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w/RIL #21

HSPH/EHS(NUREG)-880

A CRITICAL REVIEW OF SODIUM HYDROXIDE AEROSOL TOXICITY

Final Report

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Dwight W. Underhill
Michael J. Ellenbecker

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Prepared for the U.S. Nuclear Regulatory Commission
Under Contract No. AT(49-24)-0300

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Dwight W. Underhill
Michael J. Ellenbecker

December 1977

Department of Environmental Health Sciences
Harvard School of Public Health
665 Huntington Avenue
Boston, MA 02115

NOTICE

ABSTRACT

This is a critical analysis and review of the literature available with respect to the chemical toxicity of sodium hydroxide aerosols, especially those which might be formed as the result of a sodium fire. Over one hundred references are listed in the literature review. Analysis of the chemistry of sodium hydroxide aerosols indicates that most would be converted from sodium hydroxide to sodium carbonate decahydrate within minutes in the atmosphere, strong determinants of the rate of transformation being relative humidity and particle size. The U.S. standard for industrial exposure to sodium hydroxide aerosol is 2 mg/m^3 as a ceiling threshold limit value. The documentation of that standard is reviewed and found to be questionable. It is concluded that there is insufficient information on which to base a maximum acceptable concentration for accidental exposures to sodium hydroxide aerosols. The information that is available indicates that sodium carbonate could be tolerated at higher concentrations than sodium hydroxide. The rate of transformation of the hydroxide to the carbonate is analyzed and predicted to be faster than that predicted by others. A preliminary model of aerosol deposition on the eye and the pH of tears is developed to help indicate the relationship between NaOH aerosol concentration, size distribution, duration, and damage to the eye.

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NOMENCLATURE

ae	subscript: aerodynamic equivalent
c	concentration, moles/cm^3, g/cm^3
d	diameter, cm; subscript: droplet, diffusion
e	subscript: eye
g	gravitational acceleration, 981 cm/s^2
k	mass transfer coefficient, mole/cm^2-s
l	subscript: liquid
m	subscript: migration
p	subscript: particle, projected (area)
q	mole fraction
r	radial distance, cm; radius, cm; subscript: reaction
s	subscript: solid, settling
t	time, s
v	velocity, cm/s
w	subscript: water
y	mole fraction
z	square root of ratio of characteristic diffusion time to characteristic reaction time
A	area
C	Cunningham correction for gas slip
D	diffusion coefficient, cm^2/s
H	relative humidity (as a fraction, not a percentage); Henry's law constant
K	equilibrium constant
M	molecular weight, g/mole
$\dot{M}$	mass transfer rate, g/s
R	radius, cm; universal gas constant, 8.31 x 10^7 g-cm^2/g-mole-s^2-°K

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Re	Reynolds number
Sc	Schmidt number
T	absolute temperature, °K
V	volume, cm^3
$\dot{V}$	volume rate of flow, cm^3/s
γ	surface tension, dy/cm
η	efficiency
μ	viscosity, g/cm-s
π	3.14159....
ρ	density, g/cm^3
ψ	impaction parameter

ACKNOWLEDGMENTS

We gratefully acknowledge the editorial help given by several reviewers at the U.S. Nuclear Regulatory Commission, including Dr. M. C. Cullingford. Also appreciated is the considerable assistance of Mr. Eric Rothenberg (now of Bolt, Beranek and Newman) who did most of the literature search on which this report is based.

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I. INTRODUCTION AND SUMMARY

To help evaluate the potential air pollution hazard from a sodium fire, a literature review was performed to determine the chemical toxicity of sodium hydroxide aerosols which would result from such a fire as the sodium oxides reacted with water vapor in the air. Specifically, the toxicity due to inhalation, contact with skin and eyes, and ingestion was to be determined, for normal and susceptible populations, in terms of morbidity and mortality rates where available.

An extensive literature review was undertaken after consulting with specialists in toxicology and in industrial hygiene. Details of this review and a bibliography are given in Section II. Simultaneously, calculations were made and literature reviewed with regard to the assumption that sodium hydroxide was the compound of concern resulting from a sodium fire in the atmosphere. It was found that, indeed, the oxides of sodium from the fire would react with water vapor to form the strongly alkaline hydroxide, but the hydroxide would then react with the atmospheric carbon dioxide to form the less alkaline sodium carbonate (sodium carbonate decahydrate, most probably). Further, at relative humidities greater than 95 per cent and for times much longer than 10 hours, the carbonate could become the still less alkaline bicarbonate. Because toxicity of the aerosol is related directly to its alkalinity, part of the effort was directed to determining the conditions under which such transformations would take place and the times they would take to occur. Information about the chemistry and physics of the NaOH aerosol is contained in Section III and in Appendices A and B, which discuss in more detail factors relating to the rate of transformation and the effect on particle size and aerodynamic diameter of such transformations. From this work it was concluded that usually within a few minutes respirable aerosols of sodium hydroxide would be transformed to the less hazardous carbonate form, so that standards for sodium hydroxide might not be applicable to persons reached by the cloud in minutes rather than seconds after its formation. This transformation would also substantially increase the aerodynamic diameter of the particles.

In Section IV are presented the U.S. standards for inhalation of NaOH aerosols, along with the meager documentation available for those standards. Also discussed is the basis for the Soviet Union's standard. From our communications with several foreign experts, these standards seem to be the basis for similar standards adopted by Western and Eastern Bloc countries, respectively. This section presents summaries of selected relevant papers regarding inhalation, ingestion, and contact with eyes. The selection process omitted many papers on eye injury which did not include <u>both</u> dose <u>and</u> duration of exposure and numerous papers on the purposeful or accidental ingestion of solid or dissolved NaOH (lye), neither category being of real use for the study. Appendix C presents an approximate model for relating pH of tears to aerosol concentration for individuals exposed to NaOH, from which one could make comparisons with the tear pH level believed hazardous (pH = 11).

Section V contains a list of recommendations for further study:

investigation should be made of the effects of exposure of man or animals to aerosols of the hydroxide and aerosols of the carbonate, and the rates of transformation (and the species formed) for NaOH aerosols under various conditions should be estimated and confirmed by experiment. It is recommended that such research be done if a standard for maximum acceptable concentrations of NaOH aerosols during infrequent sodium fire accidents is to be promulgated.

Although much useful information was developed, it was inadequate for determining dose-response relationships for sodium hydroxide aerosols for humans.

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II. LITERATURE REVIEW

In this review was sought all the significant information on the toxicity of sodium hydroxide (NaOH) aerosols, with special emphasis on the documentation underlying existing standards. Also sought was the information needed to predict the chemical and physical behavior of such aerosols, with an emphasis on the products from a fire involving metallic sodium.

Through consultation with members of the staff of the Harvard School of Public Health, many sources of general and specific information on the toxicology of NaOH (and other caustic) aerosols were identified. Listed in the bibliography at the end of this section are the references which we identified from their abstracts or from the references themselves; we were able to designate those which seemed potentially most useful for further evaluation. One of the ways this bibliography was formed was through the references of all the sources listed under "General Toxicology" in the bibliography, including some 1,100 references on many subjects in Hamilton and Hardy (1).

Computer literature searches were performed by the Toxicology Information Response Center (as suggested by the Nuclear Safety Information Center), using their TOXLINE and MEDLINE searches for literature on: sodium hydroxide; sodium carbonate; sodium aerosols and sodium hydroxide; alkali, basic or caustic aerosols.

The following abstracting publications were checked: Air Pollution Abstracts from initial publication to June 1976; Chemical Abstracts (inhalation) from 1945 to June 1976; Index Medicus (inhalation) from 1945 to June 1976; many but not all copies of Industrial Hygiene Digest, Abstracts on Hygiene, Hygiene and Sanitation (USSR), International Archives of Occupational Health (Federal Republic of Germany), and Environmental Health (Netherlands). Further, the collections at the Countway Library (at the Harvard School of Public Health) and at the Massachusetts Division of Occupational Health were carefully searched.

All the journals in Table 1 were searched to their first issues, except the Journal of Experimental Medicine and Archives of Opthalmology, which were checked back to 1920. (There were too few references occurring to warrant search to their initial publication dates.) This table lists the journals in the first column. Successive columns are for five-year periods, with the numbers totalling all relevant articles which were published during the period shown. Relevant articles were those which contributed new information or summarized past work, directly related to biological response to NaOH aerosols. The row totals show how many articles were counted in a particular journal; the column totals show the number found for each five-year period. In general, for a given publication the number of relevant articles published per year seems to have been greater after World War II than before. Furthermore, the number of journals published was greater after World War II than before, so that there is some justification in emphasizing the abstract searches in the period following 1945.

References were also obtained from the NIOSH "Criteria for a Recommended Standard...Occupational Exposure to Sodium Hydroxide."(2) Some of the effort resulted in little additional information. None of the Hygienic Guides of the American Industrial Hygiene Association were appropriate. The American Conference of Government Industrial Hygienists Threshold Limit Values(3) had very limited documentation. Contact and correspondence with health professionals in Canada, England, Finland, and Yugoslavia uncovered no new sources of documentation of health effects.

The publications identified which were even tenuously connected to the determination of the toxicity of sodium hydroxide aerosol are listed in Table 2. Although there is no method to assure that some important information has not been overlooked, the paucity of new information and the recurrence of familiar references as the end of the search was approached suggested that virtually all the relevant references, at least in the English literature (original or translated), were identified.

Although the bibliography which follows contains the results of an extensive and intensive search of the literature, it will be seen in later sections that there remains a lack of information on which to base a decision as to what concentrations or doses of NaOH aerosol cause what degrees of harm to humans.

Table 1. Chronology of Relevant Journal Articles on NaOH, by Half Decade

Journal	1920	1925	1930	1935	1940	1945	1950	1955	1960	1965	1970	1976	Row Total
Am. Indus. Hyg. Assoc. J.				*			1	4	3	1	2		11
Am. J. Ophthal.						2	1	1	1	3	3		11
Annals Occup. Med.								*			1		1
J. Indus. Hlth.	2			4	1	2**							9
Arch. Indus. Hlth. & Occup. Med.							*2**						2
Arch. Indus. Hlth.								*3**					3
Arch. Envir. Hlth.									*4		2		6
Arch. Ophthal.						3	2	1		2	4		12
Hlth. Physics							*	1	1	1			3
J. Appl. Physiol.						*	1				1		2
J. Exper. Med.									2				2
New Eng. J. Med.		*						1		1	1		3
Nucl. Safety										*	2		2
COLUMN TOTAL	2			4	1	7	7	11	11	8	16		67

*Journal began publication

**Journal ended publication

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Table 2.

