tom Tuccinardi	(301) 903-2484	(301) 903-8714
Lynn Beasley	703 603-9086	703-603-9104
Jim Holler	(404) 639-6308	(404) 639-6315
MARK A MCCLANAHAN	770-488-7297	770-488-7335
ROBERT A. YOUNG	(423) 574-4573	(423) 574-9888
Kowetha A. Davidson	(423) 574-7799	(423) 574-9888
SYLVIA TALMAGE	(423) 574-7758	(423) 574-9888
Po-Zung Su	(423) 574-7803	(423) 574-9888
James C. Norris	423-576-6237	423-574-9888
Zarina Post	(512) 239-1332	(512) 239-1794
BILL BRESS	(802) 863-7220	(802) 863-7425
Dan Guth	919-541-4930	919-541-0245
George Alexeeff	(510) 540-2907	(510) 540-2923
George Rodgers	502 629-5834	502-629-5828
		812-951-3020
Ernest V. Falke	(202) 260-3433	(202) 260-1279
ROGER GARRETT	202-260-4302	202-260-1279
GEORGE CUSHMAC	202-366-4545	202-366-3753
GEORGE RUSCH	201-455-3672	201-455-5405
JONATHAN BORAK		

Private Sector 6-19-96

D Sandler	NAS/NRC
Robert Venezia	CMA
John Meagher	AIHA
Jim Holler	ATSDR
Lou Bernstein	CTRAPS
Jim Egenrieder	Agricultural Retailers Ass
Robert Baltz	API
Kathleen Bailey	IIAR
Sara Thurin Rollin	BNA

AEGL VALUES FOR FLUORINE

Attachment 3

- ENDPOINTS OF CONCERN
- SELECTION OF THE KEY STUDY
- SCALING BETWEEN ANIMAL EXPOSURE LEVELS AND HUMAN EXPOSURE LEVELS FOR AN EFFECT
  - ORIGINAL DRAFT
  - MODIFIED DRAFT
- UNCERTAINTY FACTOR TO ACCOUNT FOR EXTRAPOLATION FROM ANIMAL TO HUMAN EXPOSURE VALUES
- UNCERTAINTY FACTOR TO ACCOUNT FOR SENSITIVE HUMAN POPULATIONS
- SCALING BETWEEN TIME VALUES FOR AN EFFECT
- STEEPNESS OF THE DOSE RESPONSE CURVE
- DEVELOPMENT OF AEGL VALUES
- LAUGH TEST

EFFECT OF CONCERN

- SEVERE CORROSION OF THE LUNG
- RESPIRATORY FAILURE FROM PULMONARY DAMAGE:
  - EDMA
  - EMPHYSEMA
  - HEMORRHAGE
  - BRONCHIAL AND ALVEOLAR NECROSIS

ORIGINAL DRAFT DETERMINATION OF HUMAN EQUIVALENT CONCENTRATION

$$NOAEL_{[HEC]} = NOAEL_{[MOUSE]} \times RGDR_r$$

$NOAEL_{[HEC]}$ = DOSIMETRICALLY ADJUSTED EXPOSURE LEVEL TO OBTAIN AN ANALOGOUS EFFECT IN HUMANS

$NOAEL_{[MOUSE]}$ = NO OBSERVED EFFECT LEVEL IN THE MOUSE

$RGDR_r$ = RATIO OF THE REGIONAL GAS DOSE IN MOUSE (RGD_M) TO THE REGIONAL GAS DOSE (RGD_H) IN HUMANS

RGD_H = MINUTE $VOLUME_H$ / SURFACE AREA OF THE $REGION_H$

ACTUAL Vs PREDICTED LC_{50} VALUES (ppm) USING ABOVE FORMULA

SPECIES	ACTUAL 30 MINUTE LC_{50}	30 MINUTE LC50 PREDICTED FROM MOUSE LC_{50}	ACTUAL 60 MINUTE LC_{50}	60 MINUTE LC50 PREDICTED FROM MOUSE LC_{50}
MOUSE	225	*225*	150	*150*
RAT	270	*306*	185	*204*
GUINEA PIG			170	*409*
RABBIT	270	*792*		*528*
HUMAN		*740*		*493*

Species	Minute volume M3/min (Vm)	respiratory tract surface area cm2 (S)	RGD Minute Volume (Vm)/Surface Area (S)	RGDm/RGDx = RGDR::: NOAELx = NOAELm * RGDR
Rat	2.1107E-04	3,440	6.14E-08	1.36
Mouse	4.2607E-05	510	8.35E-08	1.00
Guinea Pig	2.8284E-04	9,230	3.06E-08	2.73
Human	1.3800E-02	543,400	2.54E-08	3.29
Rabbit	1.3996E-03	59,000	2.37E-08	3.52
Dog				
Hamster	5.8783E-05	3,000	1.96E-08	4.26

ADJUSTMENT OF AEGL VALUES FOR EXPOSURE TIMES

- SCALING ACROSS TIME IS BASED UPON THE RELATIONSHIP BETWEEN ACUTE TOXICITY (CONCENTRATION) AND EXPOSURE DURATION.

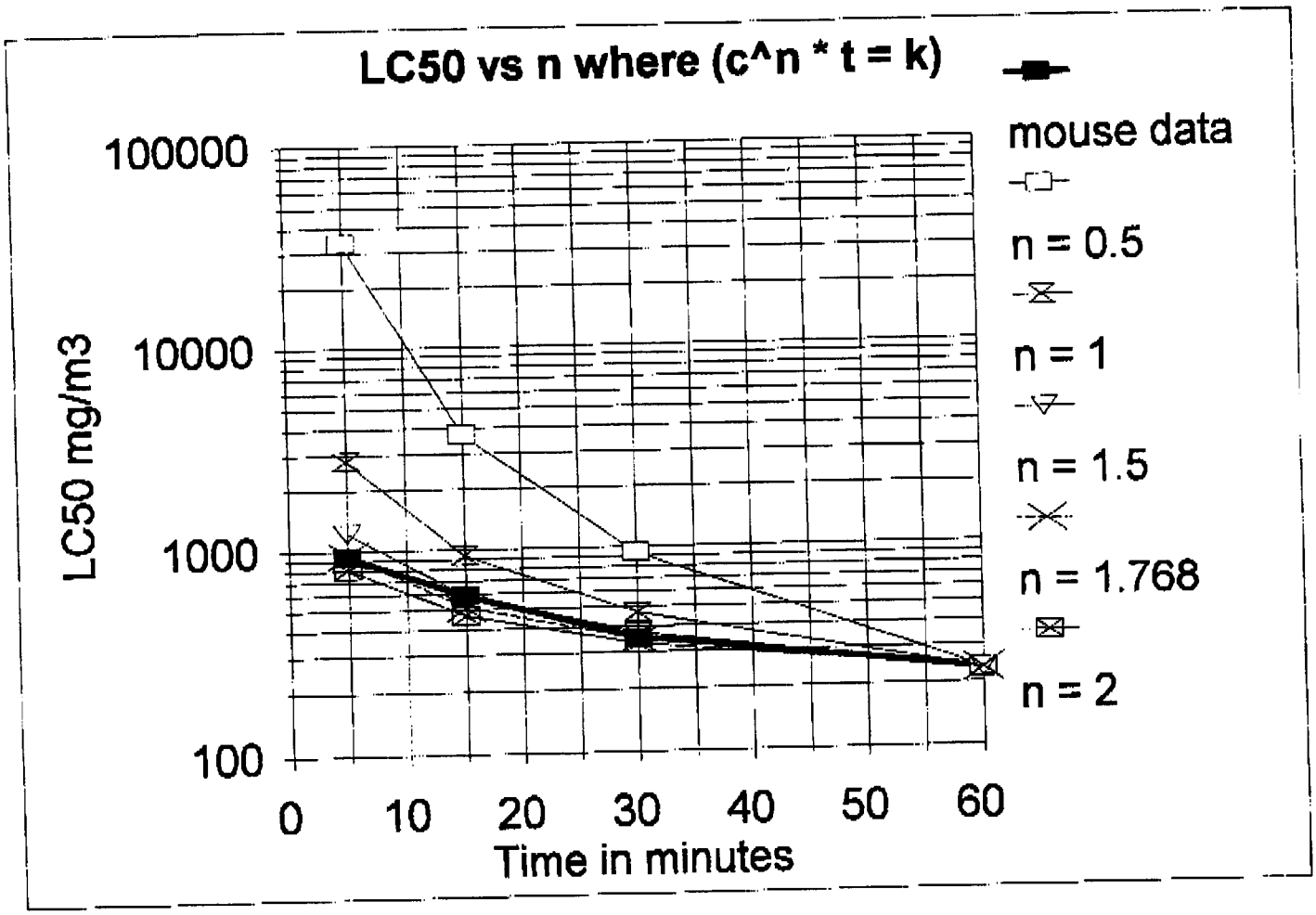
THE ONLY AVAILABLE DATA (LD_{50} DATA FOR THE RAT, MOUSE, AND GUINEA PIG FOR 5, 15, 30, AND 60-MINUTE EXPOSURE TIMES), SHOW THAT THE ASSOCIATION BETWEEN ACUTE TOXICITY (CONCENTRATION) AND EXPOSURE DURATION IS A LOGARITHMIC ONE AND THE EQUATIONS DERIVED FROM THE EMPIRICAL DATA BY REGRESSION ANALYSIS ARE EXPRESSED AS:

$$C^n \times t = k$$

C = concentration in mg/m^3

t = time in minutes

k = constant for the species and endpoint under consideration


LC50 vs n where (c^n * t = k)
mouse data
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n = 1.5
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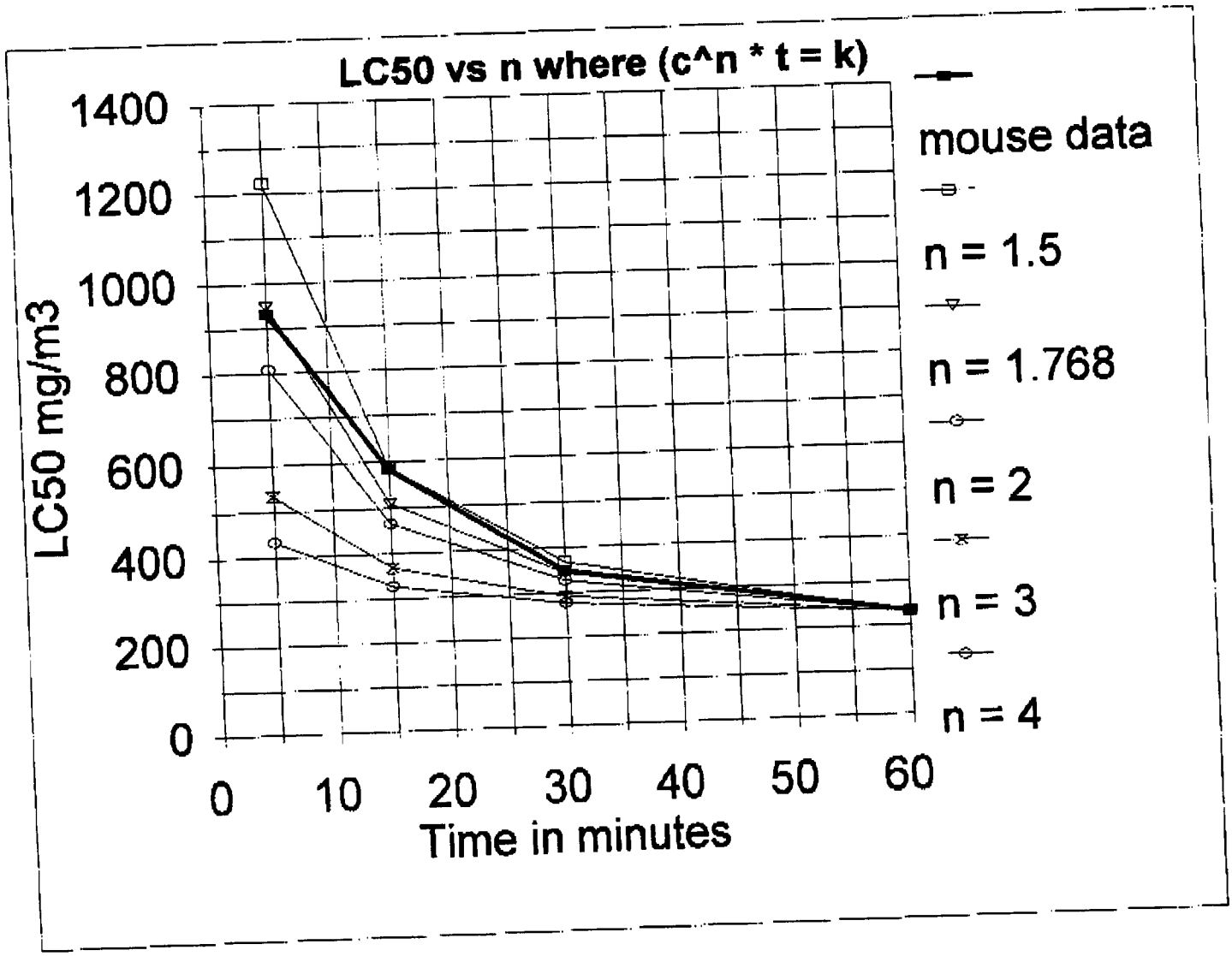
100
LC50 mg/m3
0
10
20
30
40
50
60
Time in minutes


LC50 vs n where (c^n * t = k)
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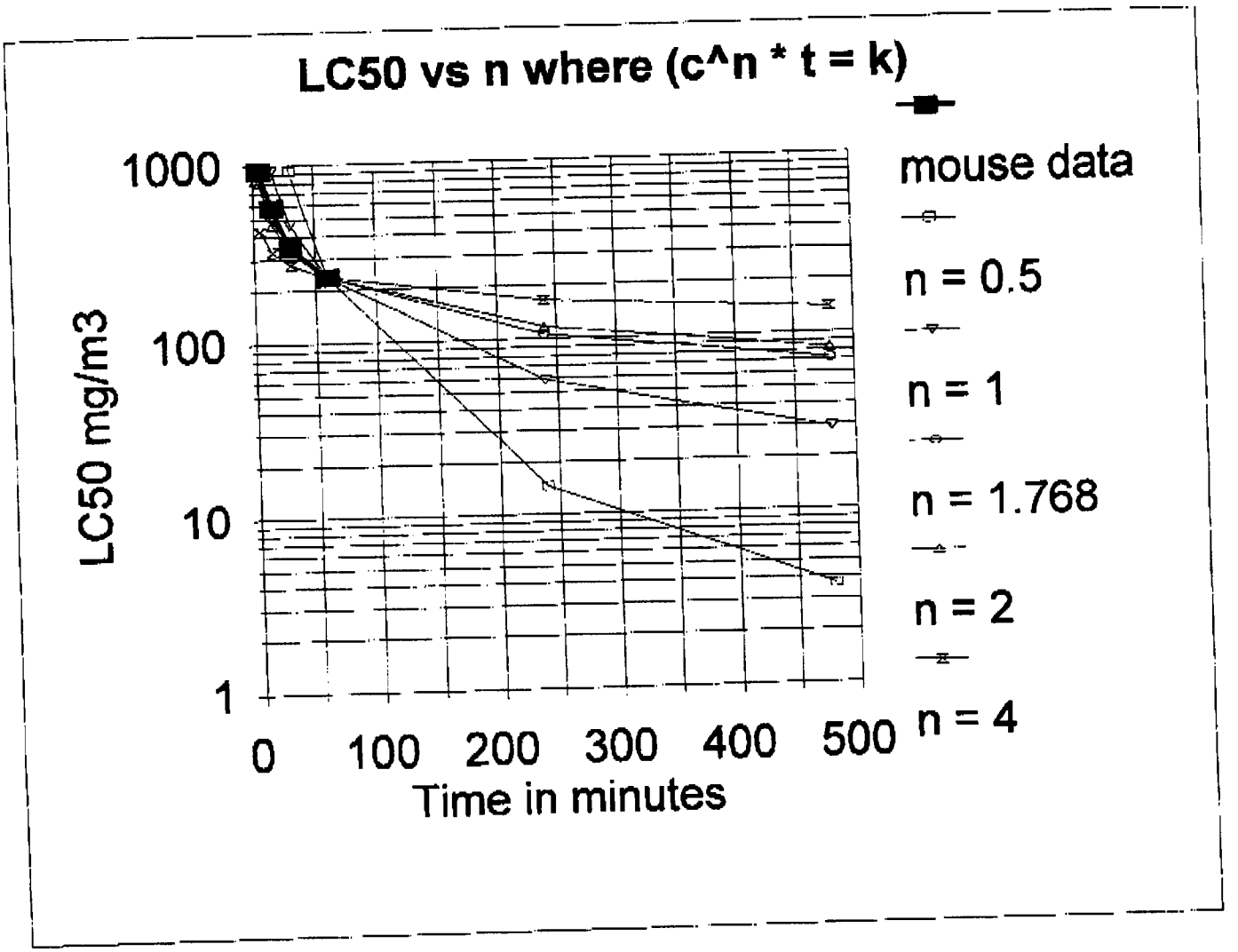
1200
1000
800
600
400
200
0
LC50 mg/m3
0
10
20
30
40
50
60
Time in minutes
mouse data
n = 1.5
n = 1.768
n = 2
n = 3
n = 4


LC50 vs n where (c^n * t = k)
1000
100
10
1
LC50 mg/m3
0
100
200
300
400
500
Time in minutes
mouse data
n = 0.5
n = 1
n = 1.768
n = 2
n = 4

AEGL-1

THE EXPOSURE CONCENTRATION OF AN AIRBORNE SUBSTANCE BELOW WHICH EXPOSED PERSONS MIGHT COMPLAIN OF ODOR, TASTE, SLIGHT OR MILD SENSORY IRRITATION, BUT ABOVE WHICH EXPOSED PERSONS MIGHT REQUEST ASSISTANCE ALTHOUGH THEIR CONDITION DOES NOT IMPAIR ESCAPE, PRODUCE DISABLEMENT OR RESULT IN PERMANENT OR LONG-LASTING EFFECTS.

AEGL 1	
TIME	AEGL-1 VALUE
30 Minute	5 mg/m3 (3.2 ppm)
1 Hour	3.4 mg/m3 (2.2 ppm) calculated from $c^{1.768} * t = k$
4 Hour	1.5 mg/m3 (1.0 ppm) calculated from $c^{1.768} * t = k$
8 Hour	1.0 mg/m3 (0.7 ppm) calculated from $c^{1.768} * t = k$

AEGL-2

THE EXPOSURE CONCENTRATION BELOW WHICH THE DIRECT TOXIC EFFECTS ARE UNLIKELY TO LEAD TO DISABILITY, PERMANENT OR LONG-LASTING EFFECTS, BUT ABOVE WHICH IMPAIRMENT OF ESCAPE OR PERMANENT OR LONG-LASTING EFFECTS OCCUR.

AEGL 2	
TIME	AEGL 2 VALUE
30 Minute	17 mg/m3 (11 ppm)
1 Hour	8 mg/m3 (5.2 ppm)
4 Hour	3.7 mg/m3 (2.4 ppm) calculated from $c^{1.768} * t = k$
8 Hour	2.5 mg/m3 (1.6 ppm) calculated from $c^{1.768} * t = k$

AEGL-3

THE EXPOSURE CONCENTRATION BELOW WHICH DEATH OR LIFE-THREATENING EFFECTS ARE UNLIKELY (INCLUDING SUSCEPTIBLE, BUT NOT HYPER-SUSCEPTIBLE INDIVIDUALS), BUT ABOVE WHICH LIFE-THREATENING EFFECTS OCCUR IMMEDIATELY OR SOON AFTER EXPOSURE.

AEGL 3	
TIME	AEGL 3 VALUE
30 Minute	29 mg/m3 (19 ppm)
1 Hour	19 mg/m3 (12 ppm)
4 Hour	8.7 mg/m3 (5.6 ppm) calculated from $c^{1.768} * t = k$
8 Hour	5.9 mg/m3 (3.8 ppm) calculated from $c^{1.768} * t = k$

TABLE SUMMARY/RELATIONSHIP OF AEGL VALUES				
Classification	30-Minute	1-Hour	4-Hour	8-Hour
AEGL-1 (Reversible, nondisabling)	5 mg/m^3 (3.2 ppm)	3.4 mg/m^3 (2.2 ppm)	1.5 mg/m^3 (1.0 ppm)	1.0 mg/m^3 (0.7 ppm)
AEGL-2 (Disabling, Irreversible)	17 mg/m^3 (11 ppm)	8 mg/m^3 (5.2 ppm)	3.7 mg/m^3 (2.4 ppm)	2.5 mg/m^3 (1.6 ppm)
AEGL-3 (Life-threatening)	29 mg/m^3 (19 ppm)	19 mg/m^3 (12 ppm)	8.7 mg/m^3 (5.6 ppm)	5.9 mg/m^3 (3.8 ppm)

- ACGIH TLV 8 HR TWA = 1 PPM (1.6 MG/M^3)
- ACGIH TLV STEL 15 MIN 2 PPM (3.1 MG/M^3)
- NIOSH/OSHA 8 HR 0.1 PPM
- NIOSH/IDLH 30 MINUTE = 25 PPM
- EPA LOC 60 MIN 2.5 PPM (0.039
- NRC (1984) EEGL 10 MINUTE = 15 PPM
  30 MINUTE = 10 PPM
  60 MINUTE = 7.5 PPM
- AIHA ERPG-1 1 HOUR = 2 PPM
  ERPG-2 1 HOUR = 7.5 PPM
  ERPG-3 1 HOUR = 10 PPM

Methyl Mercaptan

- Comments from 2 reviewers

- Manufacturer: better data available, request time to comment

Propose:

-> Review now as is

-> Incorporate changes & additional data in revision

-> Resolution @ next AEGL meeting

TABLE 1

PHYSIOCHEMICAL DATA		
COMMON NAME	METHYL MERCAPTAN	REFERENCE
Synonyms	Methanethiol, Mercaptomethane, Methyl Sulfhydrate, Thiomethyl Alcohol	ACGIH, 1991
CAS Registry No.	74-93-1	ACGIH, 1991
Chemical Formula	CH_4S	Sittig, 1985
Molecular Weight	48.11	ACGIH, 1991
Physical State	Colorless gas	Sittig, 1985
Vapor Pressure	1692 torr	ACGIH, 1991
Specific Gravity	0.8665 at 20°C	ACGIH, 1991
Melting/Boiling/Flash Point	-123°C/6.2°/ <-17.78°C(open cup)	ACGIH, 1991
Solubility	Soluble in water (23.3 g/L at 20°C), very soluble in alcohol and ether	ACGIH, 1991
Conversion factors in air	1 mg/m^3 = 0.51 ppm 1 ppm = 1.96 mg/m^3	AIHA, 1989
Incompatibility	Strong oxidizers and bleaches	Sittig, 1985
Odor Threshold	0.0016 ppm	ACGIH, 1991

SUMMARY OF PROPOSED AEGL VALUES FOR METHYL MERCAPTAN					
Classification	30-min	1-hr	4-hr	8-hr	Endpoint (Reference)
AEGL-1 (Nondisabling)	0.6 ppm	0.4 ppm	0.2 ppm	0.17 ppm	Human exposure to ethyl mercaptan (Pickler, 1918)
AEGL-2 (Disabling)	ND	ND	ND	ND	No data
AEGL-3 (Lethality)	150 ppm	100 ppm	50 ppm	25 ppm	Lethality in rats (Tansy et al., 1981)

ND: not determinable with existing data

TABLE 3

LETHALITY DATA FOR ANIMALS EXPOSED TO METHYL MERCAPTAN		
Species	Effect	Reference
Rat	675 ppm, 4-hr LC_{50} 600 ppm, 4-hr; lowest reported lethality 400 ppm, 4-hr; no lethality	Tansy et al, 1981
Rat	~10,000 ppm, 14-min; lethality	Ljunggren and Norberg, 1943
Rat	1600 ppm, 15-min CD_{50} [a]	Zieve et al., 1974
Rat	4.52 ppm. 4-hr LC_{50}	Seluzhitsky, 1972
Mouse	1664 ppm, 4-hr LC_{50}	Horiguchi, 1960
Mouse	3.3 ppm, 2-hr LC_{50}	Seluzhitsky, 1972

[a]Comatose dose

TABLE 6

COMPARISON OF THE PROPOSED AEGL-1 VALUES WITH OTHER EXPOSURE CRITERIA						
Time	AEGL-1	ACGIH TLV	NIOSH IDLH	NIOSH REL	ERPG-1	OSHA PEL
30-min	0.6 ppm		150 ppm			
1-hr	0.4 ppm				0.005 ppm	
4-hr	0.2 pppm					
8-hr	0.17 ppm					
8-hr		0.5 ppm (TWA)		0.5 ppm (ceiling)		10 ppm (ceiling)

5.3 DERIVATION OF AEGL-1

Scaled value = $[([\text{experimental value}]^n \times EET)/ASET]^{1/n}$

Where:

EET	=	experimental exposure time
ASET	=	target exposure time
n	=	1.5 to 3.5 (Wilson, 1991), for AEGL calculations a midpoint of 2 is used.

- Hydrazine -

Attachment 5

1. Molecular formula NH_2NH_2

2. Highly Reactive reducing agent.
   - Colorless liquid at room temp.
   - Fumes in air with extremely irritating odor (ammoniacal odor).
   - Rocket propellant
   - Manufacture of Pharmaceuticals
   - F-16 power unit with water. (Drives a gas turbine.)
   - Explodes on contact with metal oxides, salts etc.
   - Identified in nature as a product of nitrogen fixation.
   - Synthesis of maleic hydrazide.

3. Very soluble in water

4. High reactivity is a problem major ~~with~~ in inhalation studies.

Effects in Humans

1. Vapor is extremely irritating to eyes, nose, and throat.
2. Vapor produced immediate and violent irritation of nose and throat.
3. Itching, burning of eyes and swelling of eyelids developed.
4. Transitory blindness with moderate exposure (lasted as long as a day.)
5. Lacrimatorie, mutagenic and carcinogenic (lab animals)
6. Median detectable odor 3-4 ppm.
7. IARC 2B "Probably carcinogenic to humans."

Animal Studies

Acute studies have been conducted in several species.