Some Publications Related to Assessing the Toxicity of Sodium Hydroxide Aerosols

Aerosol Physical and Chemical Behavior

Bird, R.B., Stewart, W.E., and Lightfoot, E.N., Transport Phenomena. John Wiley and Sons, New York (1960).

Clough, W.A. and Garland, J.A., "The Behavior in the Atmosphere of the Aerosol from a Sodium Fire." Report #AERE-R-6460, Atomic Energy Research Establishment, Harwell, England (1970).

Clough, W.S. and Garland, J.A., "The behavior in the atmosphere of the aerosol from a sodium fire", J. Nucl. Energy 25: 425-435 (1971).

First, M.W., "Contamination control of sodium releases from liquid-metal-cooled fast breeder reactors", Nuclear Safety, 13:37-46 (1972).

Fuchs, N.A., The Mechanics of Aerosols. Pergamon Press-The MacMillan Company, New York (1964).

Harned, H.S. and David, H., Jr., "The ionization constant of carbonic acid in water and the solubility of carbon dioxide in water and aqueous salt solutions fron 0-50°", J. Am. Chem. Soc. 65: 2030-2037 (1943).

Linke, W.F., Solubilities of Inorganic and Metal Organic Compounds. American Chemical Society (1958).

Masaki, K., "On the CO_2 absorption velocity of NaOH - and KOH - solutions", J. Biochem. 13: 211-217 (1931).

McCoy, H.N. and Smith, N., Am. Chem. J. 29: 437 (1903).

Murata, M., Naritomi, M., Yoshida, Y. and Kokubu, M., "Behavior of sodium aerosol in atmosphere", J. Nucl. Sci. Technol. 11(2): 65-71 (1974).

Neiburger, M. and Wurtele, M.G., "On the nature and size of particles in haze, fog and stratus of the Los Angeles Region", Chem. Rev. 44: 321-335 (1949).

Perry, R.H. and Chilton, C.H., eds., Chemical Engineer's Handbook, 5th edition. McGraw-Hill, New York (1973).

Tadmor, J., "Consequences of an accidental release of sodium to the Environment from an LMFBR," Nuclear Safety 14: 324-340 (1973).

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General Toxicology

American Conference of Governmental Industrial Hygienists (ACGIH), Documentation of the Threshold Limit Values for Substances in Workroom Air, 3rd edition. ACGIH, Cincinnati, Ohio (1971).

American Conference of Governmental Industrial Hygienists (ACGIH), Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1976. ACGIH, Cincinnati, Ohio (1976).

Casarett, L.J. and Doull, J., Toxicology: The Basic Science of Poisons. MacMillan, New York (1976).

Deichman, W.B. and Gerard, H.W., Toxicology of Drugs and Chemicals. Academic Press, New York (1969).

Elkins, H.B., The Chemistry of Industrial Toxicology, 2nd edition. John Wiley, New York (1959).

Fairhall, L.T., "Inorganic Industrial Hazards", Physiology Rev. 25 (January, 1945): 182-202.

Hamilton, A., Industrial Toxicology. Harper and Brothers, New York (1934).

Hamilton, A. and Hardy, H.L., Industrial Toxicology. Publishing Sciences Group, Acton, MA (1974).

Loomis, T.A., Essentials of Toxicology, 2nd edition. Lea and Febiger, Philadelphia (1974).

National Institute of Occupational Safety and Health, U.S. Dept. of Health, Education and Welfare, The Industrial Environment - Its Evaluation and Control, Chapt. 7, "Industrial Toxicology", by M.O. Amdur. U.S. Government Printing Office, Washington, D.C. (1973).

Palmer, H.E., "Rapid Removal of Sodium Isotopes from the Body Following Accidental Internal Contamination." Report #BNWL-SA 5463, Battelle Pacific Northwest Laboratories, Richland, WA (1975).

Patty, F.A., editor, Industrial Hygiene and Toxicology, 1st edition, Vol. I. Interscience Publishers, New York (1949).

Patty, F. A., editor, Industrial Hygiene and Toxicology, 2nd edition, Vol I. Interscience Publishers, New York (1958).

Patty, F.A., editor, Industrial Hygiene and Toxicology, 2nd edition, Vol. II. Interscience Publishers, New York (1963).

Witthaus, R.A., Manual of Toxicology, 2nd edition. Wm. Wood and Company, New York (1911).

Aerosols - Respirability

Brain, J.D. and Valberg, P.A., "Models of lung retention based on ICRP task group report", Arch. Env. Hlth. 28:1-11 (1974).

Chamberlain, A. C., "Aspects of the deposition of radioactive and other gases and particles", Int. J. Air Pollut. 3:63-88 (1960).

Drinker, P., and Hatch, J., Industrial Dust. 2nd ed., New York. McGraw-Hill (1954).

Giacomelli-Maltoni, G., et al., "Deposition efficiency of monodisperse particles in human respiratory tract", Am. Ind. Hyg. Assn. J. 33:603-610 (1972).

Guyton, H.G., Decker, H.M., and Anton, G.T., "Emergency respiratory protection against radiological and biological aerosols", AMA Arch. Ind. Hlth. 20:91-95 (1959).

Harris, R.L., and Fraser, D.A., "A model for deposition of fibers in the human respiratory system", Am. Ind. Hyg. Assn. J. 37:73-89 (1976).

Hatch, T.F., and Gross, P., Pulmonary Deposition and Retention of Inhaled Aerosols. New York. Academic Press (1964).

Heyder, J., Armbruster, J., Gebhart, E., and Stahlhofen, G.W., "Total deposition of aerosol particles in the human respiratory tract for nose and mouth breathing", J. Aerosol Sci. 6:311-328 (1975).

International Commission on Radiation Protection, Task Group on Lung Dynamics. "Deposition and retention models for internal dosimetry of the human respiratory tract." Health Physics, 12:173-207 (1966).

Lippmann, M. "Respirable dust sampling", Am. Ind. Hyg. Assn. J. 31:138-159 (1970).

Morrow, P.E., "Some physical and physiological factors controlling the fate of inhaled substances", Health Physics, 2:366-378 (1960).

Morrow, P.E., Bates, D.V., Fish, B.R., Hatch, T.F., and Mercer, T.T., "Deposition and retention models for internal dosimetry of the human respiratory tract", Health Physics, 12:173-207 (1966).

Taulbee, D.B., and Yu, C.P., "A theory of aerosol deposition in the human respiratory tract", J. Appl. Physiol., 38:77-85 (1975).

Inhalation Toxicology

Amdur, M.O., and Corn, M., "The irritant potency of zinc ammonium sulfate of different particle sizes", Am. Ind. Hyg. Assn. J. 24: 326-333 (1963).

Elkins, H.B., "Maximum allowable concentrations: a comparison of USSR and USA", Arch. Env. Hlth. 2:45, (1961).

Eskenasy, A., "Experimental bases of chronic lung diseases", Rev. Roum. Morphol. Embryol. 19(2):127-139 (1975).

Fairchild, G.A., Roan, J., McCarrol, J., "Atmospheric pollutants and the pathogenesis of viral respiratory infection", Arch. Env. Hlth. 25:174-182 (1972).

Girault, "The effect of sodium carbonate on packers", Arch. Malodies Profess. (Fr.), 21:747-750 (1960).

Haggard, H.W., "Action of irritant gases on the respiratory tract", J. Ind. Hyg. Toxicology, 5:390-398 (1924).

Hardy, H.L., "Pulmonary irritants (toxic hazards)", New England J. Medicine, 263:813-814 (1969)..

Hervin, R.L., and Cohen, S.R., "Health Hazards Evaluation Determination", Report No. 72-97-135. U.S. Dept. HEW, NIOSH.

Kleinfield, M., "Acute pulmonary edema of chemical origins", Arch. Env. Hlth. 10:942-946 (1955).

Lauria, T.I., "Soda lime dust contamination of breathing circuits", Anesth. 42:628-629 (1975).

Probst, E.W. and Lanahan, F.B., "Sodium bicarbonate: an occupational medical problem", Indus. Medicine, 17:24 (1948).

Punte, C.L., Ownes, E.J., Krackow, E.N., et. al., "Influence of physical activity on the toxicity of aerosols and vapors", Arch. Ind. Hyg. 17:34 (1958).

Ryazanov, V.A., "Sensory physiology as basis for air quality standards: approach of the USSR", Arch. Env. Hlth. 5:480 (1962).

Smith, H.F., "Hygienic standards for daily inhalation", Am. Ind. Hyg. Assn. J. 17:174 (1956).

Smith, H.F., "A toxicologist's view of threshold limits", Am. Ind. Hyg. Assn. J. 23:37 (1962).

Steele, R.H. and Wilhelm, D.W., "The inflammatory reaction in chemical injury: induced vascular permeability and erythema induced by various chemicals", Brit. J. Exper. Path., 47:612-623 (1966).

U.S. Department of Health Education and Welfare, "Criteria for Recommended Standard....Occupational Exposure to Sodium Hydroxide", USDHEW, NIOSH Publication 76-105 (1975).

Vigliani, E.C., Zurlo, N., "Experience of the clinic del Lavoro with MAC's of industrial poisons", Arch. Gewortepathol. Gewerbehyg. (Ger. 13:528-534 (1955).

Vyskocil, J., Juma, J., and Dluhos, M., "The effect of aerosol inhalations of sodium hydroxide on the elimination of quartz dust from the lungs of rats", Scripta Medica, 39(1):25-29 (1966).

Ingestion Toxicology

Bailey, M., "Caustic stricture of mouth and oesophagus", Proc. Royal Soc. Med. 67:656-658 (July 1974).

Cleveland, W., et al., "Treatment of caustic burns of the esophagus", JAMA 186(3): 262 (1963).

Leape, L.L., "Accidental ingestion of liquid lye drain cleansers", Med. Trial Tech. Q. 19(1): 30 (1972).

Lowder, S.C. and Brown, R.G., "Hypertension corrected by discontinuing chronic sodium bicarbonate ingestion", Am. J. Med. 58(2): 272 (1975).

Messersmith, J.K., et al., "Gastric erosion from alkali ingestion", Amer. J. Surg., 119:740-42 (June 1970).

Contact Toxicology

Ballen, P.H., "Mucous membrane grafts in chemical (lye) burns", Am. J. Ophthal. 55:302 (1963).

Brown, S.I. and Weller, C.A., "Prevention of the ulcers of the alkali-burned cornea", Arch. Ophthal. 82:95 (1969).

Brown, S.I., et al., "Pathogenesis of ulcers of the alkali-burned cornea", Arch. Ophthal. 83:205 (1970).

Carpenter, C.P. and Smyth, H.F., "Chemical burns of the rabbit cornea", Amer. J. Ophthal. 29:1363 (1946).

Chiang, T.S., Moorman, L.R., Thomas, R.P., "Ocular hypertensive response following acid and alkali burns in rabbits", Investigative Ophthal. 10(4):270-273 (1971).

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Dautrebande, L., Shaver, J., Capps, R., "Influence of particle matter on eye irritation produced by volatile irritants and importance of particle size in connection with atmospheric pollution", Arch. Internat. Pharmacodyn. 85:17 (1951).

Fox, S.L. Industrial and Occupational Ophthalmology. Charles C. Thomas, Springfield, IL (1973).

Gmadinger, M.C., et al., "The role of collagenase in the alkali-burned cornea", Am. J. Ophthal. 68:478 (1960).

Grant, W.M., "Chemical burns of the eye", Tr. Sect. Ophthal. AMA p. 27 (1949).

Grant, W.M., Toxicology of the Eye, Charles C. Thomas, Springfield, IL (1962).

Grant, W.M., and Kern, H.L., "Action of alkalies on the cornea stroma," Arch. Ophthal. 54:931-939 (1955).

Guidry, M.A., et al., "Some biochemical characteristics of acid injury of the cornea", Am. J. Ophthal., 40:111 (1955).

Harley, R.D., "Treatment of chemical burns of the eye", Arch. Ophthal. 49:413 (1953).

Henriquez, A., et al., "Surface ultrastructure in alkali-burned rabbit corneas", Am. J. Ophthal. 81:321 (1976).

Horowitz, I.D. "Management of alkali burns of the cornea", Am. J. Ophthal. 61:340 (1966).

Hughes, W.F., "Alkali burns of the eye. I. review of the literature and summary of present knowledge", Arch. Ophthal. 35:423-449 (1946).

Hybasek, P., "Liberation of 'sorbed' water from the skin surface: VI. desorption curves of human epidemis after treatment with sodium hydroxide", Univ. Palacki Olomac Fac Med. 62:69 (1972).

Laibson, P.R. and O'Conor, J., "Explosive tear gas injuries of the eye", Tr. Am. Acad. Ophthal. Otolargyn. 74:811 (July 1970).

Last, R.J., editor, Eugene Wolff's Anatomy of the Eye and Orbit, 6th ed., W.B. Saunders, Philadelphia (1968).

Malten, K.W. and Spruit, D., "Injury to the skin by alkali and its regeneration", Dermatologica, 132:124-130 (1966).

McLaughlin, R.S., "Chemical burns of the human cornea", Am. J. Ophthal. 29:1355 (1946).

Moses, R.A., editor, Alder's Physiology of the Eye, C.V. Mosby, St. Louis (1975).

Nagad, S., et al., "The effect of sodium hydroxide and hydrochloric acid on human epidermis", ACTA Dermato-Venereologica, 52(1): 11-23 (1972).

Obenberger, J.A., et al., "Experimental corneal burns by alkalies: study on the in-vivo iodine-125 albumin absorption from the aqueous humor into the cornea", CESK Oftalmol. 29(2):83 (1973).

Obenberger, J. and Babicky, A., "Alkali burns of the rabbit cornea: corneal uptake of 14C-glucose injected into the anterior chamber", Ophthalmic Res. 5(1):1-9 (1973).

Obenberger, J. and Babicky, A., "Alkali over acid burns of the rabbit eye: uptake of intravenously injected Na 125I and Na131I into the cornea and iris", Arch. Kum. Exp. Ophthalmol. (Eng.) 194(1): 65 (1975).

Obenberger, J. and Babicky, A., "Distribution of 24 NaCl in tissues of alkali and acid-burned rabbit eyes", Exp. Eye Res. 20(3):195 (1975).

Obrig, T.E., "Solutions used with contact lenses", Arch. Ophthal. 38:668 (1947).

Opie, E.L., "On the relation of necrosis and inflamation to denaturation of proteins", J. Exper. Med. 115:597-608 (1962).

Opie, E.L., "On the relation of inflamation to the chemical constitution of injurious agents on the pharmacology of inflamation", J. Exp. Med. 117:425-448 (1963).

Paterson, C.A. and Pfister, R.R., "Intraocular pressure changes after alkali burns", Arch. Ophthal. 81:211 (March 1974).

Paterson. C.A., Pfister, R.R., Levinson, R.A., "Aqueous humor pH changes after experimental alkali burns", Am. J. Ophthal. 79:414 (1975).

Pfister, R., et al., "The anterior segments of rabbit eyes after alkali burns", Arch. Ophthal. 86:189 (1971).

Pfister, R., et al., "Collagenase activity of intact corneal epithelium in peripheral alkali burns", Arch. Ophthal., 86:308 (1971).

Shapiro, H., "Swelling and dissolution of rabbit cornea in alkali burns", Am. J. Ophthal. 42:292 (1956).

Smith, R.S., and Shear, G., "Corneal alkali burns arising from accidental instillation of hair straightener", Am. J. Ophthal. 79:602 (1975).

Sorsby, A., et al., "Further experience with amniotic membrane grafts in caustic burns of the eye", Brit. J. Ophthal. 31:409 (1947).

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Stanly, J., "Strong alkali burns of the eye", NEJ Med. 273(23): 1265 (1968).

Zauberman, H. and Refojo, M., "Keratoplasty with glued-on lenses for alkali burns", Arch. Ophthal. 89:46 (1973).

III. ASPECTS OF THE CHEMISTRY AND PHYSICS OF NaOH AEROSOLS

INTRODUCTION

As discussed more fully below, a sodium fire will produce an aerosol composed of particles of sodium oxides. These particles are expected to react quickly (less than seconds) with the water vapor in the air to form sodium hydroxide (NaOH). Whether the NaOH particles are solid or liquid (aqueous solution droplets) will depend upon the relative humidity, primarily, and on the particle size and the temperature, secondarily. The sodium hydroxide will react with the carbon dioxide (CO_2) in the atmosphere to form sodium carbonate (Na_2CO_3) and its hydrates. The time for conversion to the carbonate will depend upon whether the hydroxide is solid or liquid and upon the size of the particle, smaller particles being converted more rapidly because they have more area per mass. Diffusion of atmospheric CO_2 to the particles is one factor determining the rate of conversion; another factor is the rate of diffusion (and convection) of the CO_2 within the particle, which is much faster for a liquid droplet than for a solid particle. Once the hydroxide is completely transformed to the carbonate, the particle may still be either liquid or solid, depending upon the humidity. As the transformation from hydroxide to carbonate takes place, the transition from solid to liquid will occur at increasingly higher humidities, starting at about 35% RH (relative humidity) for pure NaOH and reaching 95% RH for sodium carbonate in the decahydrated form, $Na_2CO_3 \cdot 10\ H_2O$.

These chemical reactions are important in determining the chemical and aerodynamic behavior of the particles, and thus, their transport and their likelihood of depositing in a given location, such as the lung or the eye. NaOH is classified as an irritant, an agent which produces its damage to animal tissue primarily by chemical attack due to its alkalinity (high pH). The corrosiveness of a droplet containing NaOH will be greater than one converted to the carbonate, because the pH of the latter is lower. We shall show that this transformation to hydroxide from carbonate can be expected in some cases to occur in less than a minute. The aerodynamic behavior depends primarily upon the size and density of the particle (to a lesser extent, upon its shape); the size and density will change as the chemcial composition changes and, most significantly, as the particle absorbs water from the atmosphere or evaporates.

Chemical conversion of the hydroxide to the carbonate will lessen the major toxicity factor, the irritant potential of the aerosol. This has two implications: one must know the conditions under which the NaOH aerosol is formed and maintained to know whether it is predominantly hydroxide or predominantly carbonate when it reaches targets of concern (people, animals, plants, materials); one must also know the conditions under which the toxicity studies of the aerosol were made to assure that the toxicity of NaOH rather than Na_2CO_3 was studied. Further, because particle aerodynamic behavior will affect the transport to targets of concern, the relative

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humidity must be specified.

The chemistry and physics of these aerosols is discussed more fully in the next section (and in Appendices A & B).

CHEMICAL REACTIONS

Clough and Garland(4) have given the probable reaction sequence resulting from the combustion of sodium.

In the presence of excess oxygen, the reactions will be

$$Na + 1/2\ O_2 = 1/2\ Na_2O_2$$

$$Na + O_2 = NaO_2$$

producing, respectively, sodium peroxide and sodium superoxide. These compounds will react with water vapor as follows:

$$Na_2O_2 + H_2O = 2NaOH + 1/2\ O_2$$

$$2NaO_2 + H_2O = 2NaOH + 3/2\ O_2$$

in both cases forming sodium hydroxide, a strong alkali. (Hydrogen peroxide may also form).

At low humidities (< 35% RH), the sodium hydroxide will be a solid, but at higher relative humidities it will be a droplet (an aqueous solution). As either solid or liquid, the hydroxide can undergo the transformation

$$2NaOH + CO_2 = Na_2CO_3 + H_2O$$

forming the less alkaline carbonate. The hydroxide and carbonate in solution will precipitate, if saturated, to form a slurry and then a solid, which will have various chemical compositions depending upon the amounts of hydroxide and carbonate present. Table 3, from Linke(5), shows the amounts of hydroxide and carbonate present in a saturated solution and the chemical nature of the precipitate. If the reaction has gone to completion before the precipitate forms, then the solid phase would be $Na_2CO_3 \cdot 10\ H_2O$, sodium carbonate decahydrate, and Clough and Garland note this would become a dry particle (a solid) unless the humidity rose to above 95% RH. As can be inferred from Table 3, various hydrates of sodium carbonate will precipitate as the reaction proceeds and the hydroxide is consumed. The reaction between the NaOH and CO_2 should proceed as follows:

1. For a solid NaOH Particle:
    a. CO_2 reacts with NaOH at the surface to form Na_2CO_3 and H_2O; the water may evaporate or stay with the Na_2CO_3 as hydrates;
    b. the same reaction occurs in the interior, with CO_2 diffusing in and H_2O diffusing out or hydrating;

Table 3. Concentrations of Na_2CO_3 and NaOH in saturated solutions and the associated solid phase(5)

Grams of solute per 100 g saturaged solution at 20°C		Solid phase
Na_2CO_3	NaOH	
18.0	0.0	$Na_2CO_3 \cdot 10H_2O$
12.9	6.3	$Na_2CO_3 \cdot 10H_2O$
11.6	12.7	$Na_2CO_3 \cdot 10H_2O$
12.4	13.4	$Na_2CO_3 \cdot 10H_2O + Na_2CO_3 \cdot 7H_2O$
11.7	14.7	$Na_2CO_3 \cdot 7H_2O$
11.1	16.2	$Na_2CO_3 \cdot 7H_2O + Na_2CO_3 \cdot H_2O$
5.2	22.0	$Na_2CO_3 \cdot H_2O$
1.1	29.7	$Na_2CO_3 \cdot H_2O$
0.3	39.8	$Na_2CO_3 \cdot H_2O$
0.3	41.5	$Na_2CO_3 \cdot H_2O + Na_2CO_3$
5.4	43.9*	Na_2CO_3
9.6	43.8*	Na_2CO_3
0.0	52.1	$NaOH \cdot H_2O$

*This solution was not clear.