Little margin between lethal effects and non-lethal effects. (Steep slope.)

Laboratory studies indicate carcinogenicity in mice, rats, hamsters and dogs.

Exposure information is inconsistent due to its high reactivity.

Effects
- Upper respiratory irritation.
- Irregular respiration.
- Appetite loss & weight loss
- Tremors
- Fatigue
- Salivation, vomiting, diarrhea
- Liver & kidney effects.

Development of AEGL Levels

AEGL development is based on animal acute studies.

AEGL-1 House (1964) monkeys

AEGL-2 Becker (1991) Rats

AEGL-3 Jacobson (1955) lethality

Dr. Robert Young - ORNL -

AMMONIA

Attachment 6

- Water soluble, reactive gas

- Primary toxic action: irritation of eyes, mouth, upper respiratory tract
  -Pulmonary edema in humans exposed to high concentrations in accidents

- Overall toxicity database is adequate for establishing AEGLs
  -Minor exception LC_{50} data for 4 and 8-hours

AEGL 3

- Two approaches were used, yielding comparable AEGLs

  1. Accident reconstruction reported by Pedersen and Selig (1989), used with regression equation employed to scale to different time frames

  2. Acute inhalation toxicity data used in conjunction with uncertainty factor
     -Similar results across species; mild species differences
     -Appleman (1982) provides excellent data for most durations of interest
     -Regression equations used: $C^n \times t = k$, where $n = 2$
     -Conversion to Human Equivalent Concentration
     -Uncertainty factor = 20 or 30 applied to $LC_{.1}$ or LC_{10}, respectively
       ▸data on glottis closure indicate 3-fold inter-individual difference in response
       ▸species differences in response to irritants considered to be $< 10x$

AEGL 2

- Five studies in human volunteers considered/used
  -Data by Verberk (1977) considered most useful

- Focus was on intolerable rather than irreversible health effects

- Scaling equation by ten Berge et al. ($C^n \times t = k$) used to scale to different time frames

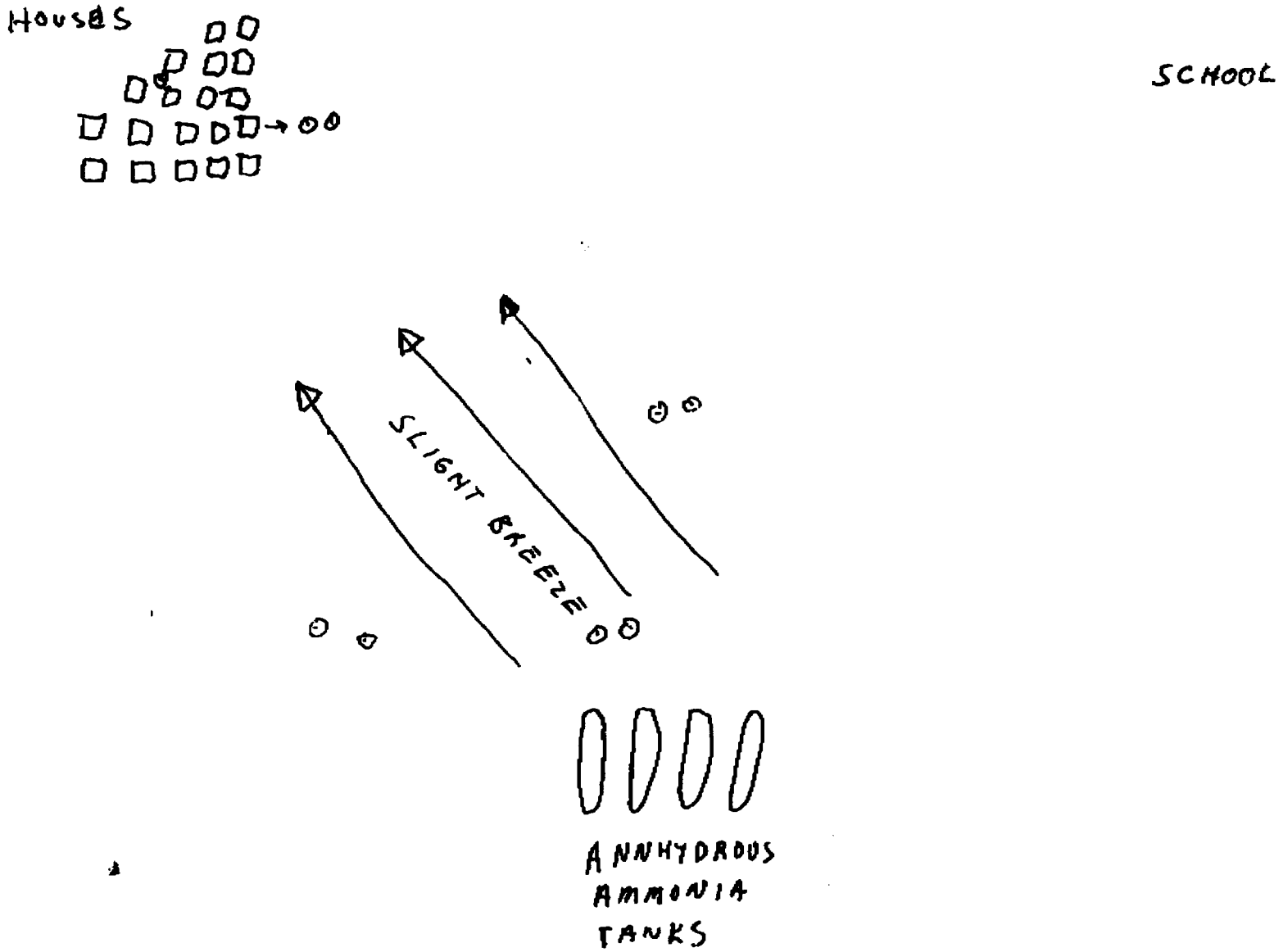
- RD_{50} in mice also considered

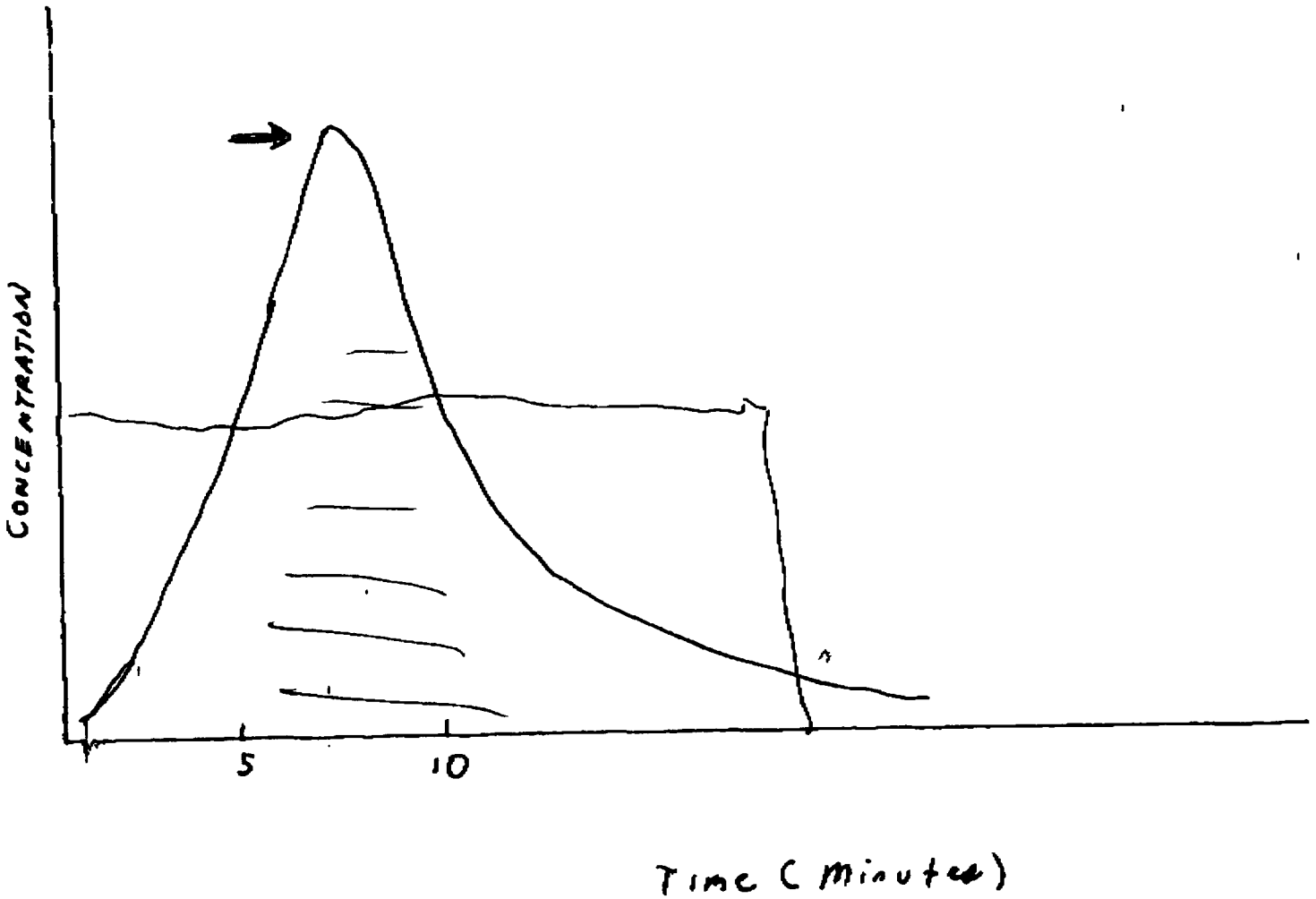
AEGL 1

- Studies in human volunteers used
  -Odor thresholds
  -Data on levels associated with transient irritation

- Scaling equation by ten Berge et al. used to scale to different time frames

Potchefstroom Incident




CONCENTRATION
5
10
Time (Minutes)

PROPOSED AEGL VALUES FOR AMMONIA					
Classification	Exposure Duration				Endpoint (Reference)
	30 min	1 hour	4 hours	8 hours	
AEGL-3 (lethality)	1700 ppm	1200 ppm	300 ppm	200 ppm	Lethality/LC_{50} (Appelman et al., 1982)
AEGL-2 (disabling)	200 ppm	150 ppm	75 ppm	50 ppm	Disabling or incapacitating (entire data set)
AEGL-1 (nondisabling)	50 ppm	35 ppm	25 ppm	25 ppm	odor and irritation to eyes, throat, and respiratory tract (entire data set)

TABLE 3. SCORES FOR SUBJECTIVE RESPONSES OF EXPERT AND NONEXPERT SUBJECTS EXPOSED TO AMMONIA[A]

Response	50 ppm	80 ppm	110 ppm[c]	140 ppm[d]
Smell	1-3[b], ½ h 1-4, 1 h 1-4, 2 h	1-4, ½ h 1-4, 1 h 1-4, 2 h	2-4, ½ h 2-4, 1 h 2-4, 2 h	1-4, ½ h 1-4, 1 h 1-4, 2 h
Eye irritation	0-3, ½ h 0-3, 1 h 0-3, 2 h	0-4, ½ h 0-3, 1 h 0-4, 2 h	0-4, ½ h 0-4, 1 h 0-4, 2 h	1-5, ½ h 1-5, 1 h 1-5, 2 h
Nose irritation	similar to eyes	similar to eyes	similar to eyes	similar to eyes
Throat irritation	0-2, ½ h 0-3, 1 h 0-3, 2 h	0-3, ½ h 0-3, 1 h 0-4, 2 h	0-4, ½ h 0-4, 1 h 0-4, 2 h	0-5, ½ h 0-5, 1 h 0-5, 2 h
Urge to cough	0-1, ½ h 0-2, 1 h 0-2, 2 h	0-2, ½ h 0-2, 1 h 0-4, 2 h	0-2, ½ h 0-3, 1 h 0-4, 2 h	0-5, ½ h 0-3, 1 h 0-4, 2 h
Irritation of chest	similar to cough	similar to cough	similar to cough	similar to cough
General discomfort	0-1, ½ h 0-1, 1 h 0-2, 2 h	0-3, ½ h 0-3, 1 h 0-3, 2 h	0-3, ½ h 0-3, 1 h 0-4, 2 h	0-4, ½ h 0-5, 1 h 0-5, 2 h

Source: Verberk, 1977

[a]Expert subjects were familiar with the effects of ammonia, the nonexpert subjects were not.

[b]Based on a scale of 1-5: 0 = sensation; 1 = just perceptible; 2 = distinctly perceptible; 3 = nuisance; 4 = offensive; 5 = unbearable.

[c]Only four of the nonexpert subjects tolerated the ammonia for 1 h.

[d]None of the nonexpert subjects tolerated the ammonia for 2 h, the upper range of the score was chosen at that recorded at 1 h or 110 ppm after 2 h (urge to cough).

TABLE 4. SUMMARY OF NONDISABLING AND REVERSIBLE EFFECTS OF INHALING AMMONIA

Conc.	Duration of Exposure	Effect[a]	Reference
30 ppm	10 min	moderately intense to penetrating odor; barely detectable irritation	MacEwen, 1970
34 ppm	5 min	nasal dryness	Industrial Bio-Test Lab, 1973
50 ppm	5 min	nasal dryness	Industrial Bio-Test Lab, 1973
50 ppm	10 min	highly penetrating odor; moderate irritation	MacEwen, 1970
50 ppm	30 min	moderately intense odor; moderate irritation to eyes and nose, mild irritation to throat and chest, slight urge to cough, slight general discomfort	Verberk, 1977
50 ppm	1 h	highly intense odor; moderate irritation to eyes, nose, throat, and chest, mild urge to cough, slight general discomfort	Verberk, 1977
50 ppm	2 h	highly intense odor; moderate irritation to eyes, nose, throat, and chest, mild urge to cough, mild general discomfort	Verberk, 1977
72 ppm	5 min	nasal, eye, and throat irritation	Industrial Bio-Test Lab, 1973
80 ppm	30 min	highly intense odor; highly intense eye and nose irritation, moderate throat and chest irritation; mild urge to cough, moderate general discomfort	Verberk, 1977
80 ppm	1 h	highly intense odor; moderate eye, nose, throat, and chest irritation, mild urge to cough, moderate general discomfort	Verberk, 1977
80 ppm	2 h	highly intense odor; highly intense eye, nose, throat, and chest irritation, highly intense urge to cough, and moderate general discomfort	Verberk, 1977
110 ppm	30 min	highly intense odor; highly intense eye, nose, throat, and chest irritation, mild urge to cough, and moderate general discomfort	Verberk, 1977
110 ppm	1 h	highly intense odor; highly intense eye, nose, throat, and chest irritation, moderate urge to cough, and moderate general discomfort	Verberk, 1977
110 ppm	2 h	highly intense odor; highly intense eye, nose, throat, chest irritation, urge to cough, and general discomfort	Verberk, 1977

TABLE 4. CONTINUED

Conc.	Duration of Exposure	Effect	Reference
140 ppm	30 min	highly intense odor; unbearable eye, nose, throat, and chest irritation, mild urge to cough, moderate general discomfort	Verberk, 1977
140 ppm	1 h	highly intense odor; unbearable eye, nose, throat, and chest irritation, moderate urge to cough, moderate general discomfort	Verberk, 1977
140 ppm	2 h	highly intense odor; unbearable eye and nose, highly intense throat and chest irritation, highly intense urge to cough, unbearable general discomfort	Verberk, 1977
143 ppm	5 min	nose, eye, throat, and chest irritation, lacrimation	Industrial Bio-Test Lab, 1973
500 ppm	15-30 min	nose and throat irritation, nasal dryness and stuffiness, excessive lacrimation, hyperventilation, unbearable	Silverman et al., 1949
570 ppm	single breath	threshold for reflex glottis closure, 21-30-year old subjects	Erskine et al., 1993
1000 ppm	single breath	threshold for reflex glottis closure, 60-year old subjects	Erskine et al., 1993
1000 ppm	NR	immediate urge to cough	Silverman et al., 1949
1790 ppm	single breath	threshold for reflex glottis closure, 86-90-year old subjects	Erskine et al., 1993

[a]Most severe response of subjects; score of 1 = slight, 2 = mild, 3 = moderate, 4 = highly intense, 5 = unbearable.

TABLE 5. ACUTE LETHALITY DATA FOR MALE AND FEMALE RATS EXPOSED TO AMMONIA

Experimental Conc.		Exposure time (min)	$C^n \times t^a$ (mg/m³•min)	Mortality Rate[c]	LC_{50}[c]	
mg/m³	ppm				mg/m³	ppm
20,950	29,334	10	5.36E+09	0/10	28,130 (9.71E+09)[b]	39,382 (1.92E+11)[b]
23,380	32,732	10	6.68E+09	1/10		
26,410	36,974	10	8.55E+09	6/10		
27,220	38,108	10	9.09E+09	5/10		
37,820	52,948	10	1.77E+10	9/10		
18,290	25,606	20	8.14E+09	3/10	19,960 (9.71E+09)	27,944 (1.92E+10)
19,030	26,642	20	8.82E+09	1/10		
19,450	27,230	20	9.22E+09	7/10		
20,420	28,588	20	1.02E+10	6/10		
23,200	32,480	20	1.32E+10	9/10		
12,620	17,668	40	7.69E+09	2/10	14,170 (9.72E+09)	19,838 (1.92E+10)
13,410	18,774	40	8.70E+09	5/10		
15,870	22,218	40	1.22E+10	5/10		
16,290	22,806	40	1.29E+10	8/10		
16,840	23,576	40	1.38E+10	7/10		
9,870	13,818	60	7.03E+09	3/10	11,590 (9.72E+09)	16,226 (1.92E+10)
10,230	14,322	60	7.55E+09	4/10		
11,300	15,820	60	9.23E+09	5/10		
12,500	17,500	60	1.13E+10	6/10		
13,240	18,536	60	1.27E+10	7/10		

Source: Appelman et al., 1982

[a]Correlation of $c^n \times t$, where c = concentration in mg/m³, n = 2.02, and t = time (Appelman et al., 1982).

[b]Values in parentheses are $c^{2.02} \times t$.

[c]Data for male and female rats combined.

TABLE 8. ESTIMATES OF LETHAL CONCENTRATIONS OF AMMONIA TO HUMANS

Probability of Mortality	Exposure Time (min)	Concentration (ppm)		
		General Pop.[a]	Sensitive Pop.[b]	Sensitive Pop.[c]
0.01	30	6205	4744	2068
	60	4388	3356	1463
	240	2194	1677	731
	480	1551	1186	517
0.001	30	5047	4183	1682
	60	3569	2958	1190
	240	1784	1479	595
	480	1262	1046	421

[a]Based on the probit equation for the general population (Probit = 1.85 ln D - 35.9).
[b]Based on the probit equation for the sensitive subpopulation (Probit = 3.04 ln D -59.1).
[c]Based on an uncertainty factor of 3 to extrapolate from the general to sensitive subpopulation.

TABLE 9. ESTIMATES OF LETHAL CONCENTRATIONS OF AMMONIA BASED ON ANIMAL DATA

Probability of Mortality	Species	Exposure Time (min)	Concentration (Animal)[a]		HEC[b]
			mg/m^3	ppm	ppm
0.1 (LC_{10})	Rat	30	12,380	17,703	49,037
		60	8,780	12,555	34,777
		240	4,416	6,315	17,493
		480	3,132	4,479	12,407
0.01 (LC_{01})	Rat	30	9,884	14,134	39,151
		60	7,010	10,024	27,766
		240	3,526	5,042	13,966
		480	2,500	3,575	9,903
0.001 ($LC_{0.1}$)	Rat	30	8,384	11,989	33,210
		60	5,945	8,501	23,548
		240	2,990	4,276	11,845
		480	2,121	3,033	8,401
0.1 (LC_{10})	Mouse	30	3,421	4,892	1,957
		60	2,443	3,493	1,397
		240	1,246	1,782	713
		480	890	1,273	509
0.01 (LC_{01})	Mouse	30	2,870	4,104	1,642
		60	2,050	2,932	1,173
		240	1,045	1,494	598
		480	746	1,067	427
0.001 ($LC_{0.1}$)	Mouse	30	2,525	3,611	1,444
		60	1,803	2,578	1,031
		240	919	1,314	526
		480	656	938	375

[a]Concentration derived using ten Berge et al. (1986) regression coefficients: $b_0 = 47.8$, $b_1 = 4.64$, and $b_2 = 2.30$ (rats); $b_0 = 54.5$, $b_1 = 5.95$, and $b_2 = 2.89$ (mouse).
[b]HEC (human equivalent concentration) calculated based on regional gas dose ratio (RGDR) , which is the ratio of the minute volume/surface area of the pulmonary region for animals and humans.

RAM TRAC Corporation

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Attachment 7

ACUTE INHALATION RISKS POTENTIALLY POSED BY ANHYDROUS AMMONIA

prepared by

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EXECUTIVE SUMMARY

The present investigation was undertaken to assess risks to human health potentially posed by inhalation of anhydrous ammonia. It revises an earlier, preliminary *'White Paper'* which elucidated ammonia toxicity benchmarks based upon a survey of available, mostly secondary literature. However, that report raised questions about the reliability of much of the current literature, given its apparent reliance on only a small number of primary research or clinical reports, all of which are quite old. The present investigation critically evaluates the ammonia database, and incorporates newer data derived from ammonia accident reconstructions and state-of-the-art air modeling to quantify concentrations to which victims were exposed. Thus, based upon such refined analyses, the findings made and conclusions drawn in the earlier *White Paper* have been significantly modified by the present investigation.

The present investigation assumes that gaseous anhydrous ammonia is released in the vicinity of employees wearing no protective breathing apparatus, or that such releases migrate beyond a facility fence line where unprotected members of the general public may encounter it. Appropriate safety and uncertainty factors are applied to assure conservatism in deriving toxicological benchmarks, thereby protecting subpopulations of people who may be more sensitive to ammonia inhalation than is typical for members of the general public. Charts are presented giving exposure times below which adverse health effects are unlikely to occur among individuals occupationally or environmentally exposed to anhydrous ammonia at selected concentrations in air. Table 1 summarizes the ammonia toxicity benchmarks derived in the present investigation.

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Table 1. Ammonia Acute Inhalation Toxicology Benchmarks*

data category	exposure concentration (ppm)	exposure duration (minutes)
standards and guidelines		
OSHA TWA-TLV	50	480
ACGIH STEL	35	15
NIOSH IDLH	300	30
odor threshold	0.04 to 57	> 0
reversible or clinically insignificant effects	50	10
irreversible effects	8,332	5
	1,387	30
	1,000	60
fatal exposure	> 33,737	5
	>5,623	30

**acronyms:*

ACGIH: American Conference of Governmental Industrial Hygienists
IDLH: Immediately Dangerous to Life or Health
NIOSH: National Institute for Occupational Safety and Health
OSHA: Occupational Safety and Health Administration
STEL: Short-Term Exposure Limit
TLV: Threshold Limit Value
TWA: time-weighted average

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Evaluation of odor and odor thresholds reveals that ammonia can be detected and recognized at concentrations at or below concentrations at which standards and guidelines control prolonged exposure. Coupled with these excellent warning properties, ammonia exhibits a vapor density lower than air, which would tend to cause it to rise out of the breathing zone if not physically confined. Ammonia released from liquid sources at cryogenic temperatures may be initially heavier than air until warmed by ambient heat.

Data extrapolated from animal bioassays suggest a significantly higher lethality concentration for humans than data modeled from human exposure studies. The discrepancy between these data sources was investigated, in part via consideration of accident reports involving human fatalities following industrial releases of anhydrous ammonia. Geometric mean concentrations of ammonia in air were inferred from accidents involving fatalities, where calculations suggested concentrations in excess of 35,000 ppm. Detailed evaluation of the Potchefstroom, South Africa, ammonia release, which killed 18 people in 1974, revealed that 83,322 ppm would be expected to kill half of individuals exposed for five-minutes, whereas no fatalities would be expected to result from exposure to 33,737 ppm for five minutes. The accident data are, therefore, consistent with human lethality concentrations derived based upon animal bioassays. Values inferred from human exposure studies are apparently flawed by reliance upon unusually old, quantitatively unreliable reports, or no reports.

Two measures were taken to assure that conclusions regarding ammonia-related lethality at higher concentrations are robust. First, the concentration associated with lethality corresponds to the lowest value suggested by animal bioassays and accident reports. The adopted value represents the no-fatality concentration derived from the

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Potchefstroom accident evaluation. Second, a program of telephone inquiries was undertaken to provide ample opportunity for representatives of insurance companies, chemical companies, trade associations, and federal agencies involved with anhydrous ammonia to refute the findings by citing data, or knowledge of the existence of data, which would either validate the human studies or reduce the lethality concentration derived from the animal bioassays and accident analyses. No such data emerged from this effort.

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INTRODUCTION

PURPOSE

The present investigation was undertaken to assess risks to human health potentially posed by inhalation of anhydrous ammonia. It revises an earlier, preliminary *'White Paper'* (57) which elucidated ammonia toxicity benchmarks based upon a survey of available, mostly secondary literature. However, that report raised questions about the reliability of much of the current literature, given its apparent reliance upon only a small number of primary research or clinical reports, all of which are quite old. The present investigation critically evaluates the ammonia database, and incorporates newer data derived from ammonia accident reconstructions and state-of-the-art air modeling to quantify concentrations to which victims were exposed.

Facility design and emergency planning must address the possibility of releases from such sources as the ammonia-rich working fluid employed in Kalina cycle power generation equipment, tanks storing anhydrous ammonia on-site, or vehicles transporting anhydrous ammonia to a site. Anhydrous ammonia also has significant uses as a refrigerant and in agriculture. This investigation identifies and critically evaluates reports of adverse health effects which have been associated with short-term exposure to ammonia, and concentrations of ammonia eliciting such effects, where known. It analyzes this data, and draws conclusions about the potential for health damage including, but not limited to, the possible risk of death of members of the general public in the vicinity of ammonia releases.

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SCOPE

This investigation assumes that gaseous anhydrous ammonia is released in the vicinity of employees wearing no protective breathing apparatus, or that such releases migrate beyond a facility fence line where unprotected members of the general public may encounter it. Ammonium, ammonium salts, and liquid (cryogenic) ammonia are excluded from consideration, as are cryogenic, high-pressure, and high temperature risks. Appropriate safety and uncertainty factors are applied to assure conservatism in deriving toxicological benchmarks, thereby protecting subpopulations of people who may be more sensitive to ammonia inhalation than is typical for members of the general public.

The National Institute for Occupational Safety and Health has downwardly revised the IDLH *(Immediately Dangerous to Life or Health)* value for anhydrous ammonia, from 500 to 300 parts per million (45, 46). However, the Agency has requested additional data, and an Agency representative indicated that reconsideration of the IDLH value may be imminent. The scope of regulatory review of ammonia toxicity may be broadened based upon the present investigation, which considers human clinical data, animal bioassay data, and reports of industrial ammonia release accidents to reconcile disparate toxicological benchmark values and synthesize them into a coherent and unified database.

This investigation develops and presents charts of exposure times below which adverse health effects are unlikely to occur among individuals occupationally or environmentally exposed to anhydrous ammonia at selected concentrations in air. This is equivalent to setting

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forth an upper time limit for reliably detecting the assumed ammonia release(s) and safely removing or otherwise protecting exposed or potentially exposed individuals. In some circumstances, such as facility planning, this information might eliminate the need for conducting complex air modeling associated with multiple scenarios of ammonia release. It might also set design and engineering limits, or demonstrate the adequacy of design and engineering already incorporated for ammonia containment and exposure management. Finally, the information might reduce the radius and area to be included in the Risk Management Plan (RMP) for proposed facilities using ammonia (78-80).

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METHODS

Information Acquisition

Information about toxic effects exerted by ammonia on humans and non-human species was obtained via three search procedures. Review articles and online databases were searched to acquire relevant information. Further searches were then undertaken for selected primary and secondary articles cited among the sources identified in the first phase. Principle resources for obtaining information by this procedure included in-house data sources, libraries, a bibliographic retrieval service, and on-line databases (such as 24, 26, 61). The third information acquisition procedure focused primarily upon determining an appropriate lethality value for anhydrous ammonia. The procedure was simply to systemicatically contact colleagues, and thereby identify additional contact individuals in chemical companies, insurance companies, trade associations, and government agencies.

Data Analysis

Acceptance criteria for input data for the present assessment were adopted, and articles were examined to identify toxicological information conforming with these criteria. Ideally, accepted studies identified the test species, and reported acute toxic inhalation effect(s) and exposure regimens, including concentration(s) in air and duration(s) of exposure. Selected input data obtained in this manner were divided into primary data categories, including the following:

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--**studies involving human subjects:** exposure range producing death or other clinically significant adverse effects in humans,

--**accident reports:** reports of accidental releases of anhydrous ammonia to the atmosphere,

--**acute studies involving animal bioassays:** range of exposure intensities of duration within one day producing death or other clinically significant adverse effects in animals, and

--**longer-term studies involving animal bioassays:** range of exposure intensities of duration exceeding one day producing death or other clinically significant adverse effects in animals.

Within each exposure range, reported exposure was ranked in ascending order of concentration in air and exposure duration, where known. Principle toxic effects associated with each exposure regimen were identified along with source documents. Primary and secondary source documents were numbered separately, and corresponding citations of all sources were also provided.

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FINDINGS

Standards and Guidelines

Standards and guidelines for airborne ammonia have been promulgated in many nations, including the U. S (24, 61; Table 2). Most are long-term time-weighted average (TWA) exposure limits. In the U. S., such TWAs are termed *Threshold Limit Values* (TLVs), and apply to time frames corresponding to an eight-hour work day or 40-hour work week, and these time frames may be similar if not identical to the TWAs of other nations.

Likewise several nations have promulgated *Short-Term Exposure Limits* (STELs) for airborne ammonia. In the U. S., STELs limit exposure over averaging periods of up to 15 minutes. Although long-term workplace exposure in the U. S. is limited to (in most cases) 50 ppm by an OSHA[1] TWA-TLV, OSHA has not promulgated a STEL.

The ACGIH,[2] which recommends a TWA-TLV of 25 ppm (half the OSHA value), recommends a STEL of 35 ppm for airborne ammonia (Table 2). However, the basis for the recommended STEL is not systemic toxicity, but the need to protect employees against routinely experiencing uncomfortable eye and respiratory irritation. NIOSH[3] has set forth a 300-ppm concentration of airborne ammonia deemed *Immediately Dangerous to Life or Health* (IDLH; Table 2). The

[1] OSHA: Occupational Safety and Health Administration, U. S. Department of Labor

[2] ACGIH: American Conference of Governmental Industrial Hygienists

[3] NIOSH: National Institute for Occupational Safety and Health, U. S. Department of Health and Human Services

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Table 2. Ammonia Occupational Standards & Guidelines*

country	institution	standard or guideline	time frame	value (ppm)
...	...	...	...	
short-term exposure limits				
Czechoslovakia	...	STEL	...	57
Finland	...	STEL	...	40
France	...	STEL	...	50
Hungary	...	STEL	...	39
India	...	STEL	...	35
Poland	...	STEL	...	29
Russia	...	STEL	...	29
Sweden	...	STEL	...	50
Switzerland	...	STEL	...	50
United Kingdom	...	STEL	...	35
United States	ACGIH	STEL	≤ 15 minutes	35
United States	NIOSH	IDLH	≤30 minutes	300
time-weighted average exposure limits				
Australia	...	TWA	...	25
Austria	...	TWA	...	50
Belgium	...	TWA	...	25
Czechoslovakia	...	TWA	...	29
Denmark	...	TWA	...	25
Finland	...	TWA	...	25
France	...	TWA	...	25
Germany	...	TWA	...	50
Hungary	...	TWA	...	26
India	...	TWA	...	25
Japan	...	TWA	...	25
Netherlands	...	TWA	...	25
New Zealand	...	TWA	...	check ACGIH
OEL-Arab Republic of Egypt	...	TWA	...	25
Philippines	...	TWA	...	50
Poland	...	TWA	...	29
Russia	...	TWA	...	25
Singapore	...	TWA	...	check ACGIH
Sweden	...	TWA	...	25
Switzerland	...	TWA	...	25
Thailand	...	TWA	...	50
Turkey	...	TWA	...	25
United Kingdom	...	TWA	...	25
United States	ACGIH	TWA-TLV	8 h/d, 40 h/wk	25
United States	MSHA	TWA	8 h/d, 40 h/wk	25
United States	OSHA	Construction Industry PEL	8 h/d, 40 h/wk	50
United States	OSHA	Fed Cont PEL	8 h/d, 40 h/wk	50
United States	OSHA	General Industry PEL	8 h/d, 40 h/wk	50
United States	OSHA	Shipyard PEL	8 h/d, 40 h/wk	50
Vietnam	...	TWA	...	check ACGIH

*Adapted from: Registry of Toxic Effects of Chemical Substances, access date 13 January 1996. Acronyms:

ACGIH: American Conference of Governmental Industrial Hygienists
IDLH: Immediately Dangerous to Life or Health
MSHA: Mine Safety and Health Administration
NIOSH: Nat'l. Inst. for Occupational Safety & Health
OSHA: Occupational Safety and Health Admin.
PEL: Permissible Exposure Limit
STEL: Short-Term Exposure Limit
TLV: Threshold Limit Value
TWA: time-weighted average

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IDLH concentration is defined by NIOSH (and MSHA[4]) as the concentration in air below which 1. escape (within OSHA's 30-minute maximum permissible exposure time for escape) is possible with neither loss of life nor permanent health damage, and 2. escape will not be inhibited by severe eye or respiratory irritation or other adverse reactions.

Odor and Odor Threshold

The odor of ammonia is distinct, pungent, and often familiar because of the common use of ammonia in cleaning. Several reports of the odor threshold for airborne ammonia have been published. These include values of 25 ppm (2, 4), 48 ppm (4, 32), 46.8 ppm (16, 24), and a range from 0.0266 to 39.6 mg/M^3 (0.04 to 57 ppm; 24, 62). Thus, the presence of airborne ammonia may be indicated to exposed individuals at concentrations in air roughly in the range of occupational exposure limits, or below the limits, described above (Table 2).

Vapor Density

The molecular weight of ammonia (NH_3) is 17.03 daltons (g/mole; 4, 41, 76, 82). Its vapor density is reported to be 0.6 relative to air, which is assigned a reference vapor density of unity (one; 24, 82). The density of liquid ammonia at one atmosphere pressure (and minus 33.35° C) is 0.6818 (41). The density of mixtures of ammonia

[4] MSHA: Mine Safety and Health Administration, U. S. Department of Labor

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with water (ammonia water) depends upon the ammonia fraction. The density is 0.957 for a 10-percent ammonia solution, and 0.90 for a Spirit of Hartshorn solution, which is 28-29 percent ammonia.

Acute Toxic Inhalation Effects

Reversible or Clinically Insignificant Effects

Several reports of reversible or clinically insignificant effects exerted by ammonia on humans were located (Table 3). These include eye irritation (4, 10, 24, 68, 81) upper airway irritation (4, 10, 24, 68, 81), lacrimation (4, 10, 24, 68, 81), altered breathing patterns (4, 68), minor biochemical changes (24, 66) and minor blood pressure and pulse rate changes (4, 68). Similar effects were also reported in animal studies. The lowest exposure concentration eliciting effects was reported to be 50 ppm over a duration of 10 minutes, eliciting faint to moderate irritation of the human upper airway (10, 24).

Irreversible Injury

Animal Studies. Several reports of potentially irreversible non-lethal clinical effects involving bioassay animals were located (Table 4; also see detailed tables in *Appendix A)*. Bioassay animals exhibiting such effects included mice (4, 16, 17, 28, 61), rats (4, 24, 83), rabbits (4, 7, 10, 24, 58, 61), and cats (7, 10, 24, 61).

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Table 3. Inhalation Effects Exerted By Ammonia In Studies Involving Humans

conc. in air	exposure duration	dose times duration	study type	reported effect(s)	primary data source(s)*	secondary data source(s)*
(ppm)	(min.)	(ppm.min.)	...	...	...	...
acute exposures (within 24 hours)						
32	5	158	NOAEL	OCULAR: eye irritation with lacrimation	4	8
46.8	...	...	...	SENSORY: odor threshold	6	8
50	5	251	NOAEL	OCULAR: eye irritation with lacrimation	4	8
72	5	359	NOAEL	OCULAR: eye irritation with lacrimation	4	8
50	10	502	TC-LO	RESPIRATORY: faint to moderate (5/6) irritation	4	8
135	5	675	TC-LO	OCULAR: eye irritation with lacrimation	4	8
135	5	675	TC-LO	RESPIRATORY: nose and throat irritation	4	8
135	5	675	TC-LO	RESPIRATORY: chest irritation (1/6)	4	8
50	120	6,000	LOAEL	RESPIRATORY: urge to cough; nose and throat irritation	13	3
50	120	6,000	LOAEL	OCULAR: eye irritation	13	3
500	30	15,000	LOAEL	RESPIRATORY: nasal and throat irritation; increased minute volume, cyclic pattern of hyperpnea	11	3
500	30	15,000	NOAEL	CARDIOLOVASCULAR: increased blood pressure and pulse rate	11	3
500	30	15,000	NOAEL	OCULAR: variable lacrimation	11	3
700	...	...	...	OCULAR: Eye irritation, permanent injury in absence of prompt remedial measures	12	8
5,000	...	...	...	LETHALITY: immediate death from spasm, inflammation, or edema of the larynx	12	8
5,000	5	25,000	LC-LO	LETHALITY: specific cause of death not reported	2	9
5,000	30	150,000	case report	LETHALITY: rapidly fatal	7	3
810	240	194,400	case report	BIOCHEMISTRY: some biochemical effects	10	8
7,000	180	1,260,000	LC-LO	LETHALITY: Environment Canada benchmark (draft)	5	8

continued on following page

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Table 3 (continued from previous page)

5,000	...	...	lethality	LETHALITY: "fatal almost immediately"	16	14
5000 - 10,000	...	...	lethality	LETHALITY: "rapidly fatal"	...	14
30,000	5	150,000	lethality	LETHALITY: BUT reported value is erroneous (see text)	18	17
330,000 (?)	...	...	lethality	LETHALITY: heart failure following lung damage	17	...

apparently longer-term study

20	?	...	TC-LO	OLFACTORY: ulcerated nasal septum	1	16
20	?	...	TC-LO	OCULAR: conjunctiva irritation	1	16
20	?	...	TC-LO	RESPIRATORY: change in trachea or bronchi	1	16

*Data sources: 'primary' denotes source of original research or stated conclusion; 'secondary' denotes additional source of information about primary source(s).

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Table 4. Derivation of 5- and 30-Minute Human and Human Equivalent Lethality Parameter Values for Ammonia from Human and Animal Studies

statistic	unit	note	lethality parameter	5-min.	30-min.
human lethality, based upon human studies					
minimum	(ppm)	[1]	LC-LO	5,000	833
geometric mean	(ppm)	[2]	LC-LO	39,482	6,580
arithmetic mean	(ppm)	[3]	LC-LO	95,000	15,833
standard deviation	(ppm)	[4]	LC-LO	98,234	16,372
coefficient of variation	(...)	[5]	LC-LO	103	103
human lethality, based upon animal studies					
minimum	(ppm)	[1]	LC-LO	203,762	33,960
geometric mean	(ppm)	[2]	LC-LO	299,013	49,836
arithmetic mean	(ppm)	[3]	LC-LO	318,830	53,138
standard deviation	(ppm)	[4]	LC-LO	141,487	23,581
coefficient of variation	(...)	[5]	LC-LO	44	44
minimum	(ppm)	[1]	LC-LO or LC-50	98,410	16,402
geometric mean	(ppm)	[2]	LC-LO or LC-50	160,525	28,663
arithmetic mean	(ppm)	[3]	LC-LO or LC-50	308,556	51,426
standard deviation	(ppm)	[4]	LC-LO or LC-50	144,290	24,048
coefficient of variation	(...)	[5]	LC-LO or LC-50	47	47

1. minimum: lowest among n values.
2. geometric mean = nth root of the product of n values = sum of logarithms of n values, divided by n.
3. arithmetic mean: sum of n values, divided by n.
4. standard deviation (SD) = square root of the variance, where the variance = sum of n squared deviations from the arithmetic mean, divided by (n-1).
5. coefficient of variation = 100 x SD/arithmetic mean.

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relevant organismal data (source: adapted from citation 12)*							parameter	reported parameter value	lethal exposure duration	data source *	5-min. human equivalent value	30-min. human equivalent value
species	body weight	lung volume	alveolar surface area	ventiliation rate	ratio of min. vol. to alveolar surface area	ratio of species/ human						
column -->	*b*	*c*	*d*	*e*	*f*	*g*	*h*	*i*	*j*	*k*	*l*	*m*
formula -->	...	...	...	...	*e/d*	*i-animal/ i-human*	...	...	...	...	*g i j /5*	*g i j /30*
...	(kg)	(mL)	(sq M)	(mL/min)	(mL/min/sq M)	...	...	(ppm)	(min)	...	(ppm)	(ppm)
human studies												
human	75	7,000	82	6,000	73	1	lethality	5,000	"rapid"	6	...	...
human	75	7,000	82	6,000	73	1	lethality	2,500	unstated	9	...	...
human	75	7,000	82	6,000	73	1	lethality	5,000	unstated	9	...	...
human	75	7,000	82	6,000	73	1	lethality	5,000	180	9	180,000	30,000
human	75	7,000	82	6,000	73	1	lethality	15,000	5	9	15,000	2,500
human	75	7,000	82	6,000	73	1	lethality	5,000	unstated	11	...	...
human	75	7,000	82	6,000	73	1	lethality	5,000	180	14	180,000	30,000
human	75	7,000	82	6,000	73	1	lethality	5,000	unstated	16	...	...
human	75	7,000	82	6,000	73	1	lethality	5,000	5	17	5,000	833
human	75	7,000	82	6,000	73	1	lethality	5,000	unstated	26	...	...
human	75	7,000	82	6,000	73	1	lethality	5,000	...	16	...	...
human	75	7,000	82	6,000	73	1	lethality	30,000	5	21	source in error	
human	75	7,000	82	6,000	73	1	lethality	330,000	...	19	...	...
animal studies												
cat	...	...	...	...	...	...	LC50	10,050	60	5	...	...
cat	...	...	...	...	...	...	LC50	1,071	60	9	...	...
cat	...	...	...	...	...	...	LC50	10,122	60	9	...	...
cat	...	...	...	...	...	...	LCLO	7,035	60	9	...	...

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guinea pig	...	...	...	...	...	...	LCLO	5,025	5	9	...	...
guinea pig	...	...	...	...	...	...	LCLO	22,612	9	9	...	...
mouse	0.023	0.74	0.068	24	353	4.82	LC50	21,430	30	12	620,209	103,368
mouse	0.023	0.74	0.068	24	353	4.82	LC50	14,260	30	12	412,701	68,784
mouse	0.023	0.74	0.068	24	353	4.82	LC50	11,299	60	20	654,013	109,002
mouse	0.023	0.74	0.068	24	353	4.82	LC50	10,201	10	9, 22, 24	98,410	16,402
mouse	0.023	0.74	0.068	24	353	4.82	LC50	6,905	30	18	199,839	33,306
mouse	0.023	0.74	0.068	24	353	4.82	LC50	6,525	30	23	188,841	31,474
mouse	0.023	0.74	0.068	24	353	4.82	LC50	6,044	30	15	174,920	29,153
mouse	0.023	0.74	0.068	24	353	4.82	LC50	5,877	30	22, 24	170,087	28,348
mouse	0.023	0.74	0.068	24	353	4.82	LC50	4,837	60	20	279,977	46,663
mouse	0.023	0.74	0.068	24	353	4.82	LC50	4,824	60	9	279,224	46,537
mouse	0.023	0.74	0.068	24	353	4.82	LC50	4,752	120	9	550,114	91,686
mouse	0.023	0.74	0.068	24	353	4.82	LC50	4,230	60	10, 15, 24	244,842	40,807
rabbit	3.6	79	5.9	1,042	177	2.41	LC50	10,050	60	5	291,089	48,515
rabbit	3.6	79	5.9	1,042	177	2.41	LC50	10,122	60	9	293,174	48,862
rabbit	3.6	79	5.9	1,042	177	2.41	LCLO	7,035	60	9	203,762	33,960
rat	0.14	6.3	0.39	84	215	2.94	LC50	40,300	10	1	237,253	39,542
rat	0.14	6.3	0.39	84	215	2.94	LC50	28,595	20	1	336,688	56,115
rat	0.14	6.3	0.39	84	215	2.94	LC50	23,300	30	3, 24	411,514	68,586
rat	0.14	6.3	0.39	84	215	2.94	LC50	21,860	30	2	386,081	64,347
rat	0.14	6.3	0.39	84	215	2.94	LC50	20,300	40	1	478,039	79,673
rat	0.14	6.3	0.39	84	215	2.94	LC50	17,401	15	20	153,664	25,611
rat	0.14	6.3	0.39	84	215	2.94	LC50	11,590	60	1	409,394	68,232
rat	0.14	6.3	0.39	84	215	2.94	LC50	11,172	30	20	197,315	32,886
rat	0.14	6.3	0.39	84	215	2.94	LC50	10,911	60	9	385,410	64,235
rat	0.14	6.3	0.39	84	215	2.94	LC50	10,480	30	18	185,093	30,849
rat	0.14	6.3	0.39	84	215	2.94	LC50	7,338	60	4	259,201	43,200
rat	0.14	6.3	0.39	84	215	2.94	LC50	5,660	30	7	99,964	16,661

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rat	0.14	6.3	0.39	84	215	2.94	LC50	2,000	240	25	282,585	47,097
rat	0.14	6.3	0.39	84	215	2.94	LCLO	2,010	240	9	283,998	47,333
rat	0.14	6.3	0.39	84	215	2.94	LCLO	7,322	60	9	258,636	43,106
rat	0.14	6.3	0.39	84	215	2.94	LCLO	2,000	240	8	282,585	47,097
rat	0.14	6.3	0.39	84	215	2.94	LOAEL	1,000	960	27	565,169	94,195

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Human Studies. Examination of Table 3 reveals no reports of potentially irreversible non-lethal clinical effects in humans. Accident reports reveal two involving permanent injuries, including impaired breathing and damage to the respiratory system, throat, and eyes (38). However, the estimated ammonia concentration range is unhelpfully broad (700 - 10,000 ppm) and exposure durations are unstated.

Fatal Exposure

Animal Studies. Studies of ammonia-induced mortality in animal bioassays are set forth in Table 4 and in *Appendix A* (Tables A-1 and A-2). A one-hour LC_{50} value[5] for cats exposed to airborne ammonia was 1,071 ppm (12, 20). Numerous bioassay reports are analyzed in Table 4, giving equivalent five- and 30-minute human inhalation LC_{LO}[6] and LC_{50} values.

Human Studies. Examination of Table 3 reveals several reports of human mortality following exposure to airborne ammonia at a concentration of 5,000 ppm. Concentrations are typically estimates obtained from reconstructions of accidental exposures. One report indicates immediate death from spasm, inflammation, or edema of the larynx (24, 75). Other reports indicate exposure durations of five minutes (61, 35, 36) or 30 minutes (4, 22). A quite different lethality benchmark; 7,000 ppm over three hours of exposure; was reported in a draft Environment Canada document (15, 24). Selected reports of

[5] LC_{50}: concentration lethal to 50 percent of individuals over a specified exposure duration in a bioassay.

[6] LC_{LO}: lowest concentration lethal to any individuals over a specified duration in a bioassay.

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human lethality were examined to distinguish between primary research or clinical reports vs. secondary reviews. Reports examined in detail are evaluated in the paragraphs that follow. Evaluations reflect the principle that, in the presence of multiple information sources, the best practice is to rely upon best evidence. Evolution of reported lethality benchmark values is traced over time in Fig. 1 (in which solid lines indicate sources which are explicitly cited, and dashed lines indicate sources which are apparent, but uncited).

ENVIRONMENT CANADA. *Technical Information for Problem Spills: Ammonia.* Draft, 1981

SUMMARY. The 1981 draft is out of print, superseded by a July-1984 final report, which was obtained. However, a 5,000-ppm lethality concentration is cited, and presumably represents the same source which was cited in the 1981 draft. The source is the 1978 edition of the Kirk-Othmer *Encyclopedia of Chemical Technology,* specifically a table referred to by Environment Canada as a *"Summary of Human Exposure."* In the table; 5,000 ppm (7,179 mg/M^3) is associated with *"serious edema, strangulation, asphyxia; fatal almost immediately."*

ADDITIONAL DATA. In addition to the above, Environment Canada provides Table 7.4.1 *"Inhalation"* (page 103), in which literature on effects and effect concentrations is cited. The Kirk-Othmer citation and effect concentration are not used. However, ammonia is associated with (apparently lethal) *"respiratory spasm and rapid asphyxia"* in a range of 3,500 to 7,000 mg/M^3 (approximately 2,500 to 5,000 ppm) in Braker 1977. The same range is cited in a 1979 National Research Council report (47), which also indicates a 0.5- to one-hour exposure duration. The table cites an RTECS 1979 report of a 5-minute LC_{LO} of

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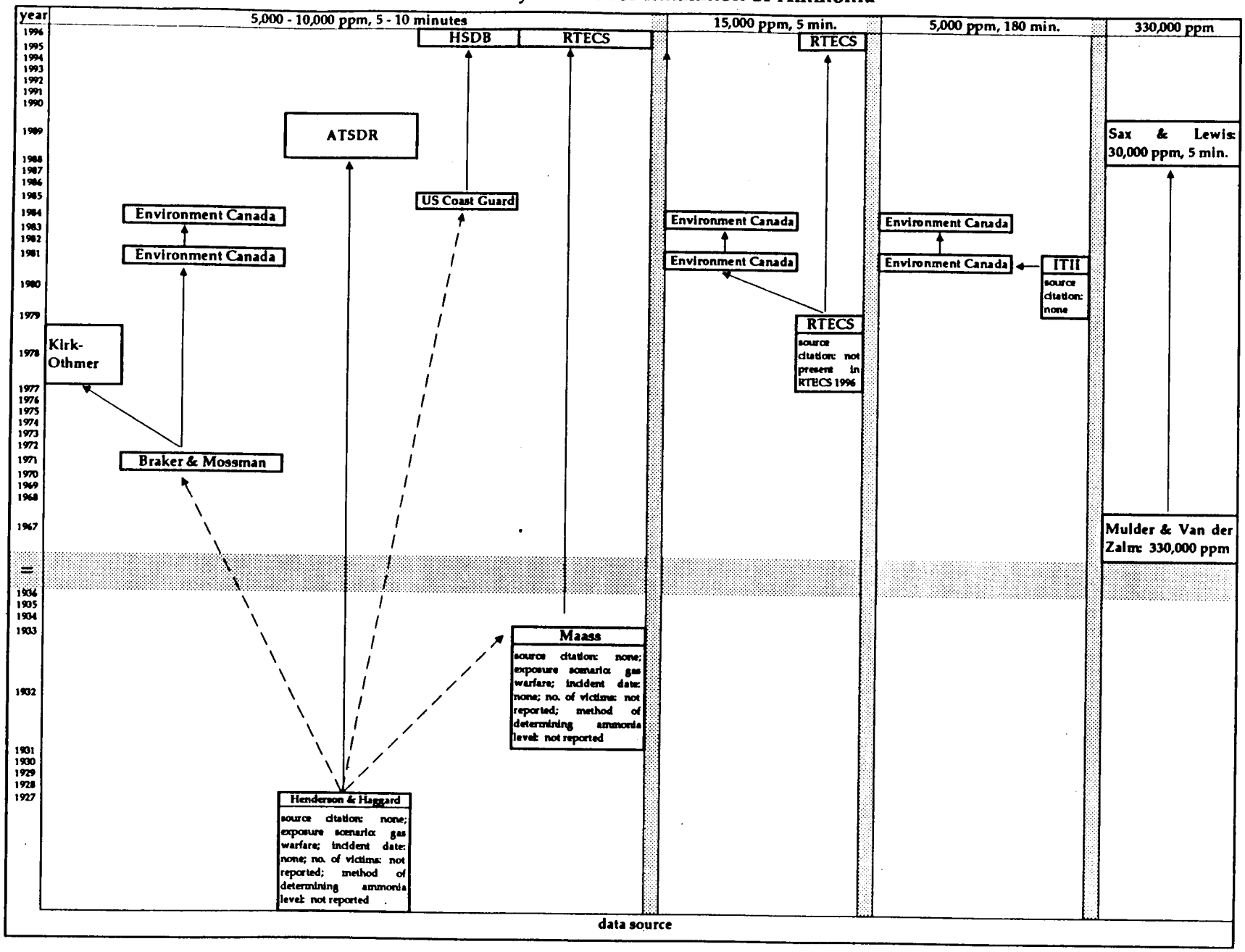
Fig. 1. Evolution of Four Different Human Lethality Values for Inhalation of Ammonia



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21,000 mg/M^3 (approximately 15,000 ppm). Table 7.4.1 also cites a 3-h LC_{LO} of 7,000 µg/M^3 (approx. 5,000 ppm) reported in ITII 1981.