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c. Na_2CO_3 will slowly diffuse into the NaOH and the NaOH will diffuse into the Na_2CO_3, both rates being slow compared to the CO_2 diffusion.

2. For a liquid NaOH droplet:

   a. CO_2 reacts at the surface to form Na_2CO_3, which in the early stages of the reaction will dissolve into the rest of the droplet;

   b. CO_2 and other molecules will diffuse through the droplet, aided by any circulation, and the reaction between the carbon dioxide and the sodium hydroxide will continue, with the formation of various sodium carbonate hydrates (see Table 3), as the droplet concentration of hydroxide decreases and the concentration of the carbonate increases; these solids may form a shell, which would slow down further reaction, or they may produce a slurry or perhaps a single solid within the liquid droplet.

The rate at which the hydroxide will be converted to the carbonate will be determined by the following factors:

1. Rate of transfer of carbon dioxide from the air to the particle;
2. Diffusion of carbon dioxide through the particle;
3. Rate of reaction of carbon dioxide and sodium hydroxide at the molecular level;
4. Rate of diffusion of the products.

Factors affecting the rate of transfer of atmospheric carbon dioxide to the particle surface include the ambient concentration (typically 330 ppm) the diffusivity (molecular and eddy) of the carbon dioxide, the relative velocity between the gas and the particle ("ventilation factor", independent of turbulence in the gas, primarily influenced by the particle settling velocity), and particle size.

Affecting the diffusion of the CO_2 from the particle surface to its interior will be the diffusivity (again both molecular and eddy, as the liquid will undergo some circulation within the droplet), the concentration at the surface, the reaction rate for conversion of hydroxide to carbonate, and the particle size.

The rate of reaction is expected to depend upon the reactant concentrations and the temperature.

Diffusion of the products will depend upon the diffusivities, the concentrations, and the particle size.

The rates at which CO_2 will diffuse to the surface of the particles and the rate of transfer within the particles (liquid and solid) are discussed more fully in Appendix B. Our analysis, given there, indicates that for solid NaOH particles of 20 µm diameter or smaller (or droplets formed therefrom), enough atmospheric CO_2 could diffuse to the surface of the particles within ten seconds or less to convert the hydroxide to carbonate for NaOH droplets. If the humidity is quite low (less than 35% RH), then the NaOH will be in solid form and diffusion within the particle will be greatly retarded; the reaction rate may be slowed as well because the NaOH is not dissolved; these slower rates could, in turn, retard diffusion to the particle surface because the surface will become saturated. Our estimates are that respirable solid NaOH would be converted to carbonate within a time scale measured in minutes.

A third set of chemical reactions of interest is the establishment of an equilibrium between sodium carbonate, sodium bicarbonate, ambient carbon dioxide, and water in the particles which become sodium carbonate at the conclusion of the reaction between sodium hydroxide and carbon dioxide. Clough and Garland(4) indicate that formation of bicarbonate should occur only at humidities above 95% RH and should require times of the order of 10-100 hours. (This reaction is significant in that it lowers the pH very nearly to neutrality.) This reaction rate was determined by McCoy and Smith(6) by bubbling CO_2 through water and should be checked to see if it holds for aerosol particles in air. If the reaction does take 10 hours, then it is a factor of no importance for people in the vicinity of a sodium fire.

The results of the theoretical analysis done by Clough and Garland(4) are: "The formation of sodium carbonate from sodium hydroxide droplets is controlled by liquid phase diffusion of reactants in the drop, and probably occurs in times less than a few minutes. The formation of sodium bicarbonate from sodium carbonate droplets is much slower and delays of tens of hours may elapse before equilibrium is achieved.

"At high relative humidities the final state will certainly be a droplet of solution. At humidities below 95 per cent the final state will be a particle of sodium carbonate, although droplets of hydroxide solution may be present for a time on of the order of 1 minute or less. The appreciable changes in size consequent upon changes in relative humidity may lead to important changes in the residence time and deposition of particles in the atmosphere.

Our analysis extended that of Clough and Garland to include solid particles and to take into account that the reaction in the droplet will increase the rate of absorption of the carbon dioxide from the atmosphere. Table 4 lists three relative humidity conditions and shows what compounds are expected for times on the order of seconds, minutes, hours, and days, respectively.

Experimental work relating to this question has been done by Murata, et al.(7). They vaporized sodium in argon to form an aerosol of 1 mg/m^3, then reacted the aerosol with oxygen at several humidities and several concentrations of carbon dioxide. The dry particles were about 2 µm in mass median aerodynamic diameter with geometric standard deviation of 1.8 to 2.0. The mean residence time

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Table 4.

Species Expected from NaOH Aerosol (2 µm Diameter Particles) in the Atmosphere at 20°C

Residence time in Atmosphere	Chemical Species Expected for the Following Relative Humidities		
	<35%	35% - 95%	>95%
∿ sec	$(\mathrm{NaOH})_s$*	$(\mathrm{NaOH})_l$	$(\mathrm{NaOH})_l$
∿ min	$(\mathrm{Na_2CO_3})_s$**	$(\mathrm{Na_2CO_3})_s$**	$(\mathrm{Na_2CO_3})_l$
∿ hr	"	"	"
∿ day	"	"	$(\mathrm{NaHCO_3})_l$

* Probably as $\mathrm{NaOH \cdot H_2O}$. The subscripts are: s = solid, l = liquid.

** Probably $\mathrm{Na_2CO_3 \cdot 10H_2O}$

in the stirred reaction chamber was 1.4 minutes. Measurements were made of sodium content and of particle aerodynamic size distribution. For humidities below 40% RH, the particles remained dry. Murata, et al. assumed the resulting aerosol to be a mixture of sodium hydroxide and sodium oxides. As the concentration of carbon dioxide in the chamber increased, the particles remained solid at increasingly higher humidities; thus, the transition to droplets for reaction at atmospheric CO_2 levels was at humidities between 40% and 60% RH, suggesting the particles were predominantly sodium hydroxide. The transition to droplets at thirty times the atmospheric carbon dioxide level was at between 70% and 90% RH, suggesting the particles were predominantly in the carbonate form. The study qualitatively agreed with the work of Clough and Garland, but the relative amounts of carbonate and hydroxide were not identified for the various conditions, and the effect of humidity was confounded: if humidity is to be used to gauge whether the hydroxide or carbonate predominated, it should be varied <u>after</u> the reaction is allowed to take place under specified carbon dioxide and humidity conditions. The work added credence to the predictions above (see Table 4) but neither verified nor contradicted them.

ALKALINITY: NaOH and Na_2CO_3

It is generally agreed that the injury from sodium hydroxide or sodium carbonate is determined by the alkalinity (concentration of OH^- ions) of the solutions formed by these substances. The relationship between the OH^- and H^+ ions in aqueous solution is given by

$$K_w = [OH^-][H^+] \tag{1}$$

in which K_w is the dissociation constant of water, itself a function of temperature. The brackets indicate "concentration of " in moles per liter. The pH of a solution is defined as

$$pH = -\log [H^+] = -\log K_w [OH^-]^{-1} = -\log K_w + \log [OH^-] \tag{2}$$

which, at 25°C, becomes ($K_w = 1.0 \times 10^{-14}$):

$$pH = 14 + \log [OH^-] \tag{3}$$

so that the higher the pH the greater the concentration of OH^- (the greater the alkalinity). For sodium hydroxide, a very strong base, virtually all the OH^- ions go into solution and the pH is easily determined. For the carbonate, there is a second equilibrium involved (carbonate-bicarbonate). Table 5 gives some values for the pH (at 25°C) for both the hydroxide and the carbonate at various normalities. It will be seen later that eye damage, for example, accelerates greatly as the pH of the solution in the eye goes above 11, which (note Table 5) will occur at NaOH concentrations of 0.001N, but not until Na_2CO_3 concentrations reach 0.04 N, forty times the NaOH concentration (normality). This means the chemical composition of the aerosol will be quite important with respect to its toxicity.

Table 5. pH values for some NaOH and Na_2CO_3 concentrations in water (at 25°C)

Concentration (normal)	pH		
	Na_2CO_3 [a]	NaOH [b]	
	Measured	Theoretical	Measured
1.04×10^{-3} N [c]	10.27	11.02	
4.0×10^{-3}	10.61	11.60	
0.01		12.00	11.94
0.016	10.92	12.22	
0.042	11.06	12.62	
0.05		12.70	12.62
0.082	11.17	12.91	
0.164	11.25	13.21	
0.346	11.34	13.54	
0.84	11.45	13.92	
2.56	11.62	14.41	
4.10	11.70	14.62	

a) L. Lortie, P. Demers, Can. J. Res. B18: 160.

b) Theoretical: $pH = 14 + \log [OH]$
Measured: R.G. Bates, G.D. Pinching, E.R. Smith (1950) J. Res. Nat. Bur. Std. A. 45: 418.

c) 1 M NaOH = 1 N NaOH, and contains the same number of Na atoms per volume as 0.5 M Na_2CO_3 = 1 N Na_2CO_3.

PARTICLE AERODYNAMIC BEHAVIOR

For particles 1-100 μm in diameter at normal temperature and atmospheric pressure, the particle size parameter of most utility is the aerodynamic diameter. The aerodynamic diameter, d_{ae}, is the diameter of a unit density sphere which, in still air, would have the same terminal settling velocity as the particle.

The settling velocity of a sphere is given by:

$$v_s = (C\rho_p d_p{}^2/18\ \mu)\ g = (C\rho_o d^2{}_{ae}/18\ \mu)\ g \qquad (4)$$

in which

C = Cunningham correction factor ($\doteq 1+0.16\text{x}10^{-4}\text{cm}/d_p$)

ρ_p = particle density

ρ_o = density of water, 1.00 g/cm^3

d_p = particle diameter

μ = gas viscosity

g = acceleration of gravity, 981 cm/s^2.

The term in parentheses is the relaxation timeof the particle, τ. The terminal velocity the particle will reach under any applied force is the force times the relaxation time divided by the particle mass. The likelihood that a particle will strike an obstacle toward which it is being carried by the air is determined by the ratio of the air velocity times the relaxation time divided by the obstacle's narrowest dimension perpendicular to the gas flow. For particles larger than about a micrometer, the aerodynamic diameter is given for spheres by

$$d_{ae} = d_p\ (\rho_p/\rho_o)^{1/2} \quad . \qquad (5)$$

As detailed in Appendix A, the transformation of a dry NaOH particle to a dry $Na_2CO_3 \cdot 10H_2O$ particle will increase the aerodynamic diameter (for $d_p > 1$ μm) about 40%, which will increase its deposition velocity due to gravitational settling and due to impaction. The increased impaction will help limit the particle deposition to the upper respiratory tract and nasal passages (unless, of course, $d_p \lesssim 1$ μm). The effect of the transformation on human respiratory retention will depend upon the particle size distribution.

Particles of NaOH will become droplets after humidities (H) exceed 35% RH. In Appendix A, it is shown that the following equation closely approximates the change in particle size due solely to the absorption of water:

$$d_d/d_p = 0.87/(1-H)^{1/3} \qquad (6)$$

(The droplet diameter is d_d; its dry diameter is d_p.) This increase in particle size is accompanied by a decrease in particle

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density; the net result is to produce the following increases in aerodynamic diameter: x 1.2 at 70% RH; x 1.4 at 90% RH; x 2.6 at 99% RH. This will increase the fraction captured in the nasal passages (for $d_p > 1$ μm).

The particle which results from the transformation of the initial NaOH particle will have an aerodynamic diameter which will depend upon the chemical reactions which take place and upon the relative humidity. Because the humidity in the nasal passages and the upper respiratory tract is nearly 100% RH, one can expect a very substantial growth in particle size, again favoring capture before the particles reach the lower respiratory tract.

SUMMARY

In order to interpret studies of NaOH aerosol toxicity or to predict toxicity, one must know what compounds are being dealt with and the particle size distribution.

For relative humidities exceeding 35% RH, it seems clear that NaOH droplets will be transformed to sodium carbonate decahydrate in the atmosphere (for NaOH aerosol concentrations $\lesssim 100$ mg/m^3) in less than a minute. If the humidity is less than 35% RH, this transformation will take longer. The carbonate will remain solid as long as the humidity remains less than 95% RH. The alkalinity of a sodium carbonate solution will be substantially less than that for the same concentration (normality) of sodium hydroxide; this means carbonate aerosols will be less hazardous, per Na atom, than hydroxide aerosols. The change from sodium hydroxide to solid carbonate will produce about a 40% per cent increase in particle aerodynamic diameter, which (for particles with diameters > 1 um) will decrease the respirable fraction of the aerosol. The growth of particles (hydroxide or carbonate) at high humidities (such as in the upper respiratory tract) will also decrease the respirable fraction.

IV. NaOH AEROSOL TOXICITY

INTRODUCTION

The chemical toxicity of a substance is its ability to cause chemical injury to an organism (in our context, man). Factors which influence the degree of such injury include: susceptibility of the individual, intensity (concentration) and duration of the chemical exposure, nature of the specific site of contact, including transfer rate from surface to substructure, and the rate at which the body detoxifies the site. Amdur(8) noted, "If a material is taken into the body at a rate sufficiently slow that the rate of excretion and/or detoxification keeps pace with the intake, it is possible that no toxic response will result even though the same total amount of material taken in at a faster rate would result in a concentration of the agent at the site of action sufficient to produce a toxic response. Information of this sort enters into the concept of a threshold limit for safe exposure." Thus, concentration (degree of localization) and the rate at which the material comes in contact with the body can affect the amount of injury, if any, produced. Such injury may range from transient to permanent.

Nearly complete knowledge of the toxicity of a substance would include knowing the degree of injury sustained by many different classes of people (based upon, for example, age, sex, health) for different durations of exposure at different intensities. If this knowledge is incomplete, assumptions must be made for decision making.

This chapter has as its goal the presentation of information to relate exposures to NaOH aerosol to different kinds and degrees of injury to exposed persons. The authors have reviewed the literature and present here what they have discovered with regard to standards already set (and the bases for those standards). This has been supplemented with information relating dose and response in humans. The information on response to NaOH came, almost exclusively, from accident reports or from experiments using animals under laboratory conditions. In the first category, the details of the dose to the individuals are often sketchy; in the second, applying the data to predict human response is difficult. The documentation for standards set by certain organizations for conccentration limits on NaOH aerosol, based upon inhalation, are far from conclusive. No standards were found for contact with skin or eyes nor for ingestion of inhaled material when coughed up and swallowed. Information on splashes and application of hydroxide and carbonate solutions to the eyes was located. This information may be combined with a preliminary model of aerosol impaction on the eye (and subsequent dilution by ocular fluids) to predict response by human eyes to NaOH aerosol exposures.

CURRENT STANDARDS

1. Inhalation

In the United States, both the American Conference of Governmental Industrial Hygienists (ACGIH) and the National Institute of Occupational Safety and Health (NIOSH) have proposed similar standards for workers.

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ACGIH set 2 mg/m^3 as its threshold limit value (TLV) for NaOH in the workplace air. This is a ceiling, not a time weighted average. The ACGIH booklet(3) listing TLV's has this to say about their definitions: "Threshold limit values refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect" (p. 1). NaOH went from being categorized as a substance which was given a time weighted average (TWA) TLV to one given a "ceiling" by the ACGIH. They wrote that the ceiling is, in effect, a "maximum allowable concentration." It was noted: "In general the bases for assigning or not assigning a 'C' value rests on whether excursions of concentration above a proposes limit *for periods up to 15 minutes* may result in a) intolerable irritation, b) chronic or irreversible tissue change, or c) narcosis of sufficient degree to increase accident proneness, impair self-rescue or materially reduce work efficiency." (The italics are theirs.)

The documentation offered by ACGIH was:(9)

> "Caustic dusts are irritating to the upper respiratory system. Although prolonged exposure to high concentrations may cause discomfort and even ulceration of nasal passages, subjective symptoms are often relied upon as an indication of the need for control.
>
> "Patty, on the basis of the irritant effects of caustic mists, encountered in concentrations of 1 to 40 mg/m^3, believes that 2 mg sodium hydroxide/m^3 of air represents a concentration that is noticeably, but not excessively, irritant."*
>
> "Soviet limit (1959), 0.5 mg/m^3."

The same documentation was offered when the TLV was placed at 2 mg/m^3 as a time weighted average, not a ceiling. As will be discussed more below, the Soviet policy is to set "limits" which are treated as goals, and which represent the lowest level at which there are clear indications of response, rather than levels that cause injury (or even discomfort).

Stokinger(10) noted that the underlying philosophy of the threshold limit values of the ACGIH is that "although all chemical substances are toxic at some concentration experienced for a period of time, a concentration exists for all substances from which no injurious effect will result no matter how often the exposure is repeated." This is in distinct contradiction, he noted, to the treatment of exposures to radioactive materials, assumed to be cumulative. Overwhelmingly, the bases for the ACGIH values were either changes in organ function or evident irritation. Thirty-eight per cent were based on worker reactions and another 11% based upon human volunteer experiments. He defends these standards further by noting that where they have been followed, injury has been prevented.

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*The term "irritant" means causing inflamation.

The following is the standard proposed by NIOSH(2): "Occupational exposure to sodium hydroxide shall be controlled so that no worker is exposed to sodium hydroxide concentration greater than 2.0 mg/m^3 of air for any 15-minute sampling period." It was pointed out by NIOSH: "The standard was not designed for the population at large and any extrapolation beyond general occupational exposure may not be warranted." They note that this is the same as that adopted by the ACGIH(3). (ACGIH referenced only the opinion of Patty(11).)

The NIOSH document noted that Patty(11) commented that he could not find published records of industrial measurements of airborne sodium hydroxide, but concluded, "from the irritant effects of caustic mists encountered in concentrations from 1 to 40 mg per cubic meter of air, 2 mg sodium hydroxide per cubic meter is believed to represent a concentration that is noticeably, but not excessively, irritant." Patty seems to be the source of the 2 mg/m3 limit, yet it is clearly not well substantiated. Note that ACGIH had used Patty's wording verbatim and, along with NIOSH, the reference is to be first edition of Patty's book; no justification of the 2 mg/m^3 is made in his second edition.(16)

A second piece of information used to support the 2 mg/m3 occupational standard is a study(2) done by NIOSH in 1974 in connection with a degreasing operation. The degreasing vat was maintained at 200°F and contained primarily a sodium hydroxide solution, having a pH of 12.5 to 13.5. "Operations adjacent to the vat involved the use of other substances (e.g., Ensis 254 oil, Stoddard solvent, Zyglo and Magna flux)." Two employees experienced nose and throat irritation, chest pains, shortness of breath. A third experienced nose and throat irritation, nausea, and vomiting. Antibiotics were effective as treatment. Sampling done six months later indicated "the airborne concentration of Stoddard solvent to be 14-780 mg/cu m; other solvent vapors (undescribed) were 276-725 mg/cu m...." (The ACGIH 1977 value for the TLV for Stoddard solvent is 575 mg/m^3, TWA.) Airborne sodium concentration, reported as sodium hydroxide was 0.005 to 0.7 mg/m^3, airborne sulfuric acid concentrations were 0.3 - 2.2 mg/m^3. (The 1977 ACGIH TLV for sulfuric acid is 1 mg/m3.) More sampling was done later; personnel were asked to fill out forms describing their health and any reactions, symptoms, etc. About half of the fifteen workers surveyed had experienced some irritation of the upper respiratory tract, but these symptoms and others were uncorrelated with changes in ventilation and removal of the vat cover and in one category (burning/dryness of the throat) were correlated with periods when the vat cover was on rather than when it was off. The NIOSH standards document concludes(2), "These observations are not entirely consistent with the conclusion reached by the investigators that the mist from the vat produced the signs and symptoms." The document also points to the high concentrations of Stoddard solvent as a possible problem.