EVALUATION. Secondary source; source quality: low. Examine Kirk-Othmer 1978; Braker 1977; ITII 1981; RTECS (1979 and/or current).

KIRK-OTHMER. *Encyclopedia of Chemical Technology*, third edition. New York City, Wiley Interscience, volume 2, pages 509-16, 1978

SUMMARY. Table 13 (page 509) indicates that 5,000 ppm of ammonia is associated with the notation: *"serious edema, strangulation, asphyxia; fatal almost immediately."* The cited source of this information is the *Matheson Gas Data Book*, 1971.

EVALUATION. Secondary source; source quality: low. Examine the *Matheson Gas Data Book*, 1971 or other available edition(s).

BRAKER, W., AND A. MOSSMAN. *Matheson Gas Data Book*, fifth edition. Lyndhurst, New Jersey; Matheson Gas Products, 1971

SUMMARY. The sixth edition was obtained. Table 1, untitled, sets forth vapor concentrations, general effects, and exposure periods. Ammonia at 5,000 - 10,000 ppm is associated with *"respiratory spasm, rapid asphyxia,"* and the exposure period indicates *"rapidly fatal."* No primary source citations are provided.

EVALUATION. Secondary source; source quality: low. The exposure period is unquantified. No primary source citation is provided. Reject in favor of more completely documented reports.

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ITIL *Toxic and Hazardous Industrial Chemicals, Safety Manual for Handling and Disposal with Toxicity and Hazard Data.* International Technical Information Institute; Tokyo, Japan; pp. 28-30, 1981

SUMMARY. A lethal inhalation concentration of 10,000 ppm at exposure duration three hours is given. No primary source citation is provided.

EVALUATION. Secondary source; source quality: low. Reject in favor of more completely documented reports.

HENDERSON, Y., AND H. W. HAGGARD. *Noxious gases and the principles of respiration influencing their action.* American Chemical Society Monograph Series. New York City, Chemical Catalogue Company, Inc.; 87:113-26, 1927

SUMMARY. This monograph is a secondary source (a review), and does not constitute a primary report of clinical or experimental findings. It includes a table (on page 126) titled *"Physiological Responses To Various Concentrations of Ammonia."* The table includes an entry indicating that ammonia is *"rapidly fatal for short exposure"* at concentrations of 5,000 - 10,000 ppm. No primary source citation is provided for this entry, in contrast to the other entries in the table. The possibility that the previous-line source citation might apply to subsequent lines of information was also considered. However, an examination of lines 1, 2, and 3 reveals the same source citation (to a 1921 Bureau of Mines technical report) on each line, suggesting that source citations, where available, are provided at each line individually and, therefore, no source citation applies to the tabulated 5,000- to 10,000-ppm value. The table also indicates that the maximum

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concentration allowable for short exposure (1/2 to one hour) is 300 - 500 ppm [citing Ronzani, E.; *Archiv für Hygiene,* 70:217, 1909 (60) and Lehmann, K.; *ibid.*, 5:1, 1886 (31)]; and that 2,500 to 4,500 ppm is dangerous for even short exposure (1/2 hour; citing Ronzani).

EVALUATION. Secondary source; source quality: low. Reject in favor of more completely documented reports. The phrase *"short exposure"* associated with lethality can refer to 1/2 hour or 1/2 - 1 hour (as specified with respect to the maximum concentration allowable for short exposure; see above paragraph), or to neither. It seems to have been interpreted subsequently as meaning five to 10 minutes in derivative sources (Fig. 1). Likewise, the range 5,000 to 10,000 ppm set forth in Henderson and Haggard can apply to one and to 1/2 hour, respectively, but is unlinked to the cited documentary support provided for values applicable to those time frames. The Henderson and Haggard report is unusually old, and apparently relies either upon no clinical data (in the case of lethality) or upon sources which are even older (1896 and 1909) and presumably more unreliable (in the case of dangerous or maximum allowable concentrations). It appears to fall short of current standards for technical documentation.

MAASS, T. A. *Biologie und Toxikologie der chemischen Kampfmittel. (Gase, Dämpfe und Zerstäubungen) [Biology and toxicology of chemical weapons (gases, vapors, aerosols)].* Tabulae Biologicae Periodicae (Berlin), 3(2):231-40, 1933 (German)

SUMMARY. The German words for ammonia are Ammoniak (gas) and Salmiakgeist (solution). This article primarily introduces principles of biology and toxicology of chemical agents used in warfare. However, ammonia appears to be unmentioned in the article, citation of which (in RTECS) was apparently erroneous.

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EVALUATION. Secondary source; source quality: low. This is the last article in volume 3 number 2. As a speculation, examine the first article in the subsequent issue (volume 3 number 3, pages 241-96) of the same journal, by same author (also in German).

MAASS, T. A. *Die im Gaskampf verwendeten Stoffe und ihre Eigenschaften. (Substances used in gas warfare and their properties.)* Tabulae Biologicae Periodicae (Berlin), 3(3):241-96, 1933 (German)

SUMMARY. An unnumbered table on page 257 titled (in translation) *"Approximate Effectiveness of Gases and Vapors for Humans"* lists 'Ammoniak.' Ammonia is listed as lethal *("tödlich")* at 5,000 ppm following exposure of duration five to 10 minutes. No primary source citation is provided for this entry. General literature is provided in an appendix *("Anhang")* on pages 249-50. However, such an appendix presumably pertains to previously presented information, not to subsequent tables (in which case it would not be 'appended'), some of which also have appended material. Finally, the literature cited appears to be of a general nature, including no primary research reports likely to be the original source of any toxicological data.

EVALUATION. Secondary source; source quality: low. The lethal value; 5,000 ppm over 10 minutes; lacks primary documentary support, so cannot be accorded definitive status. Indeed, the source table title emphasizes that the tabulated values are (in translation) *"approximate,"* suggesting that a definitive measurement may have been unavailable. In the presence of multiple information sources, the best practice is to rely upon best evidence. If higher-quality evidence suggests a different lethality concentration or range, then that other evidence should be preferred, unconstrained by this old report, which fails to meet current standards of technical documentation.

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U. S. COAST GUARD. *CHRIS - Hazardous Chemical Data,* Volume 2. Coast Guard Department of Transportation, 1984-1985

SUMMARY. The January 1991 edition was obtained. It contains a Material Safety Data Sheet (MSDS) for anhydrous ammonia. The MSDS does not cite a lethal vapor concentration.

EVALUATION. Secondary source; source quality: low. Reject in favor of more completely documented source(s). If the 1984-85 edition included a lethal vapor concentration, that parameter or its value in the case of anhydrous ammonia was abandoned in the later edition. Reject in favor of more completely documented reports.

SAX AND LEWIS. *Dangerous Properties of Industrial Materials, 7th Edition.* New York City, Van Nostrand Reinhold, Volume 2, page 231, 1989

SUMMARY. Human 5-minute inhalation LC_{LO} = 30,000 ppm.

EVALUATION. Secondary source; source quality: low due to erroneous information. Reject in favor of more accurate source(s). Examine cited reference: Mulder and Van der Zalm 1967 (43).

MULDER, J. S., AND H. O. VAN DER ZALM. *Een gaval van dodelijke ammoniakvergiftiging (Fatal case of ammonia poisoning).* Tijdschrift Voor Sociale Geneeskunde (Rotterdam, The Netherlands), 45:458-60, 1967

SUMMARY. A person engaged in filling a tank wagon with 25-percent ammonia solution died as a result of failure to wear respiratory protection during measurement of the ammonia concentration in the tank. The report fails to quantify the exposure duration, and makes

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ambiguous statements (translated from Dutch) about whether the deceased also failed to wear respiratory protection during the filling portion of the operation. It is also imprecise about ammonia concentrations in the breathing-zone air of the deceased:

> *"Layer of lung below the superficial layer had been harmed... He had inhaled great amount of concentrated ammonia... At the moment of the mishap, the ammonia water solution spread as a thin film over the tank car. The wind direction was toward the victim. The vapor pressure of a 25-percent (by weight) ammonia solution is 224 mm of mercury. As a consequence of that, the vapor contained 33-percent ammonia (by volume), or 330,000 ppm. Of course, under these circumstances, there was not a saturated vapor pressure at the level of the victim. The actual concentration of ammonia is unknown, but is roughly estimated to be multiple times 10,000 ppm."*

The deceased had been instructed to report to the first aid room for medical attention, but instead went to the coffee room for milk. He then continued to work for the remainder of the day. First aid was sought only three hours after the exposure incident, whereupon the patient was transferred to a clinic for X-raying. His heart stopped, but was restarted; he was transferred to a hospital, where his heart stopped finally. The immediate cause of death, which occurred six hours post-exposure, was heart failure, though the pathology report indicated that the underlying cause was the effect of ammonia on the deep layer of the lung, presumably creating cardiopulmonary stress which proved intolerable over the protracted period (of perhaps six hours) between exposure and initiation of medical attention.

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EVALUATION. Primary source; source quality: moderate. Sax and Lewis (1989; 64) incorrectly lists a 5-minute LC_{LO} of 30,000 ppm from this source, possibly a result of mistranslating 33-percent ammonia vapor by volume as 30,000 ppm instead of 330,000 ppm as reported. This error persists in the latest edition, which is Lewis (1996; 33).

The Mulder and Van der Zalm article presents an ammonia toxicity table, including 5,000 - 10,000 ppm listed as *"deathly after a short time,"* similar to information provided in Henderson & Haggard. However, they refute the Henderson & Haggard lethality concentration by characterizing the actual lethality concentration as *"multiple times 10,000 ppm."* Unfortunately, the multiplier is not quantified.

The 25-percent ammonia with which the tank was being filled is presumed to have also contained 25-percent ammonia previously. Also presumably, the tank being filled was needed as a source of 25-percent ammonia solution and, to prevent interruption during such use, would have been refilled before being completely emptied of its prior contents. Thus, a partially filled mobile tank (tankwagon) on a railway was presumably being refilled after being transported to a larger, possibly stationary, source. Both vessels would have been subject to similar ambient conditions, such as an external temperature of 10° C, over an unquantified period prior to the liquid transfer.

Although Mulder and Van der Zalm expressed the belief that the air at the level of the victim had not reached saturation (330,000 ppm), presumably the atmosphere within the tank being filled was saturated because of pre-existing residues of 25-percent ammonia solution, turbulent flow of ammonia solution from the source tank, or both. Prior to complete filling and subsequent overflow of the tank being filled, the ammonia-saturated vapor inside the tank being filled

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must have been displaced into the ambient air in close proximity to the deceased while he was situated on top of the tank during the filling portion of the operation. In addition, ammonia vapor, lighter than air at 10° C, would have been rising from the rich pool of ammonia from the overflowed ammonia solution on the ground. Thus, the deceased appears to have been exposed, at least sporadically, to two sources of ammonia vapor at all or a reasonably high fraction of the 330,000-ppm saturated vapor concentration during the filling and/or measuring operation.

Given the time between exposure and death, the Mulder and Van der Zalm report suggests that airborne ammonia concentrations equal to a reasonably large fraction of 330,000 ppm are not rapidly fatal, contrary to multiple sources cited and described above, and in each case found to be questionable. Given the failure of the deceased to seek immediate care, and his ability to continue working, this report suggests that both the duration and the degree of the cardiovascular stress which presumably caused his heart to fail may have been significantly and unnecessarily exacerbated. At least the duration, and perhaps also the intensity, of cardiovascular stress could have been reduced by initiation of routine medical interventions, such as provision of an oxygen-enriched breathing atmosphere, in a timely manner (before three hours, when medical attention was initiated). Thus, the inevitability of death, which occurred six hours after exposure and three hours after initiation of medical attention, must be regarded as being doubtful. Together, the facts educed from this incident and inferences made above suggest that a concentration airborne ammonia to which brief exposure may be non-lethal is significantly in excess of 10,000 ppm, and possibly as high as 330,000 ppm.

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Accident Reports. Numerous reports address accidents in which anhydrous ammonia was released with lethal effect on humans (see, for example; 6, 21, 30, 34, 38, 39, 42, 43, 48-52, 54, 84; also see news clippings of accident reports, *Appendix B)*. One of the cited reports was evaluated above (43). However, reliable measurements of concentrations of exposure to ammonia have been difficult to obtain, and the absence of such data has motivated attempts to reconstruct ambient concentrations during accidents retrospectively, for example, based upon air dispersion modeling (38, 39, 42, 54) or evidence from ammonia-induced damage to biota (flora and fauna; 38).

Several accidents in which ammonia concentrations were inferred by such means are evaluated in Table 5 to discern effect concentrations implied by reported ranges of exposure. Examination of the table reveals an absence of reliable exposure duration data. Nonetheless, the geometric mean of reported lethal exposure ranges is approximately 30,000 ppm. A reasonably conservative exposure duration assumption would appear to be five minutes based upon the amount of time required for dissipation of ammonia, as inferred from a 1974 ammonia release accident in Potchefstroom, South Africa (Table 5 indicates the same accident date, but in 1973). This accident is evaluated below in Table 6 and supporting text.

On 13 July 1974 an ammonia tank failed, releasing anhydrous ammonia. Exposure concentrations set forth in Table 5 were based upon a relatively old air dispersion model, probably WHAZAN, whereas the higher concentrations set forth in Table 6 are more reliably based upon the state-of-the-art HGSYSTEM model (42). As the table indicates, the exposure duration in zone 1 was estimated at 448 seconds

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Table 5. Synthesis of Accident Reports To Discern Ammonia Effect Levels*

nation	state	city	date	estimated concentration		geometric	release	exposure	number	number	effect
				from	to	mean	duration	duration	exposed	affected	
...	...	...	...	(ppm)	(ppm)	(ppm)	(min)	(min)	...	...	...
human fatality											
S. Afr.	...	Potchefstroom	7/13/73	2,500	200,000	22,361	instant	...	...	2	fatal
U. S.	LA	Boutte	12/15/70	5,000	200,000	31,623	3	...	...	3	fatal
U. S.	NE	Crete	2/18/69	5,000	200,000	31,623	instant	...	...	5	fatal
S. Afr.	...	Potchefstroom	7/13/73	5,000	200,000	31,623	instant	...	...	10	fatal
U. S.	TX	Houston	5/11/76	5,000	200,000	31,623	instant	...	...	2	fatal
U. S.	FL	Pensacola	11/9/77	20,000	200,000	63,246	240	...	...	2	fatal
permanent injury											
U. S.	MN	Barnesville	6/10/81	700	10,000	2,646	30	...	...	11	injury: claimed permanent, to respiratory, eyes, throat
U. S.	TX	Houston	5/11/76	700	10,000	2,646	instant	...	...	3	injury: permanent breathing impairment
reversible injury											
U. S.	LA	Boutte	12/15/70	300	5,000	1,225	3	...	...	3	injury: treated, released <24 hours
U. S.	KS	Conway	12/6/73	300	5,000	1,225	<480	...	...	1	injury: hospitalized one day, injuries unspecified
U. S.	KS	Conway	12/6/73	300	5,000	1,225	<480	...	...	1	injury: hospitalized six days: eye, nose, throat, lung burns

U. S.	LA	Boutte	12/15/70	700	10,000	2,646	3	...	...	26	injury: hospitalized <14 days; unspecified injuries
U. S.	FL	Pensacola	11/9/77	20,000	200,000	63,246	240	...	...	2	injury: children hospitalized; recovery in one month
transient or clinically insignificant effects											
U. S.	TX	Houston	5/11/76	300	500	387	instant	10	...	1	NOAEL: no apparent permanent injury
U. S.	MN	Barnesville	6/10/81	300	5,000	1,225	30	...	17+	17+	NOAEL: exposure without injury
damage to flora, fauna											
U. S.	OK	Verdigris	6/10/79	12	72	29	instant	...	...	...	flora: vegetative discoloration
U. S.	TX	Houston	5/11/76	12	72	29	instant	...	...	...	flora: vegetative discoloration
U. S.	OK	Enid	5/7/76	12	72	29	240	...	...	...	flora: wheat bleached
U. S.	FL	Crestview	4/8/79	1,000	3,300	1,817	...	...	...	...	flora: defoliation; other subst. present, incl. chlorine
U. S.	TX	Houston	5/11/76	2,000	2,300	2,145	instant	...	...	...	fauna: birds fell from trees
U. S.	NE	Blair	11/16/70	700	10,000	2,646	150	...	...	...	fauna: fatal to one of the two species (hogs or cattle)
U. S.	FL	Pensacola	11/9/77	2,300	?	...	240	...	...	...	fauna: death of small birds, wildlife
U. S.	FL	Pensacola	11/9/77	2,300	?	...	240	...	...	...	flora: withered trees and ground vegetation
U. S.	WV	Belle	1/21/70	5,000	20,000	10,000	...	...	...	9	fauna: death of nine horses

*Adapted from: Markham, Richard S. Review of damages from ammonia spills. American Institute of Chemical Engineers; Boston, Massachusetts; Ammonia Plant Safety (and Related Facilities), pp. 137-49; August, 1986. Note that some accidents are accorded multiple listings. This is done to accommodate multiple exposure scenarios, such as might prevail at different locations in the vicinity of the accident.

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Table 6. Evaluation of Potchefstroom, South Africa Ammonia Incident To Estimate Human Lethality Concentrations*

time from release	modeled ammonia concentration range			estimated exposure duration	cumulative exposure	conc. x duration	equivalent mean conc. averaged over:	
	from	to	mean				1 min.	5 min.
column --> a	*b*	*c*	*d*	*e*	*f*	*g*	*h*	*i*
formula -->	...	...	*(b+c)/2*	*a5 = (a15 - a0)/2*	*Σe*	*d x e*	*g/60*	*g/300*
(s)	(ppm)			(s)	(s)	(ppm.s)	(ppm)	(ppm)
zone 1: ≤50 meters from release point								
0	...	...	...	...	0.0	...	...	...
5	641,000	641,000	641,000	7.5	7.5	4,807,500	80,125	16,025
15	365,000	365,000	365,000	12.5	20.0	4,562,500	76,042	15,208
30	220,000	221,000	220,500	17.5	37.5	3,858,750	64,313	12,863
50	136,000	138,000	137,000	15	52.5	2,055,000	34,250	6,850
60	109,000	113,000	111,000	15	67.5	1,665,000	27,750	5,550
80	79,600	85,000	82,300	20	87.5	1,646,000	27,433	5,487
100	62,200	68,300	65,250	20	107.5	1,305,000	21,750	4,350
120	49,800	57,300	53,550	40	147.5	2,142,000	35,700	7,140
180	28,100	37,600	32,850	60	207.5	1,971,000	32,850	6,570
240	15,800	24,100	19,950	60	267.5	1,197,000	19,950	3,990
300	7,600	13,700	10,650	60	327.5	639,000	10,650	2,130
360	1,300	7,900	4,600	60	387.5	276,000	4,600	920
420	...	2,000	2,000	60	447.5	120,000	2,000	400
480	...	...	**251,093**	**67.5-second (1.125-minute) time-weighted average**				...
540	...	...	**58,647**	**447.5-second (7.458-minute) time-weighted average**				...
600	...	...						...
660	...	...	...	...	...	...	...	...
720	...	...	...	...	...	...	...	...
zone 2: >50 to ≤100 meters from release point								
0	...	...	...	...	0.0	...	...	...
5	...	...	...	...	0.0	...	...	...
15	365,000	365,000	365,000	12.5	12.5	4,562,500	76,042	15,208
30	220,000	221,000	220,500	17.5	30.0	3,858,750	64,313	12,863
50	136,000	138,000	137,000	15	45.0	2,055,000	34,250	6,850
60	109,000	113,000	111,000	15	60.0	1,665,000	27,750	5,550
80	79,600	85,000	82,300	20	80.0	1,646,000	27,433	5,487
100	62,200	68,300	65,250	20	100.0	1,305,000	21,750	4,350
120	49,800	57,300	53,550	40	140.0	2,142,000	35,700	7,140
180	28,100	37,600	32,850	60	200.0	1,971,000	32,850	6,570
240	15,800	24,100	19,950	60	260.0	1,197,000	19,950	3,990
300	8,900	13,700	11,300	60	320.0	678,000	11,300	2,260
360	2,900	8,300	5,600	60	380.0	336,000	5,600	1,120
420	...	5,500	5,500	60	440.0	330,000	5,500	1,100
480	...	3,200	3,200	60	500.0	192,000	3,200	640
540	...	1,100	1,100	60	560.0	66,000	1,100	220
600	...	...	**202,354**	**60-second (1.00-minute) time-weighted average**				...
660	...	...	**39,293**	**560-second (9.333-minute) time-weighted average**				...
720	...	...						...
zone 3: >100 to ≤200 meters from release point								
0	...	...	...	...	0.0	...	...	...
5	...	...	...	...	0.0	...	...	...
15	...	...	...	...	0.0	...	...	...
30	...	...	...	...	0.0	...	...	...
50	136,000	138,000	137,000	15	15.0	2,055,000	34,250	6,850
60	109,000	113,000	111,000	15	30.0	1,665,000	27,750	5,550
80	79,600	85,000	82,300	20	50.0	1,646,000	27,433	5,487

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100	62,200	68,300	65,250	20	70.0	1,305,000	21,750	4,350
120	49,800	57,300	53,550	40	110.0	2,142,000	35,700	7,140
180	28,100	37,600	32,850	60	170.0	1,971,000	32,850	6,570
240	15,800	24,100	19,950	60	230.0	1,197,000	19,950	3,990
300	9,100	13,700	11,400	60	290.0	684,000	11,400	2,280
360	5,400	8,300	6,850	60	350.0	411,000	6,850	1,370
420	1,900	5,600	3,750	60	410.0	225,000	3,750	750
480	...	4,000	4,000	60	470.0	240,000	4,000	800
540	...	2,400	2,400	60	530.0	144,000	2,400	480
600	...	...	80,118	70-second (1.167-minute) time-weighted average				...
660	...	...	25,821	530-second (8.833-minute) time-weighted average				...
720	...	...						...

zone 4: 250 meters from release point

0	...	...	...	...	0.0	...	...	...
5	...	...	...	...	0.0	...	...	...
15	...	...	...	...	0.0	...	...	...
30	...	...	...	...	0.0	...	...	...
50	...	...	...	...	0.0	...	...	...
60	...	...	...	...	0.0	...	...	...
80	79,600	85,000	82,300	20	20.0	1,646,000	27,433	5,487
100	62,200	68,300	65,250	20	40.0	1,305,000	21,750	4,350
120	49,800	57,300	53,550	40	80.0	2,142,000	35,700	7,140
180	28,100	37,600	32,850	60	140.0	1,971,000	32,850	6,570
240	15,800	24,100	19,950	60	200.0	1,197,000	19,950	3,990
300	9,100	13,700	11,400	60	260.0	684,000	11,400	2,280
360	5,800	8,300	7,050	60	320.0	423,000	7,050	1,410
420	2,800	5,600	4,200	60	380.0	252,000	4,200	840
480	...	4,050	4,050	60	440.0	243,000	4,050	810
540	...	2,800	2,800	60	500.0	168,000	2,800	560
600	...	1,500	1,500	60	560.0	90,000	1,500	300
660	...	...	63,663	80-second (1.333-minute) time-weighted average				...
720	...	...	18,073	560-second (9.333-minute) time-weighted average				...

Summary

fatality rate (%)	ammonia level peak (ppm)	1-min. (ppm)	5-min.* (ppm)
observed			
0%	0	0	0
0%	82,300	84,883	33,737
26%	365,000	236,080	73,347
60%	641,000	282,479	87,479
*Adjusted from 7 or 9 minutes.			
calculated *(interpolating or extrapolating observed values)*			
0	82,300	84,883	33,737
10%	191,341	143,036	48,972
20%	300,383	201,188	64,207
30%	398,000	241,539	75,010
40%	479,000	255,185	79,166
50%	560,000	268,832	83,322
60%	641,000	282,479	87,479
70%	722,000	296,126	91,635
80%	803,000	309,773	95,791
90%	884,000	323,420	99,947
100%	965,000	337,067	104,103

*number of individuals known to have been exposed:	
zone 1	10
zone 2	27
zone 3	6
zone 4	unknown
*number of fatalities:	
zone 1	6
zone 2	7
zone 3	5
zone 4	0
*number of survivors:	
zone 1	4
zone 2	20
zone 3	1
zone 4	all
*fatality rate:	
zone 1	0.60
zone 2	0.26
zone 3	0.83
zone 4	0.00
*survival rate:	
zone 1	0.40
zone 2	0.74
zone 3	0.17
zone 4	1.00

*Source: derived from: Mudan, K.; and K. Mitchell. "Report on the Potchefstroom, South Africa Ammonia Incident." Columbus, Ohio; Four Elements, Inc.; 15 pp., March 1996.

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(7.5 minutes). The time-weighted average exposure over that duration is 58,647 ppm, with 60-percent mortality. Values for other zones, respectively, are: zone 2: 9.3 minutes; 39,293 ppm, and 26-percent mortality; zone 3: 8.8 minutes; 25,821 ppm, and 83-percent mortality (but this value must be considered unreliable because other exposed individuals are presumed to have left the scene uncounted); and zone 4: 9.3 minutes; 18,073 ppm, and zero-percent mortality.