The final human study cited by the NIOSH document(2) is one done with respect to oven cleaners. Here NIOSH concluded, "air sampling and chemical analysis for sodium hydroxide and other ingredients was not extensive enough to allow identification of the specific agents responsible for this irritance." The NIOSH document cites a communication from T. R. Lewis in 1974 about his study of oven cleaners containing, among other ingredients, sodium hydroxide (producing airborne concentrations reported to be in the range 0.24 - 1.8 mg/m^3), in which respiratory irritation was produced in healthy volunteers in short periods of time (2-15 min.). Later, the document states, "the number of uncontrolled variables in the study, including questions about the reliability of the estimates of airborne sodium hydroxide, are such that confirmation and extension of this study should first be accomplished."

Animal inhalation studies by Dluhos, et al.(12), were also cited by NIOSH. The airborne concentration of sodium hydroxide was unspecified. The uncertainties of this experiment were compounded in similar work by Vyskocil, et al.(13), also cited by NIOSH. The Vyskocil work involved a quartz dust aerosol as well, in concentrations of 10 g/m^3; a concentration which is 10^5 times higher than the ACGIH TLV for quartz.(3)

The NIOSH criteria document gave the following table for the standards set by some other countries for airborne sodium hydroxide, but the sources from which these values were taken did not indicate how the values were derived.

Standards for Exposure to Sodium Hydroxide Aerosols(2)

Country	Substance	Standard (mg/m^3)
USA	sodium hydroxide	2 (TWA)[a]
Finland	sodium hydroxide	2 (Ceiling)
West Germany	sodium hydroxide	2 (Ceiling)
Yugoslavia	sodium hydroxide	2 (Ceiling)
Rumania	hydroxides (alkaline)	1 (Ceiling)
Bulgaria	alkalines (sodium hydroxide, etc.)	0.5 (Ceiling)
USSR	alkaline aerosols (as sodium hydroxide)	0.5 (Ceiling)

a) Authors' note: USA standard is now a ceiling, also.

The USSR level may have been based upon the work of Gavrilova of which we have the following abstract (<u>ChemAbs</u>. 55, 4898/i 1961):

> "G. S. Luzina's method (CA 45:10135 i) was applied to the determination of caustic concentrations in air within a plant producing alumina by the wet caustic process. These concentrations were found to be in the range from 0 to 9 mg/cu m of air. Values exceeding 1.0 mg predominate; the concentrations 0.5 mg/cu m are the rarest. Two tables show the relation between this concentration and several factors related to the technological processing. Some 500 workers of the plant underwent medical examinations to establish the effect of the aerosols on the upper respiratory tracts. The data clearly showed a health hazard created by caustic aerosols. Caustic air concentration of 0.5 mg/cu m air (converted for NaOH) is recommended as a limiting permissible value. Some measures are listed which aim to decrease the escape of aerosols into air, as well as measures related to the health protection of workers."

Again, there may have been other contaminants and the exposures are more nearly chronic than short-term.

Elkins(13) noted that the USSR values were derived from a different methodological viewpoint from that of the US. The USSR values represent levels that are related to <u>responses</u>; the US values represents levels that are related to <u>injury</u>. Ryazanakov(14) indicated that the lowest odor threshold for a group of subjects would typically be chosen as the Maximum Acceptable Concentration (MAC), for example; another response tested was dark adaptation of the eye; the results from these two methods often agreed; a third approach was that of studying conditioned electrocortical reflexes. Ryazanakov made it clear in his discussion that the USSR standards are used differently from those of the US: they are set as goals or targets for eventual achievement, the requirements for having a "clean atmosphere, and the government decides what practical concessions must be made."

The NIOSH criteria document lists the Patty statement, the NIOSH investigation of the vat exposures and the oven cleaner study as "probably the only reports in the literature which discuss either human or animal response to a known, low concentration of sodium hydroxide." We do not believe such information is adequate for determining a sodium fire accident standard based upon health effects. Not only are the data few, but the duration of industrial exposure is much greater than that expected for a sodium fire accident. The populations at risk also differ.

2. Ingestion

Standards relating to ingestion were not found, although it is widely recognized that, to avoid internal burns, NaOH solutions should not be ingested.

3. Skin and Eye Contact

Standards for exposure were not found but there are requirements that NaOH solutions be marked to indicate that contact with skin and eyes (as well as ingestion) can be dangerous.

SUPPLEMENTAL INFORMATION

1. General Toxicity

In the second revised edition of <u>Industrial Hygiene Toxicology</u>, Patty(16) wrote with respect to the physiological response to NaOH: "Characteristic irritation of nasal tissue frequently causes sneezing....The greatest hazard is that of rapid destruction of any tissue upon contact with the solid or concentration solutions. The inhalation of dust or mist is of secondary industrial importance." His comment with respect to sodium carbonate (Na_2CO_3) was, "Sodium carbonate is a primary skin irritant..., and dusts or mists are moderately irritating to the nasal membranes." Regarding a "hygienic standard of permissible exposure" to sodium carbonate, Patty wrote, "None has been proposed, but it should be higher than that permissible for sodium hydroxide." This statement reinforces the importance of determining whether the hydroxide has had time to become

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carbonate before humans are exposed to the aerosol.

Sax(17) gave the following description of the toxicology of NaOH:

> "...Mists, vapors, and dust of this compound cause small burns, and contact with the eyes, either in the solid or solution form, rapidly causes severe damage to the delicate tissue. Ingestion either in the solid or solution form causes very serious damage to the mucous membranes or other tissues with which contact is made. It can cause perforation and scarring. Inhalation of the dust or concentrated mist can cause damage to the upper respiratory tract and to lung tissue, depending upon the severity of the exposure. Thus, effects of inhalation may vary from mild irritation of the mucous membranes to a severe pneumonitis. It can cause irritant dermatitis."

From the material presented thus far, it seems clear that NaOH aerosols can be hazardous, but that the concentrations, durations, and particle size distributions which are hazardous have yet to be determined.

2. Inhalation Toxicology

Little additional information was uncovered concerning the harmful effects of inhalation of such aerosols.

In a letter to the USAEC (13 October 1965), Dr. H. W. Hays of the National Academy of Sciences wrote: "Inhalation of mists of sodium hydroxide may produce effects varying from mild irritation to severe pneumonitis depending upon the concentration. There is a wide variability among persons in their subjective response to caustic dust, but it has been observed that, in general, 6.0 mg/m^3 produced intolerable respiratory discomfort....Eye contact with mists and dust of sodium hydroxide may produce severe damage and it is therefore recommended that workers be required to wear tight fitting eyegoggles when there is a potential exposure to concentrations above 2.0 mg/m^3." That recommendation (and an "Emergency Exposure Limit" for 30 minutes of 4.0 mg/m^3) was made by the "Committee on Toxicology." There were no references cited in the letter, which was in response to an inquiry dated about a month earlier. It is not clear what considerations went into the making of these recommendations.

Caujolle et al.(18) reported studies of the exposure of guinea pigs to various alkaline aerosols. They determined the particle size distribution (by count) but did not indicate the mass concentration, making the work of negligible value in our context.

Human volunteers were exposed to measured concentrations of sodium hydroxide aerosol (presumably somewhat transformed to sodium carbonate) in one study that was primarily directed towards investigating visibility in such an aerosol, done by Hughes and Anderson (39). The aerosol was generated by burning sodium and was analyzed by transmissometry, calibrated by acid-base titration of aerosol samples caught in a bubbler. Three adult subjects were used at various times during the test. They were called subject A, subject B, and subject C. Subject C wore goggles; the other two were unprotected. The aerosol concentration was measured as equivalent amount of NaOH, from the titrations. Listed below are the concentrations and the effects:

Concentration (mg/m^3 as NaOH)	Effect
2-10	Subject A: "Characteristic odor. Slight watering of eyes and nose after inhalation for five minutes."
3	Subject C (without goggles): "Slight prickling behind eyelids."
25	Subject C (with goggles): "Slight prickling in upper nasal passage. Irritation not severe. Capable of work; quite comfortable provided it does not get any worse."
25	Subject C (with goggles): "Capable of work and prepared to stay longer. Mouth breathing induced coughing." Exposure time: 11 minutes total for Subject C in this series.
180	Subject B: "3 minute exposure of eyes (accidental) without inhalation resulted in slight irritation (prickling and watering sensation) lasting for 24 hours."

From this work, Hughes and Anderson concluded: "Short term exposure of unprotected workers up to 40 mg/m^3 NaOH in air is unlikely to result in any serious discomfort. At about 100 mg/m^3 exposure of unprotected workers will probably result in serious discomfort which will certainly cause them to consider their own survival before any work task." It should be noted that discomfort rather than injury was investigated, and injury may occur at levels higher or lower than discomfort.

It is surprising that so little useful information of NaOH aerosol inhalation dose-response exists in the published literature.

3. Ingestion Toxicity

The inhaled NaOH aerosol can be partially ingested due to coughing and swallowing. In his review of the literature on hypertension (thought by some to be caused partially by dietary salt), Page(19) cited the case of a three-year period of increased salt intake: nomadic warriors from Kenya who served several years in the army were, as a result of this service, taken from a diet which contained 2 to 3 g of Na per day to an army diet which contained 16 g of sodium per day; aside from some increase in blood pressure, no adverse effects were reported. The average dietary sodium is 4-6 g/day(20).

A NaOH aerosol concentration of 100 mg/m^3 would have 57.5 mg/m^3 of Na. With an average breathing rate(21) of 20 m^3/day = 0.833 m^3/hr, a one-hour exposure to such an aerosol would mean taking into the body 0.048 g of sodium the day the exposure occurred, much less than the dietary level of sodium.

4. Contact Toxicity

Although aerosol contact with the skin can produce dermatitis, the most sensitive area for contact is the eye, so this review focuses there. Many articles have been written about the response of the eye to alkali solutions. Virtually all deal with spashes or with deliberate applications of such solutions to the eyes of test animals. We will summarize much of that information and indicate (in Appendix C) how one might estimate the concentration of NaOH in the tears of those exposed to sodium hydroxide aerosol.

Grant(22) discussed alkali burns of the eye in general and also the effects produced by several sodium compounds, including sodium hydroxide and sodium carbonate, specifically. Grant summarized a review by Hughes(23): "He concluded that the severity of the damage to the eye is less dependent upon the character of the cation than upon the concentration of the alkali, the duration of the exposure, and the pH of the solution." Injury is strongly related to pH: above pH = 11.5, injury to the corneal stroma increased sharply. Experiments on rabbit corneas with their epithelium removed showed slight and reversible damage with a 10 minute exposure at pH = 10 but severe damage at pH = 11. The permeability of the alkali solution was used as a measure of damage to the epithelium of beef corneas, and it was found that at pH = 10 there was no increase in permeability, but at a pH = 11 and 12, there was rapid increase...with sodium hydroxide acting more rapidly than calcium hydroxide or most other hydroxides tested. Grant emphasized that in the treatment of such injuries, time is very important, and "nothing is better than immediate irrigation." Sodium carbonate forms alkaline solutions with pH up to 11.6, Grant noted, from which one can conclude that it would take the most concentrated solutions of sodium carbonate to produce rapid eye injury. He wrote that application of a pH 10.7 solution (10% solution) to rabbit eyes followed in 30 seconds by irrigation caused no detectable injury. Sodium hydroxide, on the other hand, "is severely injurious to all tissues, and causes some of the most severe injuries of the eye," presumably because of the very high pH values obtainable with concentrated solutions (a 10% solution has pH = 14.4).

The cornea is the portion of the eye most likely to be damaged to the extent of producing vision impairment, temporary or permanent. The structure of the cornea is as follows: the outer surface is a layer of epithelial cells; adjacent to this is Bowman's layer, and next to that is the bulk of the cornea, the stroma. The inner surface of the cornea is formed by the endothelium, and between the endothelium and the stroma is another thin layer, Descemet's layer. In their chapter on the toxicology of the eye, Potts and Gonasun stated(24), "A scar, the normal body reparative process, with or without vascularization, is tolerated by other body structures with no adverse effects. In the case of the cornea a scar or vascularization can destroy function completely. Hence a very small amount of corrosive substance--an amount of no consequence elsewhere on the body--can be the cause of blindness if it reaches the cornea." These same authors point out that a special feature of alkali burns is the serious late effects that can occur when there is no apparent immediate severe injury: "Even burns that at the time of injury appear to be mild can go on to opacification, vascularization, ulceration, or perforation." They cited the work of Hughes(23), who produced different degrees of injury on rabbit corneas using 0.05 N $NaOH$ solutions ($pH = 12.7$): exposure for more than three minutes led to opacification, ulceration, perforation; exposure for thirty seconds caused only temporary signs of minor damage which cleared within weeks.

In a book edited by Adler(25), Milder gives the following quantitative information about the eye. The tear film has a superficial oily layer so thin that interference patterns are not set up, indicating it is thinner than the wavelength of visible light, 0.5 µm. Beneath this is an aqueous layer 6.5 to 7.5 µm thick. Beneath this aqueous layer is a mucoid layer. The pH of tears is usually 7.4 with a typical range of 7.3 to 7.7 and the normal waking flow rate is 0.50 to 1.25 g/16 hr. The oil film reduces evaporation to the level of 20-25% of the total tear flow. Tears include a variety of electrolytes, carbohydrates, proteins. enzymes and even vitamins. Discomfort will arise from solutions outside the pH range 6.6 to 7.8. The sodium chloride content and the other salts give an osmotic pressure equivalent to 0.9% to 0.95% $NaCl$, and discomfort will arise if the fluid goes beyond the range 0.6% to 1.3%. If a hypertonic solution of 2.5% saline is placed on the eye, it is characteristically diluted to the concentration of tears within 1 1/2 to 2 minutes. Blinking rates for men are 3.6 to 29 per minute, with accelerated rates when there is excess tear formation. Some of this information has been incorporated into the preliminary model given in Appendix C.

According to Fox(26), "Mild alkalis produce only a transient irritation of the external ocular tissues and are soon neutralized by the tears. As the concentration of the hydroxyl ion increases the chemical effects on the eye become increasingly more severe. Substances with pH above 11.0 are exceedingly dangerous to the eye." He reported that subjective discomfort occurs when solutions outside the pH range 6.6 to 7.8 are put into the eye.

NIOSH(2) cited that work of Grant and Kerr(27), who found that 0.23 N $NaOH$ (at $pH = 11$) applied for 15 minutes to rabbit eyes which had their epithelium removed beforehand produced only slight and reversible damage, but a similar application of $pH = 12$ solutions produced severe injuries with opacity.

Brown et al.(28) produced ulcerations and perforations to the cornea of rabbits by applying 0.5 N $NaOH$ solutions ($pH = 13.7$) for 30 seconds, with the damage becoming evident within two to three weeks.

Carpenter and Smyth(29) graded 180 chemicals for the severity of damage they caused to the rabbit eye, and the most severe category (1 per cent solution causes "injury score" of over 5.0) was occupied by only sodium hydroxide and maleic anhydride. (A 1 per cent solution of NaOH has pH = 13.4.) They noted that the human epithelium is 45.4 μm and the rabbit epithelium is 33.6 μm thick, so that the human eye is somewhat more resistant to damage. Alkali burns, as do acid burns, require immediate irrigation for effective prevention of damage, but irrigation must be much longer for the alkali.

McLaughlin(30) summarized extensive experience with accidental chemical burns of the cornea. He cited the experience of one plant: "In a recent tabulation for 12 months in the same establishment, it was found that 52 percent of the chemical eye injuries were caused by vapors and mists, 31 percent by liquids, and 17 percent by solids." McLaughlin reported that trying to neutralize an alkaline solution was not successful in limiting damage, but removing the damaged epithelial cells was. Below is a truncated version of the table he presented showing the results of his treatment (denuding) for corneal chemical burns:

	Prompt healing, within 48 hrs.	Slow healing, 3-10 days	Some loss of vision
Total cases	456	37	7
NaOH (solid)	12	2	—
NaOH, > 50% (pH > 15.1)	—	2	—
NaOH, 26-50% (14.8-15.1)	27	1	1*
NaOH, 10-25% (14.4-14.8)	89	3	—

*Not treated until 48 hours later; final vision was 20/50.

From his study of 500 consecutive cases, McLaughlin(30) concluded:

1. "Immediate first aid, followed by long flushing with water is of the utmost importance. No attempt at neutralization is recommended.

2. Specialized treatment by an ophthalmologist should be given within two hours after the injury. Further delay threatens recovery of full vision.

3. Denudation is the treatment of choice.

4. It is noted that the majority of cases, 456, or 91.2 percent, healed in 48 hours with no loss of vision; 37, or 7.4 percent healed slowly, but without loss of vision; 7 cases, or 1.4 percent, healed with a residual loss of vision. The loss of vision in all seven cases was the result of inadequate preliminary care or health conditions present before the eyes were burned.

5. There is no mention made of conjunctival adhesions in this discussion, and it is notable that with the technique described the author had no such complication in any of these 500 consecutive cases."

From the material on contact toxicology, it is concluded that aerosol concentrations which produce in the eyes pH values of 11 or more for a few seconds or longer have a significant risk of producing eye damage. Little is known about the effect of lower concentrations (lower pH values) for durations longer than 1 minute. For splashes into the eye of very concentrated solutions (pH greater than 14), only 1 case in 123 reported by McLaughlin sustained a permanent decrease in visual acuity, and this case went untreated for 48 hours; the treatment being irrigation of the eye followed by abrasive removal of the damaged corneal tissue. (The eye fluids reduce the concentration at the surface of the eye to nearly that of tears within a few minutes, however, in the event of a splash or other very short exposure.) Calculations concerning pH values in fluids at the surface of the eye are presented in Appendix C; the preliminary model predicts pH values greater than 11 under some circumstances. These calculations have been performed on the basis of several pessimistic assumptions, and, in the next section, further work is recommended to formulate a more realistic model.

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V. RESEARCH NEEDS

Assessment of the potential hazard of airborne products from a sodium fire will require knowledge about their source, their transmission, and their effects on receptors. The following areas need further research:

1. The chemical transformations which occur to such aerosols as they are transported; in particular, how chemical composition will depend upon temperature, humidity, particle size distribution and concentration, time, and turbulence.

2. The particle size distribution which will result from those chemical transformations, with emphasis on the effect of humidity.

3. The effect the chemical transformations and changes in particle size distribution will have on scavenging of the aerosol by precipitation (rain, sleet, snow).

4. The interactive influence on the above of the potential presence of a wide range of fission product and fuel aerosols.

5. The dose-response relationships for humans. This may involve formulating mathematical model for the effects on humans and on animals, then testing the model through experimentation on animals or human volunteers.

6. The long-term fate of sodium aerosols released to the environment, including dispersal, possible decontamination measures, and potential long-term health effects.

APPENDIX A

CHEMICAL EFFECTS WITH RESPECT TO AEROSOL PARTICLE SIZE

CHANGE IN PARTICLE SIZE: SODIUM HYDROXIDE TO SODIUM CARBONATE

The particle size parameter usually used to characterize respirability of aerosols is the aerodynamic diameter, which is the diameter of a unit (= water) density sphere having the same settling velocity in still air at standard temperature and pressure as the particle. For spherical particles larger than a micrometer (μm) or so, this is the product of the particle diameter and the square root of its specific gravity.

Dry sodium hydroxide particles will react with water to form sodium hydroxide solutions at humidities above their transition humidity, given by Clough and Garland(4) as 35% RH. The droplets of NaOH solution react with carbon dioxide in the air to form solid sodium carbonate decahydrate, which will only become droplets at much higher humidities (95% RH). One gram of NaOH has 1/40 moles of sodium and occupies 0.496 cm^3. It will react and hydrate to form 1/80 moles of $Na_2CO_3 \cdot 10H_2O$ (sodium carbonate decahydrate, washing soda) which will weigh 3.5 g and occupy 2.48 cm^3. This is a factor of 5.0 increase in volume, a factor of 1.71 increase in particle diameter for a sphere, a factor of 0.68 decrease in density (densities from Weast(31)).

Ignoring the Cunningham slip correction (which becomes less than a few percent for particles having diameters larger than 2 μm), the change in aerodynamic diameter will be an increase by a factor of 1.41 (or 41 per cent) for the change from a sodium hydroxide particle to a sodium carbonate decahydrate particle. This would be expected to shift some of the respirable particles to the non-respirable range, but the impact on dose could only be ascertained from a detailed knowledge of the original sodium hydroxide aerosol size distribution and the respiratory tract retention as a function of particle diameter.

CHANGE IN PARTICLE SIZE: SODIUM HYDROXIDE AND HUMIDITY

Sodium hydroxide (NaOH) is quite hygroscopic. The behavior of hygroscopic salt particles has been described by Orr, et al.(32): as the humidity is increased from zero percent relative humidity (0% RH), the surface of the particle begins to be covered by water molecules. At a "transition humidity" (which is almost independent of particle size for particles larger than a few tenths micrometers) the particle is covered by several molecular layers and starts to dissolve, accumulating more water and becoming a droplet. The droplet will have a definite equilibrium size for a given humidity, having started with a certain dry size. If the humidity is decreased, this droplet will remain liquid down to a lower humidity than the transition humidity; below this lower humidity, it becomes a solid.