A spectrum of predicted ammonia concentration-effect benchmarks are calculated in Table 6 based upon 'observed' values, which are also tabulated. Most notably; peak, one-minute, and five-minute human inhalation LC_{50} and LC_0 values are tabulated. They are: LC_{50}: 560,000 ppm (peak), 268,832 ppm (one-minute), and 83,322 ppm (five-minute); LC_0: 82,300 ppm (peak), 84,883 ppm (one-minute), and 33,737 ppm (five-minute).

Personal communications. To augment the available database pertaining to ammonia lethality concentrations, a program of telephone inquiries was implemented. Target contacts included representatives of insurance companies which might have experienced losses associated with anhydrous ammonia release, chemical companies manufacturing ammonia, trade associations representing industries facing challenges of handling ammonia, and federal agencies regulating ammonia manufacture, transport, and/or use. The result of this program is that no data beyond that identified as described were made available, and no individual indicated that additional human lethality data could be found. Details of the telephone inquiry activities are summarized in Table 7.

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Table 7. Record of Contacts for Augmenting Anhydrous Ammonia Lethality Database

individual contacted	date	affiliation	telephone	lethality data	notes
insurance companies					
Roland J. Land, PE, ARM Vice President	4/4/96	Alexander & Alexander Insurance Company	212/973-4200	no data	Has no ammonia data; does not believe such info on humans is likely to be available. Cites great uncertainty about exact levels at which ammonia is lethal, but no specific data.
Henry L. Febo, Jr., PE, Engineering Specialist Special Hazards Section	4/24/96	Factory Mutual Research, Norwood, MA	617/762-4300	no data	Insures property, not life or injury; much loss data, but not with measured air levels of NH3.
William J. Satterfield, PE; Principal Engineer	4/5/96, 4/24/96	Industrial Risk Insurers	860/520-5717	...	Formerly with Hartford Steam Boiler.
John Davenport	4/24/96	Industrial Risk Insurers	860/520-7362	...	out of office
Peter Langan	4/24/96	Industrial Risk Insurers	860/520-7554	no data	Insures property, not life or injury; much loss data, but not with measured air levels of NH3.
Gary Robinson, Industrial Hygienist	4/4/96, 4/5/96	Liberty Mutual Insurance Company	815/282-2600, ext. 261	no data	Knows of no catastrophic types of claim involving ammonia. If such claims exist, coding would be non-standard, rendering data retrieval very difficult.
Anthony M. Ordile	4/24/96	Loss Control Associates, Langhorne, PA	215/750-6841	no data	Loss history mostly involves fires; no history of human injury or death from exposure to ammonia.
Mark I. Grossman, CIH, CSP; Director, Environmental and Chemical Sciences	4/4/96	Reliance National Insurance Company	212/858-3599	no data	Data on anhydrous ammonia is difficult if not impossible to retrieve because of the coding system for claims, in which "inhalation" is a more likely category than "ammonia." Perhaps original NIOSH Criteria Document has accident data.
chemical companies					
...	4/4/96	Chevron Corporations	510/242-7000	...	no return telephone call
Norman E. Scheffler, PE Process Safety/Loss Prevention Associate	4/4/96	Dow Chemical Company	517/636-2457	no data	referral to Steve Phillips (517/656-0228), product steward for Dow anhydrous ammonia
Steve Phillips, Chief Product Steward (formerly including 'obsolete products')	4/4/96	Dow Chemical Company	517/656-0228	no data	Dow no longer produces NH3. No knowledge of data on lethality. Such data, if it exists, would be difficult to retrieve. Referral to Marlies Greenwood, Product Steward, Freeport, TX (409/238-2805).
Marlies Greenwood, Product Steward (including 'obsolete products')	4/4/96	Dow Chemical Company	409/238-2805	no data	Recalls truck overturning (1981-86?) on Houston Freeway; ammonia release --> injuries but probabaly no deaths. Referral to TX DOT, Austin (512/482-5474). Dow stopped producing NH3 because of business reasons. Recalls no product liability/claims/losses.
Alan Holiday, Technical Services Superintendent	4/4/96	Farmland Industries	913/865-1227	no data	referral to Jim Skillen, CHHM; Director of Environment and Energy Programs, National Fertilizer Institute; Washington, DC; (202/675-8250)

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Bruce A. Jacobsen	...	Olin Chemical Company	203/356-2000	...	...
Manfred Noack	...	Olin Chemical Company	203/356-2000	...	...
Paul S. Loomis Senior Applications Specialist	...	Olin Chemical Company	203/271-4171	...	...
Stu Grossberg, Industrial Hygienist	4/4/96	Olin Corporation	203/271-4157	no data	Olin no longer produces anhydrous ammonia. Referral to Olin toxicologist Nicholas Skoulas, PhD; Metals Research Laboratory, Science Park, New Haven, CT (203/495-8550)
Nicholas Skoulas, PhD	4/10/96, 4/12/96, 4/25/96	Olin Corporation, Metals Research Lab., Science Park, New Haven, CT	203/495-8550	no data	Believes cessation of anhydrous ammonia production was merely a business decision; Olin produces far more dangerous materials, incl. explosives. Plant records archived.
federal agencies					
Sue Cairelli, Toxicologist	4/24/96, 4/25/96	NIOSH, US DHHS	800/356-4674; 202/401-0721; 513/533-8364 (direct)	no data	IDLH changed from 500 –> 300 ppm 9/15/94; documentation provided. Value is based upon Henderson & Haggard, 1946; and Silverman, Whittenberger, and Muller; 1946. Several complaints received; IDLH will be reevaluated, with time frame undecided.
Robert Lobby	4/8/96	NTSB (National Transportation Safety Board)	202/382-6735	no data	He recalls no ammonia transport incident investigation during his five-year tenure at NTSB. Referral to Bob Chipkevich, Chief of HazMat, Pipeline Division (202/382-6585).
Bob Chipkevich, Chief of HazMat Operations, Pipeline Division	4/10/96	NTSB (National Transportation Safety Board)	202/382-6585	no data	Will provide NTSB-RAR-71-2 (railroad accident report).
Ira Wainless	4/4/96, 4/5/96	U. S. Dept. of Labor/OSHA	202/219-7056	LC-LO = 30,000 ppm @ 5 min. (Lewis, 1996)	source: Mulder, J. S., and H. O. Van Der Zalm. Een gaval van dodelijke ammoniakvergiftiging (Fatal case of ammonia poisoning). Tijdschrift Voor Sociale Geneeskunde (Rotterdam, The Netherlands), 45:458-60, 1967. Source document analyzed in text.
trade associations					
Jim Skillen, CHHM; Director of Environment and Energy Programs	4/4/1996, 4/11/96	National Fertilizer Institute	202/675-8250	no data	Interested in RAM TRAC's analysis; want to meet with me, evaluate white paper, and possibly initiate federal regulatory or legislative change.
Donna Slayton, Fire Analysis Dept.	4/24/96	National Fire Protection Association	617/770-3000; 617/984-7463 (direct)	no data	Conducted database search for fire incidents involving ammonia. For results, see Appendix B.

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CONCLUSIONS

Standards and Guidelines, Odor Threshold, and Vapor Density

The reported range of the ammonia odor threshold (0.04 to 57 ppm) corresponds to the range of occupational standards and guidelines for prolonged exposure to ammonia. The odor threshold is also significantly below the ammonia IDLH value of 300 ppm. This value represents the concentration which, if exceeded for 30 minutes, may render an exposed individual incapable of escape. Consequently, the distinct odor of ammonia, and the relatively low odor threshold, constitute excellent warning properties. Individuals would be capable of detecting ammonia nearly simultaneously with the onset of exposure. In circumstances in which ammonia might gradually increase to clinically significant concentrations, the low odor threshold may constitute an early warning system, alerting exposed individuals of the need to take corrective and/or protective actions. The low odor threshold of ammonia may be regarded as a risk mitigating factor rather than a risk-enhancing factor, compared with more-difficult-to-detect gases, such as carbon monoxide. Nonetheless, the excellent warning properties of ammonia do not constitute a substitute for its routine quantitative monitoring at potential release sites.

The finding that gaseous ammonia exhibits a density of 0.6 relative to air indicates that ammonia releases would tend to rise in tranquil air. However, liquid ammonia may be cryogenically cooled, such that its initial density upon release may be heavier than air (42). Equilibration with outdoor ambient temperature would be expected to result in a gradual transition to a lighter-than-air condition.

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Nonetheless, indoor settings may inhibit effective ammonia dissipation, and both outdoor and indoor air might be sufficiently turbulent to overcome the density-driven tendency of ammonia to rise out of the personal breathing zone of occupationally exposed individuals. In contrast, environmentally exposed individuals would presumably be situated outdoors, where ammonia typically would not be confined. The low vapor density of ammonia relative to air may be regarded, therefore, as a risk mitigating factor rather than a risk-enhancing factor, compared with heavier-than-air gases such as carbon monoxide or chlorine.

Relationship Between Concentration In Air Vs. Exposure Time In Producing Toxic Effect

An approximation in toxicology (sometimes termed 'Haber's rule') is that the product of the dose and duration of exposure to a particular toxic substance equals a constant value (54, 72). This rule is approximate for several reasons, explication of which is beyond the scope of this report (72). However, the rule often may be usefully applied to closely spaced exposure durations, such as one minute vs. one hour. Thus, a one-minute inhalation exposure to a particular dosing rate (or concentration in air) might be approximately equivalent to a one-hour exposure to 1/60th of the one-minute dosing rate (or concentration in air). The relationship elucidated above may be used to define upper time limits for inhalation exposure to ammonia over a time frame of up to one hour (54).

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Adoption of Acceptable Risk Criteria

Risk acceptability is a subjective judgment, and cannot be defined scientifically. However, qualitatively different adverse health effects may be proposed as being acceptable, and selection of an acceptable risk may be made from a menu of adverse effects presented. The menu items might range from no adverse health effect, to clinically insignificant effects, to reversible injury, to permanent injury, to lethality. For anhydrous ammonia these benchmarks may be quantified based upon holistic consideration of detailed toxicological data, including human clinical data, animal bioassay data, and reports of industrial ammonia release accidents. Ideally, all data sources converge upon a single value for each toxicological benchmark parameter to be quantified. However, as described earlier and in detail in the *Findings* section, clinical reports have produced lower values than bioassay studies involving animals or studies reconstructing industrial accidents involving ammonia release. Reconciling these disparate data sources constitutes the central focus of the following subsections, which seek to quantify toxicology benchmark parameters characterizing occupational and environmental exposure to anhydrous ammonia.

Reversible or Clinically Insignificant Effect Concentration

Each of the adverse health effects for which benchmarks have been identified above may be proposed as an effect exerted by ammonia to be averted by an industrial policy of limiting maximum exposure concentrations and durations. With respect to reversible or clinically

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insignificant effects, the OSHA TLV of 50 ppm may be adopted. This benchmark also corresponds to the concentration causing faint to moderate upper airway irritation in five of six volunteers inhaling ammonia at 50 ppm for 10 minutes (Table 3).

Irreversible Injury Concentration

A reasonable candidate value for the benchmark for irreversible injury is 300 ppm for 30 minutes, corresponding to the NIOSH IDLH value (Table 2). Selection of this regulatory criterion in preference over toxicological data would seem desirable, given the apparent unavailability of detailed dose-response human toxicology data documenting irreversible injury from ammonia inhalation. However, use of the IDLH value is undesirable for two reasons. First, the IDLH value is based upon secondary literature, specifically, Henderson and Haggard's second edition (1943), which reflects the value initially published in the first edition (1927; 22). Technical support provided in this source was evaluated in the *Findings* section, and found to consist of old reports published in 1886 and 1909 in a German source, *Archiv für Hygiene*. The age of these articles, and the NIOSH statement that they were not evaluated, undermine the confidence to be placed in the NIOSH IDLH derivation.

Second, IDLH values intrinsically do not qualify as toxicological benchmarks because they do not necessarily indicate the concentration at which a toxic effect occurs. Rather, they may indicate the onset of a physiological condition which might inhibit escape from further exposure to an entirely unrelated peril, such as fire. The IDLH might also indicate the concentration which, if exceeded, would inhibit escape

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from further exposure to the subject substance, where the further exposure added to the 30 minutes of initial exposure (but not the initial exposure by itself) might induce irreversible toxic effects or death. Thus, even a non-toxic sleeping pill might be assigned an IDLH value because of its potential effect upon a person possibly needing to escape an industrial emergency.

The irreversible injury concentration may be derived in a manner consistent with derivation of the IDLH value (defined above), but adjusted to assure that the basis for the value is a technically supportable toxic effect of ammonia. The *Introduction* section of the 1994 NIOSH *Documentation for Immediately Dangerous to Life or Health Concentrations* (IDLHs; 46) sets forth the recommended method of IDLH derivation. Specifically, the IDLH is initially estimated as one tenth the LC_{50} value. Subsequent adjustment may be necessary based upon examination of other types of data, such as explosivity limits and the concentrations at which respiration is reduced 50 percent in mice or rats exposed for 10 minutes (RD_{50}). This criterion is adopted for the ammonia irreversible injury concentration, as quantified in the next subsection, pertaining to fatal exposure concentrations.

Fatal Exposure Concentration

The benchmark concentration in air selected for fatal exposure of humans to anhydrous ammonia may be derived from any of several sources, including animal studies, human studies, and ammonia accident reports in which human exposure has resulted in fatalities. These sources should ideally produce consistent results but, in the case

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of ammonia, they do not. They will be addressed sequentially in the paragraphs that follow, and then synthesized to produce a conservative estimate of the ammonia lethality concentration.

Animal Studies

Numerous animal bioassay reports were addressed in the *Findings* section and, in Table 4, adjusted to equivalent human lethality values using standard U. S. EPA methodology (77), which differs somewhat from NIOSH methodology for deriving IDLH values (46, 72). Concentrations of airborne ammonia whose lethality to humans is estimated based upon animal bioassay studies produced an arithmetic mean inhalation LC_{LO} value of 318,830 ppm at five minutes, and 53,138 ppm at 30 minutes. Geometric mean values were 299,013 ppm and 49,836 ppm; respectively. The minimum human equivalent LC_{LO} value was 203,762 at five minutes, and 33,960 at 30 minutes.

Although the LC_{LO} parameter is more conservative than the LC_{50} parameter, the dearth of LC_{LO} values has produced a situation in which the LC_{50} values are lower than the LC_{LO} values. Consequently, both parameters will be considered together. The arithmetic mean human equivalent inhalation LC_{50} or LC_{LO} value was 308,556 ppm at five minutes, and 51,426 at 30 minutes. Geometric mean values were 144,290 ppm and 24,048 ppm; respectively. The minimum human equivalent LC_{50} or LC_{LO} value was 98,410 ppm (actually an LC_{50} value) at five minutes, and 16,402 at 30 minutes.

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Human Studies

Human lethality values derived from animal data, as synopsized above, were significantly higher than lethality values derived from clinical reports of human exposure incidents. The latter produced a five-minute arithmetic mean inhalation lethality value of 95,000 ppm, and a 30-minute value of 15,833 ppm. Geometric mean values were 39,482 ppm and 6,580 ppm; respectively. The minimum human equivalent lethality value was 5,000 ppm at five minutes, and 833 ppm at 30 minutes. Thus, a disparity exists between lethality data derived from animal vs. human studies, and this disparity is approximately 20-fold when the most conservative (minimum) lethality values are considered (98,410 ppm/5,000 ppm = 20 at five minutes; 16,402 ppm/ 833 ppm = 20 at 30 minutes).

Accident Reports

The human lethality concentrations resulting from evaluation of accident reports may validate either the human lethality values suggested by clinical reports, the human lethality values suggested by reports of animal bioassays, or neither. Clearly, the most reliable and best documented source of available accident data is the Potchefstroom, South Africa ammonia release incident, which killed 18 people, as described in detail in the *Findings* section and in Table 6 supporting the text description. The findings of the Table 6 analysis may be visualized in Fig. 2.

Fig. 2 illustrates the effect of increasing peak, one-minute, and five-minute time-weighted average anhydrous ammonia concentrations in air on the human fatality rate. The LC_{50} values are estimated to be a peak of 560,000 ppm, a one-minute mean of 268,832 ppm, and a

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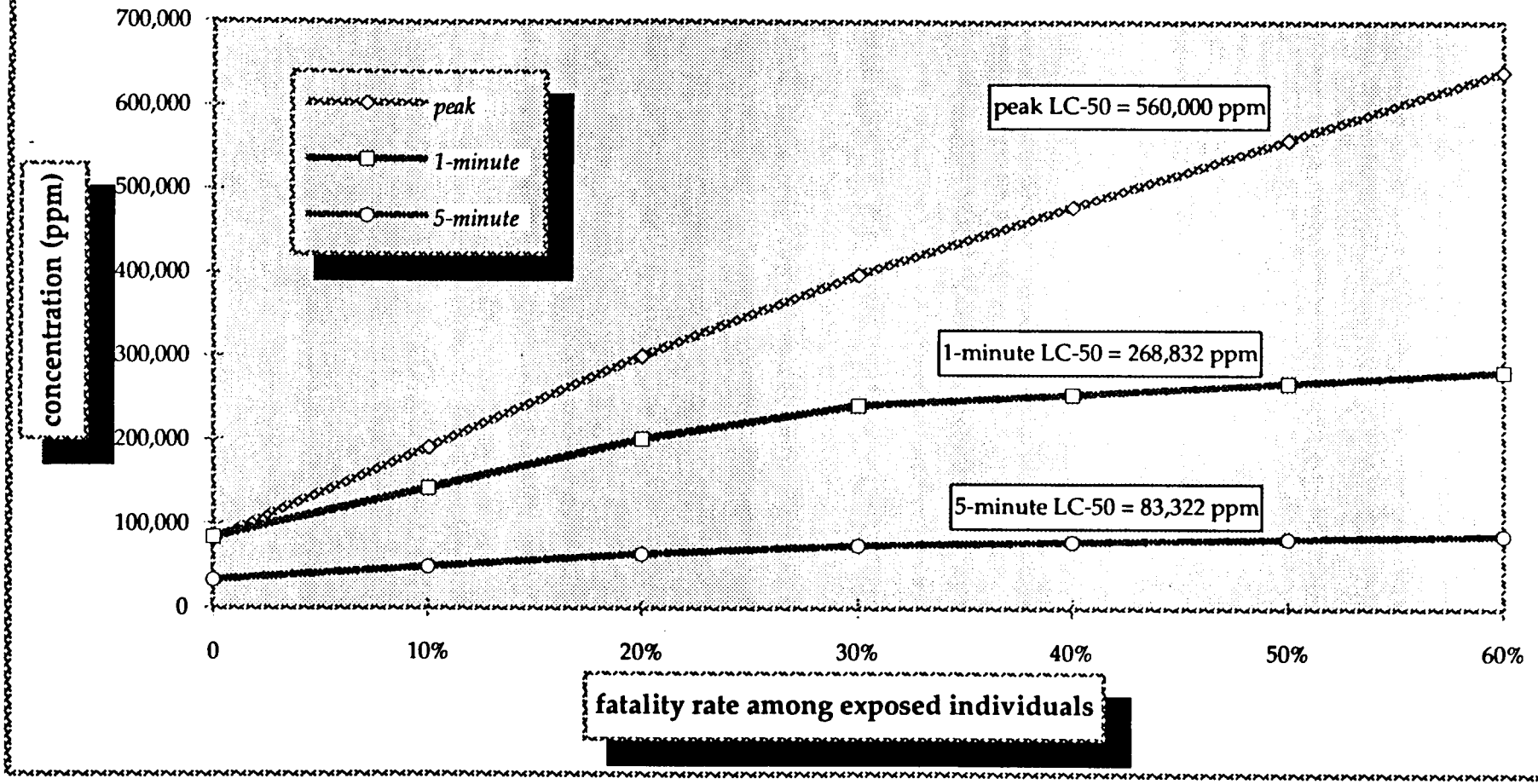
Fig. 2. Estimated Peak, 1-Minute, and 5-Minute Lethal Ammonia Concentrations Based Upon Fatalities In A 1974 Potchefstroom, South Africa Accident*



*Source: derived from Mudan, K., and K. Mitchell. "Report on the Potchefstroom, South Africa Ammonia Incident." Columbus, Ohio; Four Elements, Inc.; 15 pp., March 1996.

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five-minute mean of 83,322 ppm. Longer-term values were not generated because the air modeling addressed an exposure period of under 10 minutes. However, the five-minute LC_{50} value is comparable to (85 percent of) the minimum five-minute human lethality value (LC_{LO} or LC_{50}) derived from animal bioassay data, specifically; 98,410 ppm (Table 4).

In contrast, the 83,322-ppm five-minute LC_{50} value generated from the Potchefstroom, South Africa accident analysis is nearly 17 times the minimum lethality value (5,000 ppm) cited in multiple sources based upon clinical data (Table 4). However, it is more nearly consistent with Henderson and Haggard (1927; 22) assuming that their use of the phrase *"short duration"* is interpreted to mean one hour at 5,000 ppm and 1/2 hour at 10,000 ppm. In either case, this assumption produces a five-minute equivalent lethality value of 60,000 ppm, which is within 28 percent of the accident-analysis-based five-minute LC_{50} value of 83,322 ppm. A reasonable inference from analysis of available accident data is that they validate adjustment of animal lethality concentrations to human equivalent values, and refute lower values reported in clinical reports; except for Henderson and Haggard, under the assumption stated above.

Personal Communications

The purpose of the telephone inquiry program was to provide opportunities for contacted individuals to refute the findings of minimum ammonia lethality concentrations by citing evidence of even lower lethality values. As Table 7 demonstrates, significant effort was expended to challenge the quantitative lethality findings made based upon animal bioassay data and industrial accident analyses. As detailed in *Findings,* these efforts resulted neither in confirmation of

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the significantly lower human lethality values derived based upon clinical reports, nor in data showing lethality concentrations below those derived from animal bioassays or accident report analyses. This absence of data refuting the higher human lethality values supports their reliability.

Selection of Human Lethality and Irreversible Injury Values

Lethality. The Potchefstroom accident database represents the most conservative source of data, assuming rejection of the patently flawed human clinical database suggesting that 5,000 ppm would be fatal within five to 10 minutes. The most conservative five-minute inhalation lethality concentration available within the Potchefstroom database is the zero-fatality benchmark of 33,737 ppm. Thus, a highly conservative five-minute human inhalation lethality concentration for anhydrous ammonia would be >33,737 ppm, equivalent to >5,623 ppm over 30 minutes.

Irreversible injury. The adopted criterion for irreversible injury, elucidated and justified earlier, is 1/10th the human LC_{50} value. The human LC_{50} value derived here, specifically; 83,322 ppm over five minutes; is the minimum LC_{50} value derived from a large database of animal bioassays and accident analyses. This yields an irreversible injury concentration of 8,322 ppm at five minutes, and 1,387 ppm at 30 minutes. Extrapolating beyond 30 minutes yields a 60-minute value of 694 ppm, which is 31 percent below the 60-minute ERPG 3 value of 1,000 ppm (78-80). However, the two one-hour values must be deemed indistinguishable given the approximate nature of Haber's rule. Thus,

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a 60-minute irreversible injury level of 1,000 ppm will be adopted, with values for durations between 30 and 60 minutes to be derived by interpolating between 1,387 and 1,000 ppm, respectively, which are the concentration values corresponding to the lower and upper boundaries of this exposure duration range. For example, a 45-minute irreversible injury value, midway between 30 and 60 minutes, can be obtained by calculating the midpoint between 1,387 and 1,000 ppm, which is 1,194 ppm (rounded upward from 1,193.5 ppm).

Dose-response isolines. The above single-point benchmarks, defined by a single combination of exposure concentration and duration, may be permuted based upon the approximation described earlier that the product of dose and duration equals a constant. The resulting dose-response isolines are depicted in Figures 3 and 4. Both figures provide equivalent information, except that Figure 4 depicts the ammonia concentration as a logarithm because of the difficulty, evident in Figure 3, of graphing a concentration range spanning multiple orders of magnitude on a single arithmetically scaled axis.

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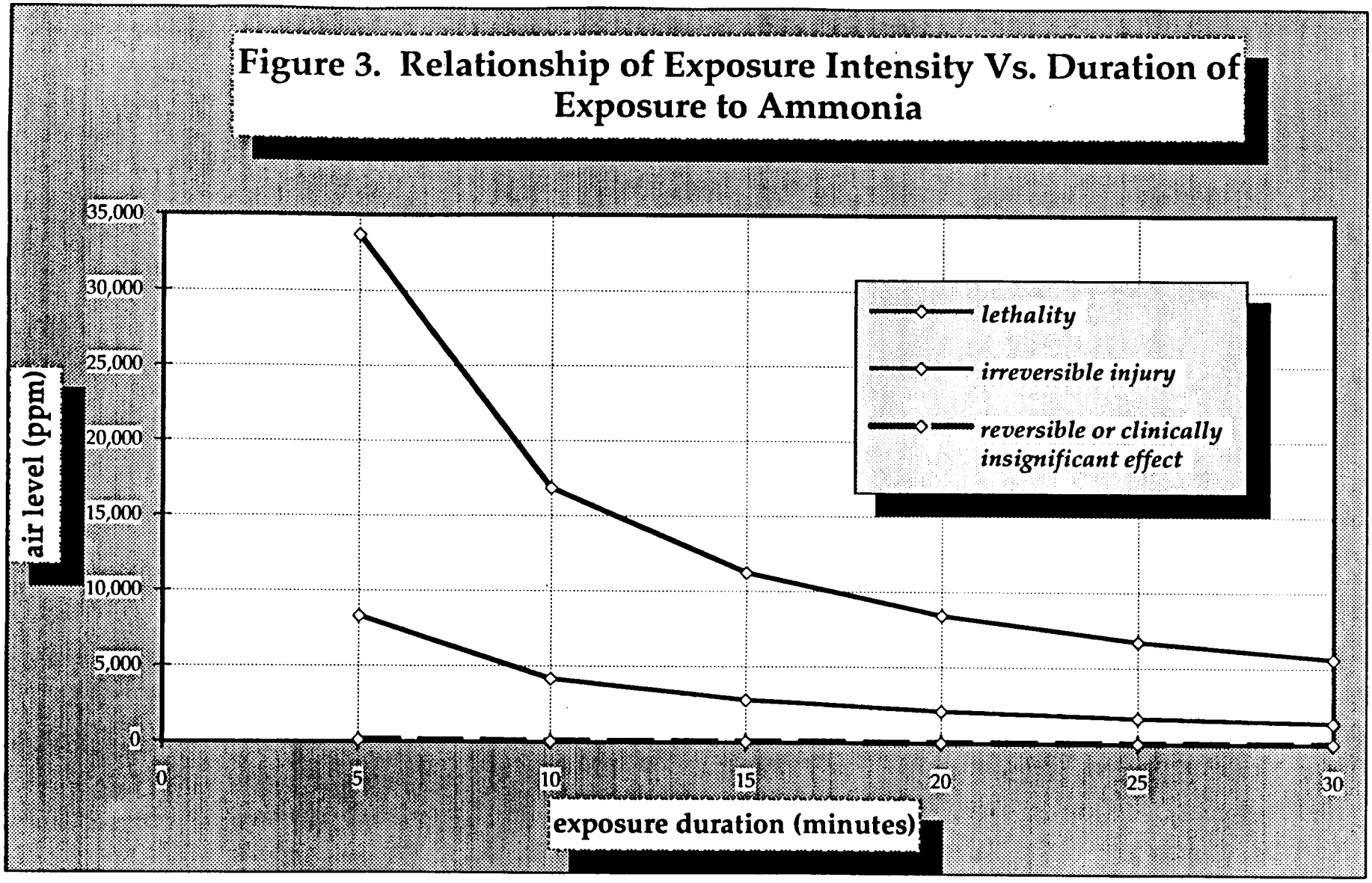

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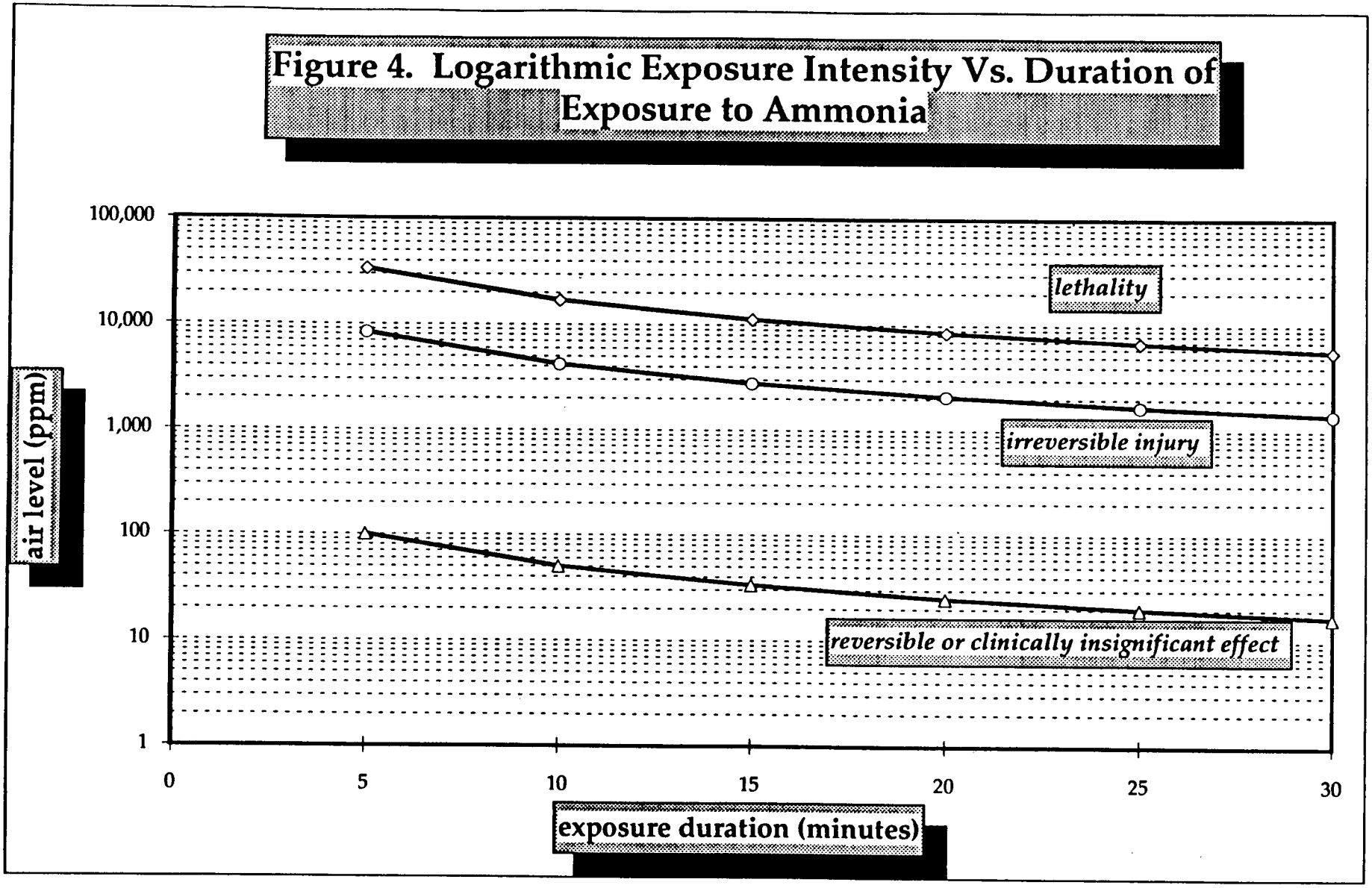

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UNCERTAINTIES

Confidence in estimated exposure concentrations reported in the literature must be regarded as being low given the difficulty of reconstructing exposure scenarios resulting in fatal or non-fatal poisoning of people. Ammonia concentrations infered from discernable effects on plants and animals in the vicinity of ammonia releases also must be regarded as being low. Further, this report was based upon a variety of data source types, including examination of a mixture of primary research articles and secondary review-type articles and on-line database reports. In the small fraction of cases in which primary research articles were not obtained, RAM TRAC cannot be certain that each secondary source accurately reported the primary source data, or that all useful primary data were reported in the secondary sources.

To compensate for these and other possible sources of uncertainty, criteria for each toxic effects benchmark identified in this report have been quantified in a highly conservative manner. Despite the conservatism arising from this precaution, however, additional conservatism might be feasibly achieved. For example, in designing facilities against the risk of ammonia release, reductions in exposure durations and/or concentrations might be assured. Such caution is generally advisable.

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APPENDIX A:

AMMONIA

INHALATION TOXICOLOGY

BENCHMARKS

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Table A-1. Acute Inhalation Effects Exerted By Ammonia In Studies Involving Animals, By Species

conc. in air	assumed exposure duration	dose x duration	study type	reported effect(s)	primary data source(s)*	secondary data source(s)*
(ppm)	(min.)	(ppm.min.)	...	...	...	...
studies involving cats						
1,071	60	64,262	LC-50	LETHALITY: dynamic air flow; Environment Canada benchmark (draft)	8	10
7,035	60	422,096	LC-LO	LETHALITY: Environment Canada benchmark (draft)	8	10
10,050	60	602,995	LC-50	NEUROLOGICAL: flaccid paralysis without anaesthesia	5	7, 10, 15
10,050	60	602,995	LC-50	NEUROLOGICAL: excitement	5	7, 10, 15
10,050	60	603,000	LC-50	LETHALITY: lethal to half of exposed animals	5	4, 7, 10
10,122	60	607,302	LC-50	LETHALITY: static air flow; Environment Canada benchmark (draft)	8	10
studies involving guinea pigs						
5,000	5	25,000	LC-LO	LETHALITY: toxic effects not reported	3	15
5,025	5	25,125	LC-LO	LETHALITY: Environment Canada benchmark (draft)	8	10
studies involving mice						
100	360	36,000	LOAEL	NEUROLOGICAL: sensory irritation	16	4
303	...	...	RD-50	RESPIRATORY: 50-percent reduction of respiratory rate	6	10
10,201	10	102,007	LC-50	LETHALITY: Environment Canada benchmark (draft)	8	10
4,070	60	244,200	LOAEL	RESPIRATORY: alveolar destruction	11	4
4,070	60	244,200	LOAEL	CARDIOLOVASCULAR: atrophy of pericardial fat	11	4
4,070	60	244,200	LOAEL	RESPIRATORY: intra-alveolar hemorrhage	11	4
4,070	60	244,200	LOAEL	HEPATIC: increased liver weight, necrosis	11	4

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Table A-1 (continued from previous page)

conc. in air	assumed exposure duration	dose x duration	study type	reported effect(s)	primary data source(s)*	secondary data source(s)*
(ppm)	(min.)	(ppm.min.)	...	...	...	...
4,230	60	253,800	LC-50	NEUROLOGICAL: tremor; convulsions or effect on seizure threshold; ataxia	2	15
4,230	60	253,800	LC-50	LETHALITY: lethal to half of exposed animals	11	4
4,824	60	289,437	LC-50	LETHALITY: Environment Canada benchmark (draft)	8	10
4,752	120	570,261	LC-50	LETHALITY: Environment Canada benchmark (draft)	8	10
21,430	30	642,900	LC-50	LETHALITY: lethal to half of exposed animals	9	4
11,299	60	677,940	LC-50	LETHALITY: lethal to half of exposed animals	13	4
1,000	960	960,000	NOAEL	LETHALITY: no observed effect (lethality 0/4)	17	4, 10
studies involving rabbits						
50	180	9,000	LOAEL	RESPIRATORY: decreased respiratory rate, increased depth	12	4
100	180	18,000	NOAEL	HEPATIC: unspecified	12	4
100	180	18,000	NOAEL	RENAL: unspecified	12	4
2,500	60	150,000	LOAEL	CARDIOVASCULAR: bradycardia	14	4
5,000	60	300,000	LOAEL	RESPIRATORY: acute pulmonary edema	14	4
5,000	60	300,000	LOAEL	CARDIOVASCULAR: hypertension, acidosis, EKG change	14	4
7,035	60	422,096	LC-LO	LETHALITY: Environment Canada benchmark (draft)	8	10
10,050	60	602,995	LC-50	NEUROLOGICAL: flaccid paralysis without anaesthesia	5	7, 10, 15
10,050	60	602,995	LC-50	NEUROLOGICAL: excitement	5	7, 10, 15
10,050	60	603,000	LC-50	LETHALITY: lethal to half of exposed animals	5	4, 10
10,122	60	607,302	LC-50	LETHALITY: Environment Canada benchmark (draft)	8	10
studies involving rats						
2,010	240	482,396	LC-LO	LETHALITY: Environment Canada benchmark (draft)	8	10
100	360	36,000	LOAEL	NEUROLOGICAL: sensory irritation	16	4

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Table A-1 (continued from previous page)

conc. in air	assumed exposure duration	dose x duration	study type	reported effect(s)	primary data source(s)*	secondary data source(s)*
(ppm)	(min.)	(ppm.min.)	...	...	...	...
17,401	15	261,015	LC-50	LETHALITY: lethal to half of exposed animals	13	4
7,322	60	439,325	LC-LO	LETHALITY: Environment Canada benchmark (draft)	8	10
2,000	240	480,000	LC-50	LETHALITY: toxic effects not reported	1	15
10,911	60	654,680	LC-50	LETHALITY: Environment Canada benchmark (draft)	8	10
1,000	960	960,000	LOAEL	LETHALITY: lethal (1/8), no observed effect (7/8)	17	4, 10
1,000	960	960,000	LOAEL	NEUROLOGICAL: brain congestion at autopsy following death 12 h postexposure (1/8)	17	4, 10

*Data sources: 'primary' denotes source of original research or stated conclusion; 'secondary' denotes additional source of information about primary source(s).

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Table A-2. Acute Inhalation Effects Exerted By Ammonia In Studies Involving Animals, By Effect

conc. in air	assumed exposure duration	exposure regimen	dose x duration	species	study type	reported effect(s)	primary data source(s)*	secondary data source(s)*
(ppm)	(min.)	(h/d)	(ppm.min.)	...	...	...	...	...
cardiovascular effects								
2,500	60	1.00	150,000	rabbit	LOAEL	CARDIOVASCULAR: bradycardia	14	4
4,070	60	1.00	244,200	mouse	LOAEL	CARDIOLOVASCULAR: atrophy of pericardial fat	11	4
5,000	60	1.00	300,000	rabbit	LOAEL	CARDIOVASCULAR: hypertension, acidosis, EKG change	14	4
hepatic (liver) effects								
100	180	3.00	18,000	rabbit	NOAEL	HEPATIC: unspecified	12	4
4,070	60	1.00	244,200	mouse	LOAEL	HEPATIC: increased liver weight, necrosis	11	4
lethality benchmarks recognized by Environment Canada								
2,010	5	0.08	10,050	rat	LC-LO	LETHALITY: Environment Canada benchmark (draft)	8	10
5,025	5	0.08	25,125	guinea pig	LC-LO	LETHALITY: Environment Canada benchmark (draft)	8	10
1,071	60	1.00	64,262	cat	LC-50	LETHALITY: dynamic air flow; Environment Canada benchmark (draft)	8	10
10,201	10	0.17	102,007	mouse	LC-50	LETHALITY: Environment Canada benchmark (draft)	8	10
4,824	60	1.00	289,437	mouse	LC-50	LETHALITY: Environment Canada benchmark (draft)	8	10
7,035	60	1.00	422,096	rabbit	LC-LO	LETHALITY: Environment Canada benchmark (draft)	8	10
7,035	60	1.00	422,096	cat	LC-LO	LETHALITY: Environment Canada benchmark (draft)	8	10
7,322	60	1.00	439,325	rat	LC-LO	LETHALITY: Environment Canada benchmark (draft)	8	10
4,752	120	2.00	570,261	mouse	LC-50	LETHALITY: Environment Canada benchmark (draft)	8	10
10,122	60	1.00	607,302	rabbit	LC-50	LETHALITY: Environment Canada benchmark (draft)	8	10
10,911	60	1.00	654,680	rat	LC-50	LETHALITY: Environment Canada benchmark (draft)	8	10

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Table A-2 (continued from previous page)

conc. in air	assumed exposure duration	exposure regimen	dose x duration	species	study type	reported effect(s)	primary data source(s)*	secondary data source(s)*
(ppm)	(min.)	(h/d)	(ppm.min.)	...	...	...	...	...
lethality studies								
5,000	5	0.08	25,000	mammal	LC-LO	LETHALITY: toxic effects not reported	3	15
4,230	60	1.00	253,800	mouse	LC-50	LETHALITY: lethal to half of exposed animals	11	4
17,401	15	0.25	261,015	rat	LC-50	LETHALITY: lethal to half of exposed animals	13	4
2,000	240	4.00	480,000	rat	LC-50	LETHALITY: toxic effects not reported	1	15
10,050	60	1.00	603,000	cat	LC-50	LETHALITY: lethal to half of exposed animals	5	4, 7, 10
10,050	60	1.00	603,000	rabbit	LC-50	LETHALITY: lethal to half of exposed animals	5	4, 10
10,122	60	1.00	607,302	cat	LC-50	LETHALITY: static air flow; Environment Canada benchmark (draft)	8	10
21,430	30	0.50	642,900	mouse	LC-50	LETHALITY: lethal to half of exposed animals	9	4
11,299	60	1.00	677,940	mouse	LC-50	LETHALITY: lethal to half of exposed animals	13	4
1,000	960	16.00	960,000	rat	LOAEL	LETHALITY: lethal (1/8), no observed effect (7/8)	17	4, 10
1,000	960	16.00	960,000	mouse	NOAEL	LETHALITY: no observed effect (lethality 0/4)	17	4, 10
neurological effects								
100	360	6.00	36,000	rat	LOAEL	NEUROLOGICAL: sensory irritation	16	4
100	360	6.00	36,000	mouse	LOAEL	NEUROLOGICAL: sensory irritation	16	4
4,230	60	1.00	253,800	mouse	LC-50	NEUROLOGICAL: tremor; convulsions or effect on seizure threshold; ataxia	2	15
10,050	60	1.00	602,995	cat	LC-50	NEUROLOGICAL: excitement	5	7, 10, 15
10,050	60	1.00	602,995	rabbit	LC-50	NEUROLOGICAL: excitement	5	7, 10, 15
10,050	60	1.00	602,995	cat	LC-50	NEUROLOGICAL: flaccid paralysis without anaesthesia	5	7, 10, 15
10,050	60	1.00	602,995	rabbit	LC-50	NEUROLOGICAL: flaccid paralysis without anaesthesia	5	7, 10, 15
1,000	960	16.00	960,000	rat	LOAEL	NEUROLOGICAL: brain congestion at autopsy following death 12 h postexposure (1/8)	17	4, 10
renal (kidney) effects								
100	180	3.00	18,000	rabbit	NOAEL	RENAL: unspecified	12	4

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Table A-2 (continued from previous page)

conc. in air	assumed exposure duration	exposure regimen	dose x duration	species	study type	reported effect(s)	primary data source(s)*	secondary data source(s)*
(ppm)	(min.)	(h/d)	(ppm.min.)	...	...	...	...	...
respiratory system effects								
50	180	3.00	9,000	rabbit	LOAEL	RESPIRATORY: decreased respiratory rate, increased depth	12	4
303	...	...	...	mouse	RD-50	RESPIRATORY: 50-percent reduction of respiratory rate	6	10
4,070	60	1.00	244,200	mouse	LOAEL	RESPIRATORY: alveolar destruction	11	4
4,070	60	1.00	244,200	mouse	LOAEL	RESPIRATORY: intra-alveolar hemorrhage	11	4
5,000	60	1.00	300,000	rabbit	LOAEL	RESPIRATORY: acute pulmonary edema	14	4

*Data sources: 'primary' denotes source of original research or stated conclusion; 'secondary' denotes additional source of information about primary source(s).

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Table A-3. Inhalation Effects Exerted By Multi-Day Exposure of Animals To Ammonia

conc. in air	assumed exposure duration	exposure regimen (known or assumed)	dose x duration	species	reported effect(s)	primary data source(s)*	secondary data source(s)*
(ppm)	(days)	(h/d)	(ppm.d.h/d.)	...	...	...	...
studies reporting no-observed-adverse-effect-levels (NOAELs)							
100	7	24	16,800	pig	HEMATOLOGICAL: unspecified	4	2
714	7	24	119,952	rat	LETHALITY: absence of systemic toxicity	10	2
10	3	24	720	pig	METABOLIC: reduced food intake and weight gain	11	2
10	3	24	720	pig	OCULAR: irritation	11	2
10	35	24	8,400	pig	OCULAR: ocular irritation	11	2
155	5	8	6,200	rabbit	OCULAR: temporary lacrimation	3	2
10	3	24	720	pig	RESPIRATORY: frequent coughing	11	2
10	3	24	720	pig	RESPIRATORY: oral and nasal irritation	11	2
155	5	8	6,200	dog	RESPIRATORY: temporary dyspnea	3	2
225	5	8	9,000	rabbit	RESPIRATORY: temporary dyspnea	3	2
714	7	24	119,952	rat	RESPIRATORY: absence of lesions of trachea, lungs, respiratory epithelium	10	2
studies reporting lowest-observed-adverse-effect-levels (LOAELs)							
500	7	24	84,000	mouse	IMMUNOLOGICAL: decreased resistance to infection	7	2
653	90	8	470,336	rat	LETHALITY: death in the multiple dose data field	3	8
675	90	8	485,841	guinea pig	LETHALITY: death in the multiple dose data field	3	8

continued on following page

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Table A-3 (continued from previous page)

conc. in air	assumed exposure duration	exposure regimen (known or assumed)	dose x duration	species	reported effect(s)	primary data source(s)*	secondary data source(s)*
(ppm)	(days)	(h/d)	(ppm.d.h/d.)	...	...	...	...
50	3	24	3,600	pig	METABOLIC: reduced food intake and weight gain	11	2
50	7	24	8,400	pig	METABOLIC: reduced weight gain	5	2
300	5	6	9,000	rat	METABOLIC: elevated amino acids in blood and brain	6	8
500	5	24	60,000	mouse	METABOLIC: altered enzyme activity	9	2
500	7	24	84,000	rat	METABOLIC: decreased food intake and body weight	7	2
145	35	24	121,800	pig	METABOLIC: food intake, altered weight gain rate	11	8
300	5	6	9,000	rat	NEUROLOGICAL: degenerative changes in brain and/or coverings	6	8
500	5	24	60,000	mouse	NEUROLOGICAL: lethargy	9	2
100	28	24	67,200	pig	NEUROLOGICAL: lethargy	5	2
1,378	119	4	656,058	rat	NEUROLOGICAL: recordings from specific central nervous system areas	1	8
50	3	24	3,600	pig	OCULAR: irritation	11	2
50	7	24	8,400	pig	OCULAR: excessive lacrimation	5	2
100	7	24	16,800	pig	OCULAR: irritation	4	2
50	35	24	42,000	pig	OCULAR: ocular irritation	11	2
1,105	5	8	44,200	rabbit	OCULAR: temporary lacrimation	3	2
675	90	8	485,841	rabbit	OCULAR: corneal damage; other eye effects	3	8
675	90	8	485,841	dog	OCULAR: lacrimation	3	8
675	90	8	485,841	guinea pig	RENAL: changes in tubules	3	8

continued on following page

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Table A-3 (continued from previous page)

conc. in air	assumed exposure duration	exposure regimen (known or assumed)	dose x duration	species	reported effect(s)	primary data source(s)*	secondary data source(s)*
(ppm)	(days)	(h/d)	(ppm.d.h/d.)	...	...	...	...
50	3	24	3,600	pig	RESPIRATORY: frequent coughing	11	2
50	3	24	3,600	pig	RESPIRATORY: oral and nasal irritation	11	2
100	7	24	16,800	pig	RESPIRATORY: excessive nasal secretion, coughing	5	2
770	5	8	30,800	dog	RESPIRATORY: temporary dyspnea	3	2
1,105	5	8	44,200	rabbit	RESPIRATORY: temporary dyspnea	3	2
500	7	24	84,000	rat	RESPIRATORY: irritation	7	2
714	7	24	119,952	rat	RESPIRATORY: mild epithelial damage	10	2
653	90	8	470,336	rat	RESPIRATORY: nasal irritation, dyspnea	3	8
675	90	8	485,841	guinea pig	RESPIRATORY: other changes	3	8

*Data sources: 'primary' denotes source of original research or stated conclusion; 'secondary' denotes additional source of information about primary source(s).

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APPENDIX B:

NEWS CLIPPINGS

OF ANHYDROUS AMMONIA

RELEASE

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THE DAILY GAZETTE
B
FRIDAY
MARCH 29, 1996

Toxic leak is feared

Judge orders drain of ammonia from Albany warehouse

By BRIAN NEARING
Gazette Reporter

ALBANY — Concern over a possible toxic ammonia leak from the aging refrigeration system of a massive warehouse prompted a federal bankruptcy judge Thursday to order a plan for the system to be drained.

Small amounts of ammonia have apparently leaked from the system at Central Warehouse in the past, but those discharges were never reported to the state, according to state Attorney General Dennis Vacco's office.

"As we speak, we are not pursuing any criminal charges for any past releases, but they may be potentially pursued in the future," said Vacco spokesman Marc Carey.

The order by federal Bankruptcy Court Judge Robert E. Littlefield came after Vacco stepped into the case involving the owners of Central Warehouse, an 11-story structure at the corner of Colonie and Montgomery streets.

Vacco was concerned that the 69-year-old system could leak ammonia after C.W. Associates vacated the building as planned on Thursday, said Carey.

That would have left no one to tend the building's deteriorating refrigeration system, which functions on about 10,000 pounds of ammonia under pressure. Electricity to the system also would have stopped, said Carey.

"We need to act quickly to ensure that this dangerous gas isn't allowed to escape and potentially form a cloud over Albany," said Vacco in a prepared statement.

But the lawyer for C.W. Associates said there was never a risk of having the power turned off at the warehouse. "It's irrational," said Richard Weisz. "I don't know who thought that someone there was going to press a button and turn off the power."

Weisz said the company has prepaid power bills with the Niagara Mohawk Power Corp. "We were ahead of the game," said Weisz.

He said a federal bankruptcy trustee had moved in Bankruptcy Court to take control of the building. "We were willing to keep operating it. It was not our idea," said Weisz.

State officials had inspected the warehouse at Vacco's request earlier this month and found that there was a leak risk if the refrigeration system was abandoned and the power turned off.

"The majority of the piping is extremely old and shows visible signs of corrosion," according to an affidavit filed March 11 by Marcia Ellis, an environmental engineer for the state Department of Environmental Conservation.

The system was built in 1927 and modified in the 1960s, she wrote.

And, according to affidavits from the Ellis and Vacco offices, ammonia has gotten out before, although the state was not informed about it.

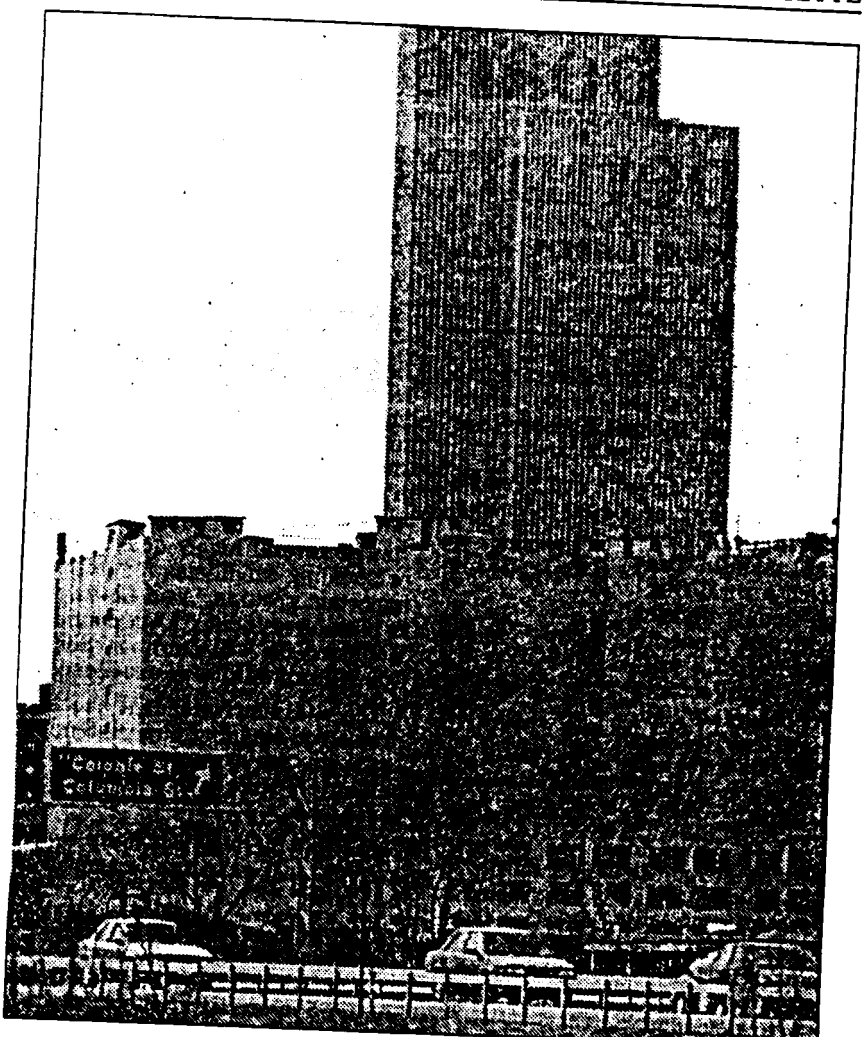
"There have been numerous releases of ammonia in the past, apparently due to the age of the antiquated refrigeration system," according to an affidavit by Maureen F. Leary, an assistant attorney general with the Environmental Protection Bureau within the attorney general's office.

None of those leaks were reported to DEC as required by law, wrote Leary.

Ellis learned about the previous ammonia leaks "based on conversations with employees . . . that there had been small leaks, described as minor," said Gary Sheffer, a DEC spokesman.

After hearing the concerns raised by Vacco, the federal bankruptcy judge ruled that C.W. Associates had

See AMMONIA, Page B4



BRUCE SQUIERS *Gazette Photographer*

A judge ordered ammonia from the refrigeration system in the Central Warehouse in Albany drained on Thursday after officials feared a potential toxic leak.

Ammonia to be drained from Albany warehouse

Continued from Page B1

to keep the electricity on and get offers from companies to drain the ammonia from the system, said Carey.

Ammonia is a colorless, highly irritating gas with a sharp, suffocating smell. It can cause irritation of the eyes, skin, respiratory tract and skin.

In high concentrations, it can cause temporary blindness and even death. It is also flammable.

"We have been involved with this from the beginning . . . that there was the potential for some problems," said Albany Mayor Jerry Jennings. "Everyone agreed . . . that they weren't going to just shut off the juice."

C.W. Associates filed for bankruptcy protection last September, listing $6.3 million in debts on its court petition. It did not list assets.

The company claimed that revenues had dropped rapidly during the past five years with the loss of big contracts with the government, General Electric Co., Kraft, General Foods and Grand Union.

7-1-94 OH
016
COURIER-CRESCENT
ORRVILLE, OH
WEEKLY 2,558
OH454
JUL 7 1994
BURRELLE'S
262 RO

7/1/94

Six injured in Smucker's leak

3082

Five Orrville fire fighters and an employee of the J.M. Smucker Co. were injured last week while attempting to contain an anhydrous ammonia leak at the Smucker cold storage building.

The injuries were not serious and five of the six were treated and released at Dunlap Memorial Hospital.

The leak was caused when a pipe burst accidently last Friday night according to Orrville Fire Chief Robert C. Ballentine. He explained that there was no danger to the community because the leak was contained to a single buliding

Jennifer Nichols, spokesperson for the Ohio Enviromental Protection Agency, confirmed Ballentine's statement and said "The ammonia only could have been lethal if mixed with another substance such as bleach." She added that in a pure gas form as was the case at Smuckers, it only causes irritation to the respiratory system.

Nichols also said that Smuckers has no record of EPA violations in the past.

The leak was reported to the Orrville Fire Department by plant employees last Friday night at 11:16 p.m. The department dispatched four firefighters to the scene with the department's support vehicle carrying spare air bottles.

The team surveyed the leak to determine its magnitude and discovered it was more serious than originally suspected. A general alarm was turned in and at 11:50 p.m. another entry crew was sent into the building along with Smucker staff to attempt to close the leak. They were unsuccessful in this attempt and four firefighters sustained minor burns due to exposure to the

See LEAK, page 7

04/26/96 14:57 ☎ NFPA QUINCY 003

TYPE INC NUMBER EXP. NO YEAR

9 OWNER, OCCUPANT, BUSINESS

39 STATE 41 TYPE SIT. 43 F. D. CODE 48 DATE 54 TIME 58 REPORT STATUS

62 TIME IGN-DET 63 METHOD DETECT 64 AUTO DET TYPE 66 COVRGE 67 PERF. 68 TIME DET-ALARM 69 METHOD ALARM 70 ALARM DELAY 71 TIME ALRM-ARR 72 TIME ARR.-APL. 73 DELAY ARRIVAL 74 DELAY APPL. 75 FIRE DEV 76 OPERATIONAL DATA

77 FIXED PROP USE 80 MOB PROP TYPE 82 COMPLEX 84 STRUC TYPE 85 CONST. TYPE 86 CONST. METHOD 87 HEIGHT 89 G. F. AREA 90 OPERATING STATUS

91 CIV. KILLED 94 CIV. INJURED 97 F. F. KILLED 100 F. F. INJ

RECORD TYPE 1 2 9 LEVEL OF ORIGIN AREA 12

FIRE SPREAD 24 TYPE MAT. 26 FORM MAT. 28 ACTION RESP. FA 30

SUPPRESSION SYSTEM 40 TYPE 42 ACT. 43 EFF./PERF. 45 COV.

SUPPRESSION SYSTEM 54 TYPE 56 ACT. 57 EFF./PERF. 59 COV. N

SUPPRESSION SYSTEM 68 TYPE 70 ACT. 71 EFF./PERF. 73 COV. N

SMOKE/FIRE CONTROL 75 TYPE 77 ACT. 78 EFF./PERF. 79 TYP

SMOKE/FIRE CONTROL 87 TYPE 89 ACT. 90 EFF./PERF. 91 TYPE

OCCUPANTS 95 TOTAL 98 NO. ESCAPED 101 NO. RES

RECORD TYPE 1 3 9 KEYWORD 13

VALUE 25 STRUCTURE 33

ESTIMATED DAMAGE 49 STRUCTURE 57

INSURED DAMAGE 73 STRUCTURE 81

97 INDIRECT LOSS 105 LARGE LOSS

FIRE CONTROL WATER SMOKE/HEAT FLAME

WELL D... Residential
...H FORD Dealership
Crown Victoria F150-250-350 Tempo Wagon

...five acres ... construction of a ... and this area was of interest ... the library board. He explained after the meeting that a swift decision on the project will be greatly appreciated so the library can begin to move forward with their plans. "We've got to do something, folks, we're breaking at the

(7/1/94 continued)

(Continued From Page 1)

ammonia.

At 12:04 a.m. Saturday, the Hazardous Materials Response Unit from Madison Township in Richland County was summoned to the scene. The building was evacuated and no further entry attempts were made pending the arrival of the Madison unit, which made it to the scene at 1:38 a.m.

At 1 a.m. the Wooster Township Fire Department was contacted to dispatch their air support unit to provide additional breathing air for self contained breathing apparatus.

When the Madison department arrived, a decision was made to again attempt to close the leak. At 1:43 a.m. the Wayne County Hazardous Materials Decontamination Unit was activated to assist with this effort.

At 1:49 a.m., communications were established with the Ohio Environmental Protection Agency and the Director of the Wayne County Emergency Management Agency, Ralph Linsalata, because of the possibility of a significant release of ammonia into the air.

At 2:48 a.m., the first of two attempts were made by Madison Township personnel to close the leak. This attempt was unsuccessful and during decontamination from this entry, a fifth Orrville Firefighter sustained minor injuries from chemical irritation.

At 5 a.m., the Madison Township entry team sealed the leak and at 5:15 a.m. a final entry was made to determine the success of the seal which was found to have held the leak. The building was then ventilated by the fire department which was completed at 5:50 a.m.

At 6:46 a.m. the scene was declared secure and safe for re-entry by Smucker staff.

004
NFPA QUINCY
04/26/96 14:58

David C. Coates / The Detroit News

About 120 firefighters battled the four-alarm factory fire at Moenart and Outer Drive, on Detroit's northeast side.

Chemical fire forces 50 families to evacuate

9/12/94

No one injured but toxic fumes from factory blaze on Detroit's northeast side posed a danger.

By Kimberly Thomas 9-12-94
The Detroit News

A Detroit factory fire, fueled by exploding chemicals, raced into a four-alarm blaze this morning that was battled by more than 120 firefighters with 35 pieces of equipment.

No one was injured in the fire that was first reported at about 12:10 a.m. in a multi-story factory building, containing a half dozen private businesses at Moenart and Outer Drive on the city's northeast side.

Residents in 40 to 50 homes on Moenart, Keystone and Conley were evacuated for several hours before being allowed to return home.

"In the early stages of the fire we had isopropyl alcohol, ammonia and propane causing explosions," said Rodney Parnell, a spokesman for the Detroit Fire Department.

"The explosions didn't endanger the dwellings, but the substances were fueling the fire."

Parnell said there also were Styrofoam products and materials to make steering wheels in one section of the factory that could give off toxic fumes.

"We evacuated the residents just as a precaution, but the winds were carrying any fumes up and away from the homes," Parnell said.

An added danger to firefighters were tanks containing 300 pounds of propane, which they feared might cause a larger explosion in the 500 feet by 200 feet factory.

"Propane is always a danger, even an ounce," said Detroit Fire Chief Archie L. Warde.

Blake Arnold, 20, of Grosse Pointe Woods, witnessed the fire start and called the fire department.

"The center portion of the building was fully involved and quickly spread to other portions of the factory," Arnold said.

Parnell said the cause of the fire would not be known until investigators were able to get inside the factory.

5 10 93

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TELEGRAPH HERALD

DUBUQUE, IA
DAILY 34,400
30724
TUESDAY
MAY 11 1993
BURRELLE'S
940 OF

5/10/95

Compiled from TH wire services

7 firefighters hurt in ammonia leak

30332

MIDDLESWORTH — An ammonia leak from a farm chemical plant injured at least seven firefighters and forced the evacuation of about 40 to 50 people, authorities said today.

A cloud of anhydrous ammonia leaked from a faulty shutoff valve at an FS Inc. plant in this tiny central Illinois town about four miles east of Shelbyville late Monday night, Shelby County Sheriff Randall Sims said.

Fire Chief Rick Musser said most residents were allowed back into their homes about 4 a.m.

Shelbyville Memorial Hospital nursing supervisor Jeane Caughran said two firefighters were admitted in fair condition and one was in intensive care with burns. The other four firefighters were treated and released.

04/26/96 15:02 NEPA QUINCY 006
TUESDAY
SEP 17 1991
BURRELLE'S
9/16/91

ship burns

BARRY SWEET / The Associated Press

The Omnisea owned by Dutch Harbor Seafoods burns at Seattle's Pier 91 Monday. The five-alarm fire aboard the 300-foot fish processing ship sent a thick cloud of smoke over the city. Two firefighters were injured.

Alaska processor burns in Seattle

SEATTLE — More than 130 firefighters battled a five-alarm blaze Monday aboard an Alaska-based fish-processing ship, eventually pumping carbon dioxide into the hold to smother a fire that sent a thick cloud of smoke over the city.

Two firefighters were injured, a Seattle Fire Department spokeswoman said.

Fire officials evacuated Pier 91 on Seattle's waterfront at one point, fearing an explosion of an ammonia tank on board the 300-foot Omnisea, fire spokeswoman Georgia Dean said.

A crane removed one tank of ammonia from the vessel but another believed to contain about 6,000 pounds of ammonia was still below deck, she said.

"It's intensely hot inside," Dean said Monday night. "We feel we have it somewhat under control but things could change depending on what happens to the tank and the wind."

The Omnisea is owned by Dutch Harbor Seafoods and operated by Unisea Inc., both of Redmond, Wash. It is homeported in Juneau.

The 46-year-old Omnisea was worth about $8 million, Unisea said.

"It's very important to our process activity," Unisea president J. Richard Pace told The Seattle Times. "As many as 30 boats depend upon it for their market and livelihood. Even if it's able to be repaired we could lose six months or more."

Dean said efforts to extinguish the fire could continue into today.

Fire officials tried to seal hatches with large steel plates lowered to the ship's deck by a crane before flooding the hold with 47,000 pounds of carbon dioxide, television reports said.

Smothering the fire was considered safer than using hosing down the flames because the ship could sink if too much water was poured into it, Dean said. The U.S. Coast Guard monitored the ship's stability.

One firefighter injured a knee and another reported a rapid heartbeat.

The cause of the fire was not known, Dean said, although several crew members said they understood a welding operation touched off the blaze.

The Omnisea usually carries about 100 crew members. Thirty were on board when fire broke out at 8:4 a.m. ADT. None was reported injured, Dean said.

But crew members worried about losing personal belongings and their jobs.

"They only have two ships," crewman Mike Rooker said. "They have the Omnisea and Galaxy, and the Galaxy is up north and its already full crewed. We might be out of work for a while."

04/26/96 15:06 NFPA QUINCY 007
AUG 4 1991
BURRELLE'S

Blaze injures 5 fire

8/3/91

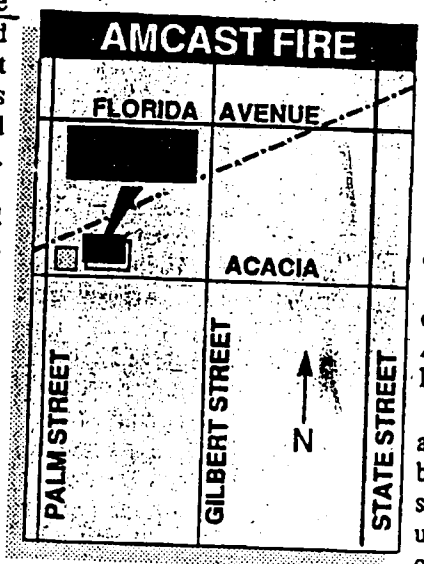
Plant employee battles flames with extinguisher

3082 FRONT PAGE

by A. LEE GRAHAM
Hemet News staff writer

A propane leak most likely caused a chemical fire Saturday which injured five firefighters and damaged a Hemet aerospace parts factory, said Hemet Fire Capt. Jim Snodgrass.

The fire burned three wax storage tanks outside Amcast Aerospace Products, Hemet division on 760 West Acacia Avenue. The fire started at about 12:04 p.m. It lasted only a half-hour, though flames later ignited twice as it burned itself out.



BILL BAKER/THE HEMET NEWS

A small amount of ammonia gas escaped from a tank later that afternoon. It is not known how much of the chemical leaked from the canister, but Snodgrass said area residents are in no danger because of the fire. He said the ammonia dissipated into the atmosphere.

The wax and ammonia tanks were stored outside in an enclosure at the northwest corner of the building. The ammonia tank stood only 20 feet from one of the wax tanks.

An Amcast employee attempted to fight the blaze with a fire extinguisher when it started. A plant security guard called the Hemet Fire Department for help shortly thereafter.

Members of the San Jacinto Fire Department; Little Lake, Ryan Field, San Jacinto and Valle Vista county fire stations; Riverside County Hazardous Materials Team (HazMat); Riverside County Health Department; and Hemet Police Department responded to the call.

"I was working inside the building when another employee said 'the wax tanks are on fire,'" said Amcast employee Luis Panduro.

"I'm glad the ammonia didn't burn," Panduro said, although an unknown amount of the chemical later leaked from the canister.

Amcast employee Barb Johnson said she was working upstairs in the building when someone called from the other building, about 100 yards west of the fire, and told them to evacuate the structure. She said everyone ran from the building, not realizing there was a fire.

William Pshebelski, the security guard on duty when the fire occurred, said he did not hear the explosion, but said he saw the flames and called the Hemet Fire Department.

Another employee said he did not know how much wax was in the tanks, but said the capacity was 2,300 pounds.

Gil Salazar had just completed his shift when the fire occurred. He remained at the scene, helping police

(See BLAZE, Page A-3)

A Hemet firefighter douses a rush of flames at at Palm and Acacia avenues.

Plumes of smoke billow from a chemical fire Saturday at Amcast Aerospace Products Hemet division on 760 W Acacia Avenue. The blaze burned three vats of hot wax and released a small amount of ammonia gas into the The fire was contained in about a half-hour.

Hemet News staff p

BLAZE: Leak targeted as possible caus

A (8/31/91 contined)

(Continued from Page A-1)

make sure everyone was out of the building. He said the fire started with the wax tanks.

County firefighters Robert Puddy and Greg Eakens of the Ryan Field station, Hemet firefighter Harlan Hancock, Hemet fire engineer Art Deyo and Hemet Capt. Jim Snodgrass were taken to Hemet Valley Medical Center, where they were treated and released for ammonia exposure.

All fire personnel returned to duty later the same afternoon, but Hancock was ordered home because some of the chemical had penetrated his eyes. He was asked to consult a physician Monday, Snodgrass said.

"We're positive the propane ignited and probably caused it," Snodgrass said. "We're not sure if the ignition source was the pilot light or what."

Snodgrass described propane as a heavy chemical which lays low to the ground. "It burns until it finds the ignition source, then it burns back to the tank."

Without modern fire-fighting technology, the fire would not have been so easily contained, said Hemet firefighter Gary Lane.

"It would have been really hard without the foam we used," Lane said. "In most fires, water used against it sinks to the bottom.

"Foam mixes with the water so it floats on top of the flammable liquid (ammonia). It has a cooling effect and a smothering effect at the same time. It covers the flammable liquid until it can't breathe."

Hemet firefighter Ken Harrison agreed.

"It was a hard fire to put out," Harrison said. "When you have a hot liquid like that, it expands when water hits it. I'm glad we had the foam."

Despite technological advantages, the exposure of some metals to chemicals and heat complicated the job.

"We have a lot of molten metals in there," Snodgrass said. "If you hit them directly with water, they tend to explode."

The fire caused mostly smoke damage inside the building, but destroyed the wax vats outside. Though firefighters extinguished the blaze shortly after 12:30 p.m., the most time-consuming task was cleanup.

Members of the Rive County Hazardous Materials assisted in cleanup proce Members of the Riverside C Health Department desc which chemicals the firefi were dealing with.

Cleanup crews completed job late Saturday afternoon Amcast still has a lot of work

"It's a hard fire to clean Snodgrass said. "There were inches of water inside the bui and a lot of equipment dam Wax was everywhere. There's a lot for (Amcast employee do."

An estimated amount of da should be calculated by Mo Snodgrass said.

TIES: (Please provide information on each individual casualty. Check the appropriate box to i
or an INJURY and if he was a CIVILIAN or a FIRE FIGHTER. List FATALITIES first. If
information on separate sheet.)

Injury	Civilian	✓ Fire Fighter	Age	Sex	Causes of Death/Injury
34	34		V	BOTH	AMMONIA GAS IN

PERTY DAMAGE (DOLLARS)

	Value of contents
loss	Estimated contents loss

REMARKS

ie narrative details that will contribute to an understanding of factors responsibl
ketches of the incident scene.

S WAS AN AMMONIA
VERTENTLY CUTTING INTO
FOUR PERSONS WERE TR
STY PERSONS WERE TR
ERING (?) FROM VARIOUS
NAGATION.

43095

USA Today

6-30-95

6/29/95

ARKANSAS

FAYETTEVILLE — An employee cutting through some pipe at Campbell Soup apparently cut into a pipe carrying ammonia, forcing the evacuation of more than 500 workers, officials said. Hospitals treated 39 workers. 13 were held overnight for observation.

NEWS OF THE WEEK

DEC 27 1994

12/13/94

FERTILIZER UNIT EXPLOSION

Massive blast at Terra plant kills four

An early morning explosion Dec. 13 killed four employees and injured 18 at Terra Industries' Port Neal, Iowa, nitrogen fertilizer plant, 16 miles south of Sioux City near the Missouri River.

The blast leveled half the facility and forced evacuation of more than 2,500 people from nearby towns because of an ammonia cloud released from a ruptured storage tank. Road and air traffic were diverted.

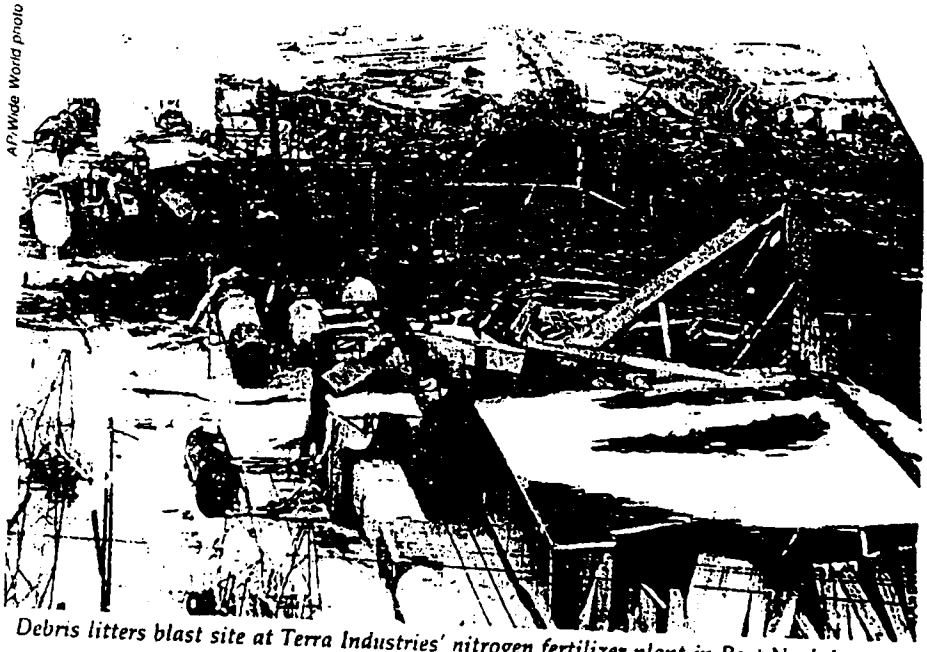
Of the injured workers, 11 were treated and released the same day. Two were still in "very serious condition" at press time. Some were injured by falling debris and others by the impact of the explosion. Many suffered from ammonia inhalation. The plant employs 119 people in three shifts, but only 30 were there when the explosion occurred.

"We are most concerned about our employees and their families; our initial efforts are directed to helping them," says Terra president and chief executive officer Burton M. Joyce. "We are cooperating with local emergency teams to contain the situation so that we can begin a thorough investigation."

Fertilizer prices are expected to be affected. Terra's plant produced about 12% of the U.S. nitrogen-based liquid solution fertilizer market, according to investment analyst Paul K. Raman of New York City-based S. G. Warburg.

"There will definitely be a tightening of [liquid solution fertilizer] supply in the western Corn Belt," Joyce adds. The incident should not affect ammonia markets and prices, since little of the plant's ammonia was sold.

AP/Wide World photo



Debris litters blast site at Terra Industries' nitrogen fertilizer plant in Port Neal, Iowa.

The site "looks like a bomb or a tornado went off," Joyce says. "About one half of the plant is totally destroyed, the other half of the plant is damaged." The explosion was heard 50 to 60 miles away and shook Sioux City. The plant will be down for six to 12 months during reconstruction, he notes.

"We had absolutely no warning," Joyce adds. And the cause is unknown. "It will probably be some time before we do know. There's even a possibility that because of the extent of the damage, we might never know exactly." Terra, the Environmental Protection Agency, the Occupational Safety & Health Administration (OSHA), and the Iowa fire marshal's office are investigating the causes.

Terra says the explosion probably occurred in the ammonium nitrate production area. Shrapnel from the blast ripped open a 15,000-ton anhydrous ammonia storage tank, letting loose an ammonia plume that could be smelled 30 miles away. A 2-mph wind kept the ammonia from dissipating quickly. The metal fragments also punctured a nitric acid tank, spilling up to 100 tons of 56% nitric acid.

The ammonia tank, initially about a third full, stopped leaking when the liquid level dropped below the puncture. Terra says it closed off drainage to prevent groundwater contamination. The ammonia and nitric acid were in secondary containment units at press time.

Off-plant damage included blown-out windows and doors torn off their hinges. There was also structural damage at a nearby power plant. A Terra spokeswoman says no off-site injuries were reported.

Emergency crews responded from Iowa, Nebraska, and South Dakota. A State Emergency Operations Center was activated. EPA, OSHA, the Coast Guard, and the Department of Transportation were among the many federal, state, local, and private agencies participating in the response.

Terra's plant will take up to a year to rebuild, Joyce says, too late for the spring planting season. "The ammonia plant—the core of the plant—is still standing [although] there are some pipes that appear to be damaged, some hoses." However, he adds, "the nitric acid plant was totally destroyed. The ammo-

No C&EN on Dec. 26

Chemical & Engineering News will not publish an issue on Dec. 26, 1994. The editors wish all our readers a very happy holiday season. Our next issue will be Jan. 2, 1995.

(12/13/94 continued)

nium nitrate plant was totally destroyed. Several of the warehouses were totally destroyed." Complete site assessment is expected to take up to a month.

Joyce says the company's insurance will cover workers' compensation, property replacement costs, third-party liability, and lost profits from business interruption. "We are expecting [to put aside] reserves against this that could be $5 million" to pay insurance deductibles and items not covered by insurance, he notes.

The Port Neal plant, built in 1967, accounted for 15% of the company's nitrogen fertilizer production (including capacity from the Oct. 20 acquisition of Agricultural Minerals & Chemicals). The plant contributed $11 million, about 25% of Terra's operating income for the first nine months of 1994. It had capacity of 350,000 tons per year for anhydrous ammonia. Of that, 85% was upgraded into other products, including 500,000 tons of nitrogen solutions for fertilizer, 100,000 tons of urea, and about 60,000 tons of ammonium nitrate.

Elisabeth Kirschner

Designed protein represses leukemia gene

A designed protein containing "zinc fingers" that bind specific DNA sequences in a leukemia gene has been found to stop uncontrolled cell growth in mouse cells.

This is the first time a DNA-binding protein has been engineered de novo to inhibit gene expression, suggesting the potential for development of a new class of therapeutic medicines.

The work was carried out by Yen Choo, Isidro Sánchez-Garcia, and Aaron Klug of the Medical Research Council Laboratory of Molecular Biology, Cambridge, England. It is described in last week's *Nature* [372, 642 (1994)].

James A. Wells of Genentech, whose research interests include zinc-finger proteins, says Klug and coworkers have shown that "a designed repressor can actually regulate a gene of interest. That's an important next step to take in demonstrating that designed zinc fingers could be useful in gene therapy."

A zinc finger is a DNA-binding domain in a protein. The domain's folded structure is stabilized by a zinc ion coordinated to four amino acid residues. Klug first proposed the zinc-finger concept in 1985. Since then, researchers have shown that many eukaryotic DNA-binding proteins contain zinc-finger structures.

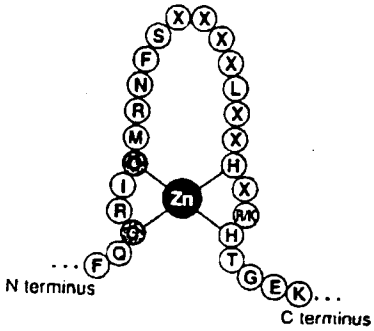
Earlier this year, Klug and coworkers showed that zinc fingers can discriminate between closely related DNA triplets (three-nucleotide sequences). They also proposed that zinc fingers could be linked together to form structures that could recognize longer DNA sequences.

Now, they have created a protein with three zinc-finger domains—each designed to bind to one DNA triplet in a nine-nucleotide sequence in the oncogene responsible for acute lymphoblastic leukemia. The protein binds specifically to the sequence and inhibits expression of the oncogene.

The protein was created by combinatorial and rational design techniques. Klug and coworkers identified an initial set of zinc fingers that bound the oncogene sequence by isolating them from phage-display libraries of randomized zinc fingers. They then refined the structures by rational design, based on known recognition rules for zinc-finger binding.

Use of a DNA-binding protein with zinc fingers to target gene sequences is similar conceptually to antisense and triple-helix-binding strategies, in which RNA- and DNA-binding oligonucleotides are used to inhibit gene expression.

Zinc finger is stabilized by zinc coordination



Consensus sequence for three zinc finger domains in protein designed by Klug and coworkers

C = cysteine, E = glutamic acid, F = phenylalanine, G = glycine, H = histidine, I = isoleucine, K = lysine, L = leucine, M = methionine, N = asparagine, Q = glutamine, R = arginine, R/K = arginine or lysine, S = serine, T = threonine, X = variable residue

But Klug and coworkers point out that zinc-finger proteins can also enhance gene expression by binding to gene-activation domains—a role that oligonucleotide agents cannot play. Indeed, in their *Nature* paper, they report another experiment in which the oncogene DNA sequence is used as a promoter for expression of another gene. They show that the designed protein, linked to a domain that activates transcription, successfully switches on the gene.

According to Klug, "The experiments so far prove the principle that one can design a novel protein for recognizing a given sequence of DNA, in this case an oncogene. However, we still need to refine and develop methods of getting proteins into cells. Ultimately, we may be able to insert a gene into cells to enable them to manufacture their own blocking proteins, and this is one of the areas we'll be looking into now."

Stu Borman

Tenneco plans to sell Albright & Wilson unit

Tenneco plans to sell its U.K.-based chemical subsidiary, Albright & Wilson, through a public offering. The sale will leave Tenneco with no chemical operations.

The Houston-based firm expects to sell 100% of A&W during the first quarter of 1995, with the shares offered only in the U.K. and listed on the London Stock Exchange. The total number of shares to be offered and tentative offering price per share have yet to be set.

The move continues Tenneco's shedding of units to focus on its core businesses of packaging, automotive parts, and natural gas pipelines. "A&W's conversion to a separate publicly traded company will substantially increase the resources and flexibility we are generating to grow our core businesses," notes Dana G. Mead, chairman and chief executive officer of Tenneco and chairman of A&W. Tenneco already has sold the public 55% of its Case Corp. farm & construction equipment subsidiary.

As an autonomous company, A&W likely will rank as the fourth largest independent chemical maker in the U.K. in terms of sales, says Jeremy Chantry, U.K. chemicals analyst at Kleinwort Benson in London. He notes that Tenneco tried, and failed, to sell A&W previous-

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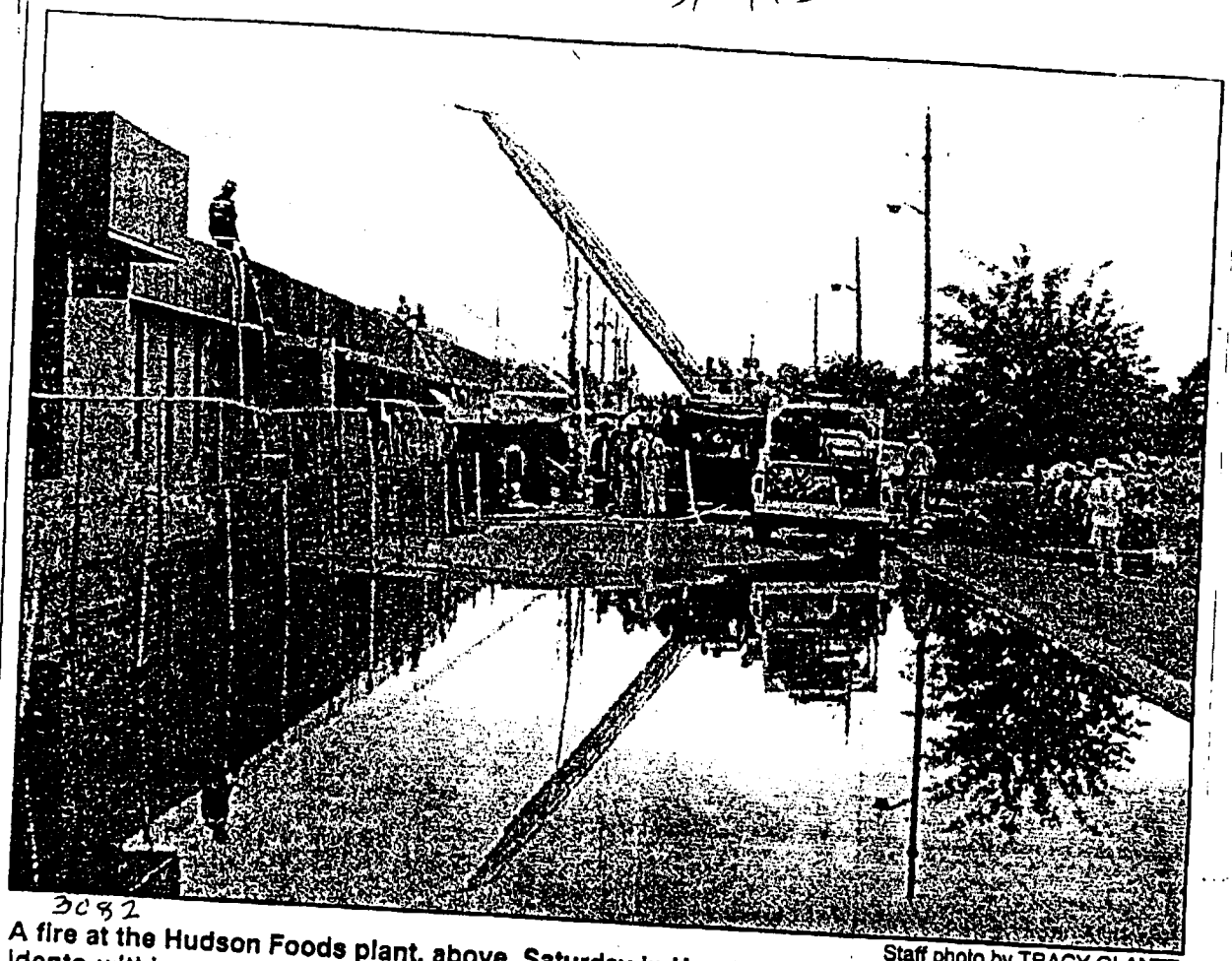


3082

A fire at the Hudson Foods plant, above, Saturday in Hope, Ark., forced the evacuation of residents within a 5-mile radius. Firefighters put out the the fire about midnight Saturday and residents were allowed to return home Sunday.

Staff photo by TRACY GLANTZ

Blaze sends two firefighters to hospital

FRONT PAGE

By RUSSELL MINOR
Of the Gazette Staff

HOPE, Ark.—Exploding pipes leading to chlorine and ammonia tanks in the Hudson Foods processing plant may have caused the symptoms that sent two firefighters battling the blaze to the hospital, one of the men said.

About 9:25 p.m. Saturday firefighters Howard Tippit and Steve Tarpley of the Hope Fire Department were taken to the Medical Park Hospital in Hope from the Hudson plant, which burned from 2:30 p.m until midnight.

Both men were released after treatment.

"I'm still having headaches and my throat hurts," Tippit said Sunday evening. "I'm real sore and I have been coughing quite a bit."

At the hospital, Tippit said he and Tarpley were given chest X-rays to assess the extent of their smoke inhalation.

He said his X-ray came back relatively clean because he vomited at the scene.

"I remember I turned around and grabbed the chain-link fence and got sick big time," he said. "Then I went down to my knees."

Tippit said the sickness he feels is nothing serious and he will be back to work soon.

He and Tarpley were not at the plant when the fire was finally extinguished at midnight, but he said the plant was damaged extensively. Officials also evacuated homes within a 5-mile radius of the plant until the fire was out.

"At one point, I went into the back part of the plant and knocked a part of a wall down with another guy," he said. "We went into the main part of the plant. It was destroyed real bad."

Tippit believes exploding pipes leading to the ammonia and chlorine tanks may have made him sick.

"The tanks were sealed off, but the pipes were popping," he said.

Damage to the plant may reach several million dollars, company officials said. Employees of the temporarily closed plant will continue to receive paychecks.

Blaze guts potato plant i

FRONT PAGE

Fire displaces 90 workers

By Aimee Green
Bulletin Staff Writer 3082

7/6/95

METOLIUS — A raging fire at a Metolius potato-processing plant caused more than $1 million in damage Thursday and put 90 employees out of work.

Thick gray smoke from the blaze, apparently touched off by workers using welding torches on a roof-repair project, billowed high into the sky. The smoke could be seen for 25 miles, attracting onlookers from Madras to the north and Culver to the south.

"Once we realized the fire started, we got out our fire hoses," said maintenance supervisor Tom Schott. "We had it virtually out at one point," he said, but the flames slipped into the attic and quickly spread into the main part of the 50-year-old wood-frame structure.

Seventy firefighters from 10 departments, including Bend, Redmond, Madras and Prineville, fought the blaze for nine hours before bringing it under control. By then, the heart of the complex, easily the community's largest employer, lay in ruins.

Chemicals and fuels — including propane, chlorine, ammonia, diesel and bottled oxygen — posed extreme danger to firefighters, said Earl Cordes, chief of Jefferson County Fire District 1. They were relieved when a 400-gallon tank of ammonia burned itself out without mishap, he said.

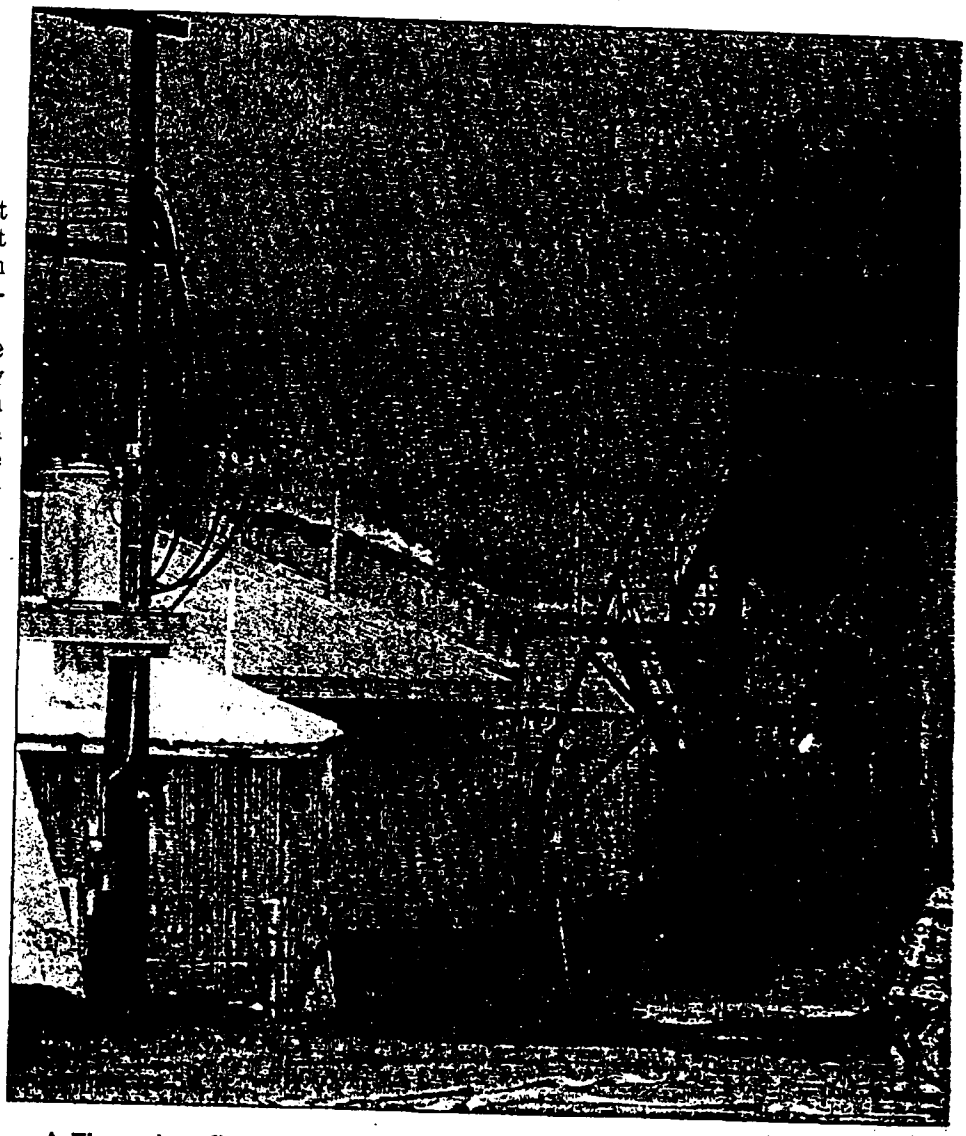
Cordes said it took a heroic effort by firefighting crews to prevent the blaze from reaching the plant's chemical storage room.

"They were literally standing in the door beating back the flames," he said. "This was amid exploding diesel drums and oxygen tanks."

Cordes noted, "It was extremely difficult to access the seed of the fire. With big timbers in the attic area, you're setting up the equation for disaster."

He said firefighters would remain at the scene all day today, making sure the smoldering ruins didn't re-ignite and spread to other buildings nearby.

Due to the danger, roads were blocked in all directions Thursday. However, traffic was moving nor-



A Thursday fire inflicted about $1 million in damage on the Logan

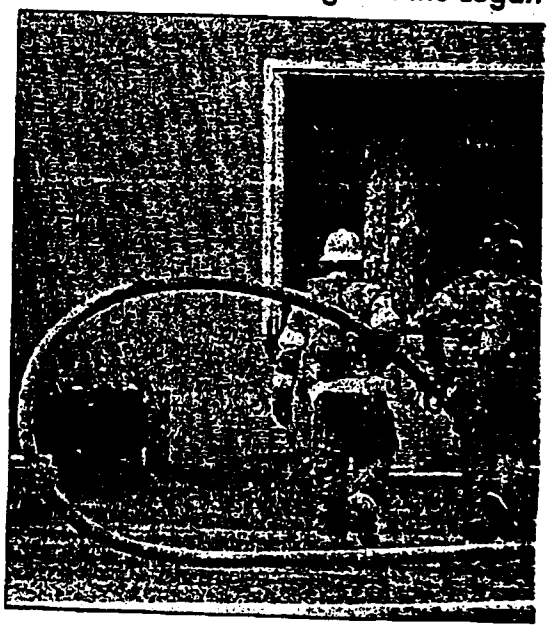
mally again this morning.

The plant, owned by Logan International, produced frozen french fries. It was just gearing up for the July 17 start of the 1995 season.

Four smaller outbuildings escaped the blaze, but the main building where the processing occurred was a near-total loss. Oregon farmers, who typically send the plant about 200 tons of potatoes a day, will need to find another outlet this season.

"The production area of the plant is gone, but we were able to salvage some of the machinery," Cordes said.

Manager Paul Eatinger said the plant, which had recently undergone extensive remodeling, was fully insured and would definitely be rebuilt. He said the timetable had yet to be determined.



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5/30/90

▶ **CHEMICAL LEAK:** A toxic cloud of ammonia and hydrogen chloride forced evacuation of 2,000 residents and 1,850 grade school pupils in Freeport, Texas. Officials said chemicals were mixed accidentally as a ship was loaded.

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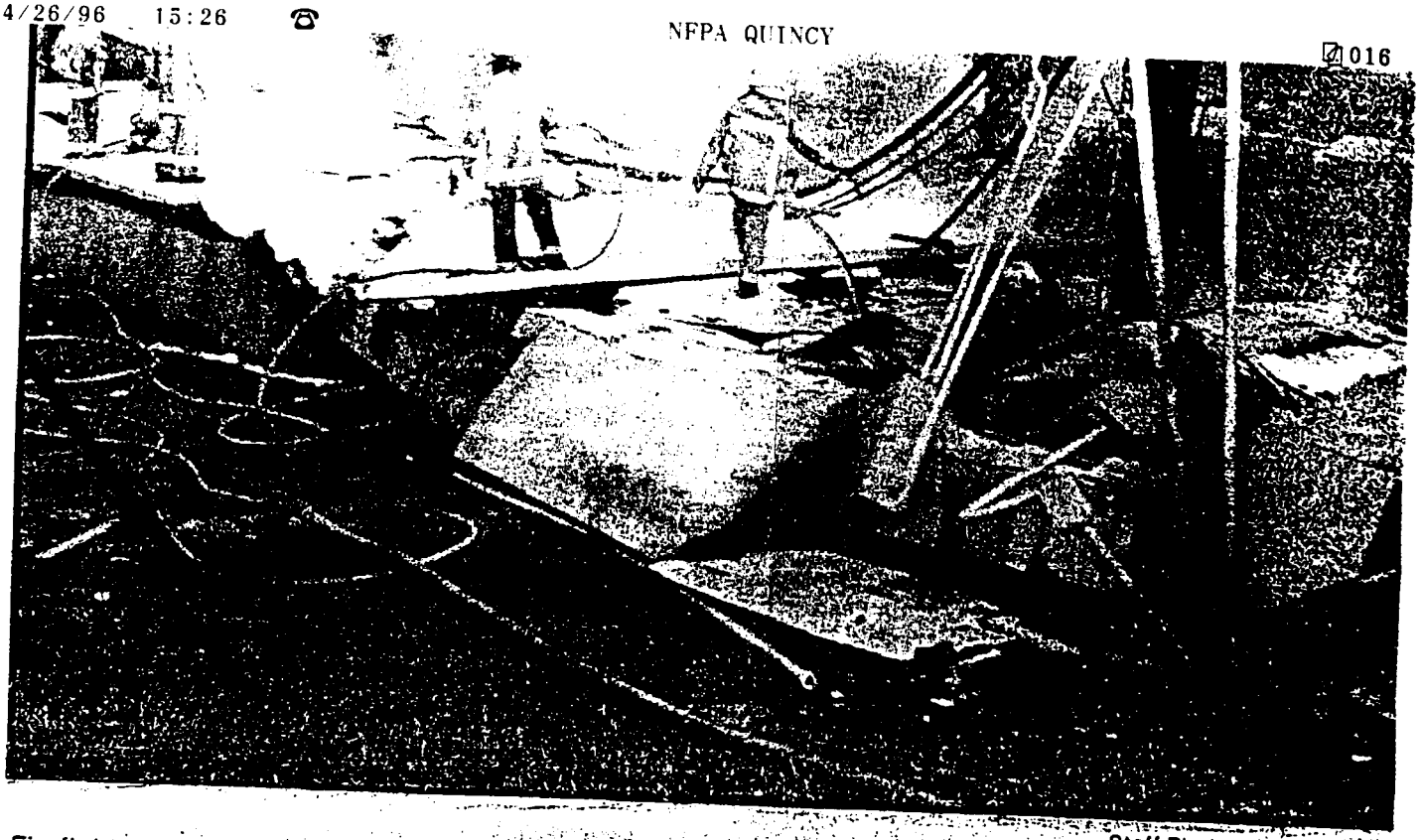
USA Today 8 31 90

8/30/90

OREGON

EUGENE — Bill limiting log exports from private land on parcels larger than 320 acres will be introduced by U.S. Rep. Peter DeFazio. He says it won't affect 90% of state's small tree farmers but will force log exporting firms to invest in USA mills, workers. ... **PORTLAND — Explosion, fire caused by ammonia leak in West Coast Grocery warehouse closed Southeast McLaughlin Blvd. for 4½ hours. No injuries were reported; 50 firefighters fought blaze.**



Firefighters douse the smoldering rubble of the Claremont Ice Company after an early morning fire Friday.

Staff Photos by Walt Weis

Cold storage plant burns

Claremont fire loss total is $1.85 million

By Sharon Greengold
Bulletin Staff Writer

3082 FIRE FRONT PAGE

A three-alarm blaze destroyed the refrigeration unit at the Claremont Cold Storage plant early Friday, causing $1.85 million in damage and releasing an ammonia cloud that dissipated without causing injuries, authorities said.

Some 60 firefighters, using two aerial-hose trucks, controlled the blaze at Claremont Cold Storage, 128 N. Oberlin Ave., by 4 a.m. and had it fully extinguished by noon, said Claremont police Capt. Cal Stephens.

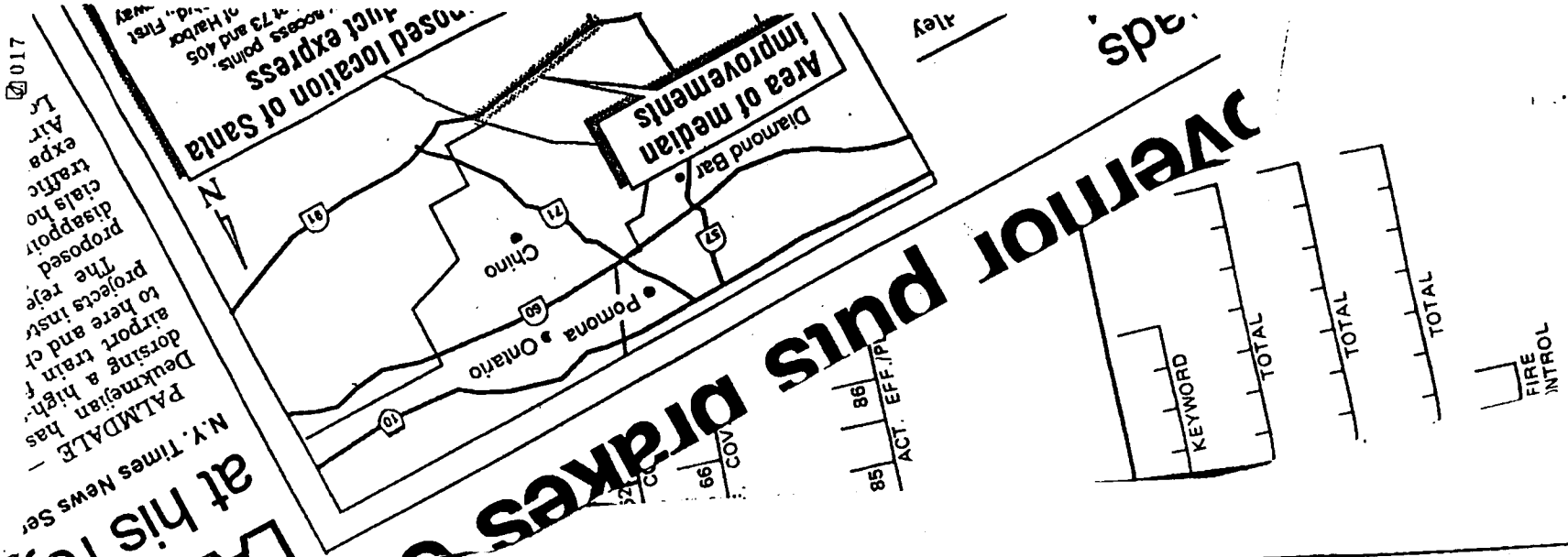
The company is owned by Bill Silzle of San Juan Capistrano. The same building was the site of a small wall fire roughly a year ago.

"That place was built years and years and years ago when sawdust was used as insulation," Stephens said. He said there was no connection between the fire a year ago and the one Friday morning.

See FIRE/Page 2

A Claremont Ice Company sign is all that is left of the ruined building.

9/14/90



Just after 2 a.m., Tim Norman, 24, of Chino, operations engineer at Claremont Cold Storage, discovered a serious ammonia leak and shut down the refrigeration system, according to Lt. Ted Whitall of the Claremont Police Department.

Norman said he called the Claremont Fire Department and reported the leak at 2:27 a.m., however, before the ammonia could dissipate, it reportedly triggered a flash fire in the building's electrical panels.

When firefighters arrived the building was fully engulfed, Whitall said.

"I had just phoned the Fire Department and told them there was no fire!" Norman said.

He said he then knocked on the doors of neighboring houses to notify anybody inside about the fire.

The fire released an ammonia cloud over the plant, but no evacuations were ordered and the cloud dissipated by 3:35 a.m., said Los Angeles County fire dispatcher Ed Connolly.

No other structures were damaged but equipment that pumped cooling fluid to two adjacent storage buildings was destroyed.

Concentrated orange juice and whipped cream stored in those buildings will remain, Norman said.

"With no equipment at all, we're good for three to four days. We'll see how fast we can get portable compressors hooked up," he said.

The Claremont Ice Co., w leases storage space in the l ing that was destroyed, w able to continue supplying the Los Angeles County Norman said.

The ice, he said, is not factured there but is purc by the company and the tributed.

(9/14/90 continued)