Clough and Garland(4) calculated the change in droplet radius (r_d) as a function of relative humidity (H) by starting with the well-known Kelvin equation:

$$H/H' = \exp\left(2\gamma_d M_w/\rho_d R T r_d\right) \tag{A.1}$$

which is implicit for the droplet radius. The various terms are defined as follows:

H = relative humidity, fractional

H' = humidity which would be in equilibrium with a droplet of infinite radius (a plane surface)

γ_d = droplet surface tension, dy/cm

M_w = molecular weight of water, g/mole

ρ_d = density of droplet, g/cm^3

R = universal gas constant, erg/mole - °K

T = absolute temperature, °K

The problem of determining r_d is made more difficult by the dependence of the surface tension and the droplet density on the concentration of the solution, which in turn depends upon the ratio of the droplet radius to the original dry particle radius. From the results of calculations presented by Clough and Garland, we have adapted that which follows, showing the ratio of the droplet radius to the dry particle radius at the indicated humidity:

Particle Radius (μm)	Humidity 70% RH	 90% RH	 99% RH
0.3	1.4	1.8	3.7
1.0	1.4	1.8	3.8
3.0	1.4	1.8	3.7
10	1.4	1.8	4.0

We conclude that the effect of changes in humidity will be to change the particle size by essentially the same proportionality factor for a particular humidity for a size range from a few tenths micrometers to a few tens of micrometers. Thus, the aerosol will have its mass median shifted by these factors, but its relative standard deviation (or geometric standard deviation) will be almost unchanged.

Clough and Garland used a computer to solve the Kelvin equation for droplet equilibrium with its vapor. This equation gives the droplet size implicitly. Neiburger and Wurtele presented the following approximate solution(33):

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$$d_d/d_p = A/(1-H)^{1/3} \tag{A.2}$$

in which A is a function of solvent and solute properties, d_d/d_p is the ratio of droplet diameter to dry diameter, and H is fractional relative humidity. Using the results reported by Clough and Garland, we found the value of A to be 0.93 for H = 0.70 (70% RH), 0.85 for H = 0.9, and 0.81 for H = 0.99. The formula

$$d_d/d_p = 0.87/(1-H)^{1/3}$$

(A.

fits the results well (see Figure A.1) and gives $d_d/d_p = 1$ at 35% RH, where the dry particle is just about to become a droplet. It is much easier to use this latter formula than to try to solve the Kelvin equation each time a new humidity is investigated.

The aerodynamic diameter will be changed (neglecting the Cunningham correction factor) by the ratio of the wet droplet diameter to dry particle diameter and by the square root of the ratio of the droplet density to the particle density. The droplet density is:*

$$\frac{\rho_d}{\rho_w} \doteq 0.5(1+(1+4.4(d_p/d_d)^3\rho_p)^{1/2})$$

(A.4

for dry particle diameter d_p and water density ρ_w. The density of water is 1.00 g/cm^3 and the density of NaOH in the dry form is 2.13 g/cm^3, so that we have the following density change factors to apply:

	70% RH	90% RH	99% RH
$(\rho_d/\rho_p)^{1/2}$	.88	.78	.70

Taking both the particle size change as it becomes a droplet and the particle density change, we obtain the following conversion factors by which the dry particle aerodynamic diameter should be multiplied to obtain the droplet aerodynamic diameter:

	70% RH	90% RH	99% RH
conversion factor	1.2	1.40	2.6

Given the uncertainty in our knowledge of the original aerosol size distribution, this correction may not be significant for humidities less than 90% RH.

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*This is based on data from Weast(31) that fit the equation:

$$\rho_d = \rho_w(1+1.10(\rho_p r_p{}^3/\rho_d d_d{}^3))$$

which is quadratic in ρ_d and can be solved analytically.

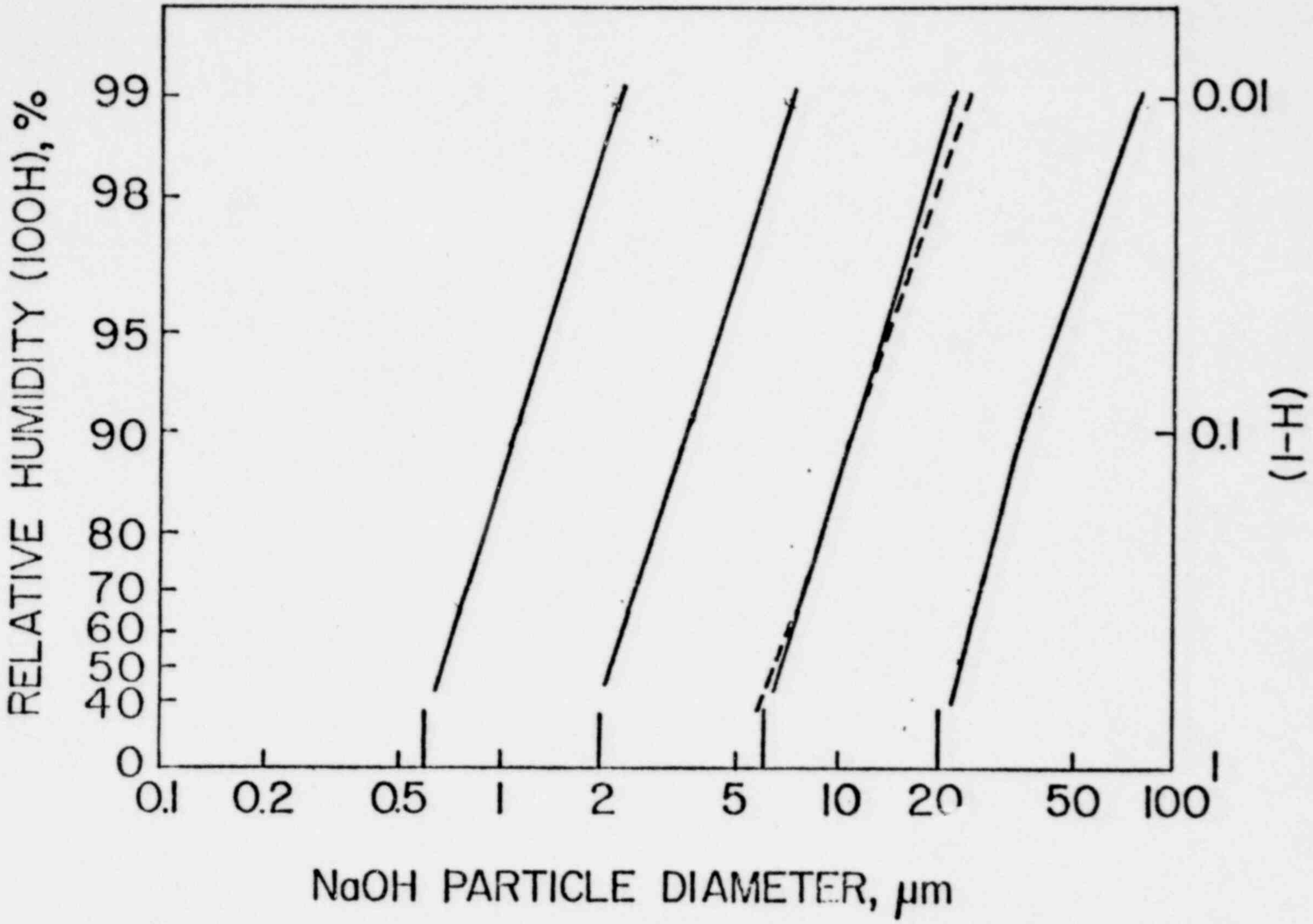

Figure A.1 Relationship Between NaOH Particle Diameter and Relative Humidity. Values interpolated from calculations by Clough and Garland (34): (————————). Values from $d_d/d_p = 0.87/(1-H)^{1/3}$: (-----------).

APPENDIX B

ANALYSIS OF MASS TRANSFER RATES

DIFFUSION OF ATMOSPHERIC CO_2 TO THE PARTICLE SURFACE

Let W be the rate (in moles/s) at which carbon dioxide diffuses to an absorbing sphere, such as an NaOH particle. For particles of diameter d (cm) this mass transfer to the particle is described by the equation(34):

$$W = -k\pi d^2 \, (y_o - y_\infty)/(1 - y_o) \qquad \text{(B.1}$$

in which k is the mass transfer coefficient (mole/cm^2-s) and y is the mole fraction of the carbon dioxide (330 ppm for the atmosphere, average). The subscripts zero and infinity refer respectively to the particle surface and to regions far from the particle; at the particle surface, the CO_2 mole fraction approaches zero due to absorption, and far away the mole fraction is the ambient concentration. Equation (B.1) becomes

$$W = k\pi d^2 \, y_\infty \qquad \text{(B.2}$$

The mass transfer coefficient is given by(34):

$$k = \frac{cD}{d} \, (2.0 + 0.60 \, Re^{1/2} \, Sc^{1/3}) \qquad \text{(B.3)}$$

in which D is the diffusion coefficient (cm^2/s) for carbon dioxide in air, c is the concentration (moles/cm^3) of the air, Re is the Reynolds number at terminal velocity for the falling particle, and Sc is the Schmidt number, $\mu/\rho D$, in which ρ is the gas density and μ is the gas viscosity. Bird, et al.(34) note that if temperature or composition differences between the surface and the medium significantly change the gas characteristics, then Re and Sc should be based upon the average temperature and composition, the "film" value. The Reynolds number at terminal settling velocity for a 20 μm diameter unit density sphere is less than 2×10^{-3} and the value of Sc is 1.0 here, thus the second term in the brackets produces less than a 10 per cent correction which we neglected. Combining the equations for mass transfer yields:

$$W = 2\pi c D d \, y_\infty \qquad \text{(B.4}$$

which means that the rate will be proportional to particle diameter, ignoring a small correction for gas mean free path.

Table B.1 has the rate of diffusion of carbon dioxide to spheres of diameters from 0.5 to 16 μm, the sodium content of such a sphere if it be pure NaOH, and the time it takes enough carbon dioxide to diffuse to the surface to react completely with the sodium. D, ρ, and μ were interpolated for T = 20°C from values given by Bird, et al.(34).

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Table B.1. Minimum Times Required for the Diffusion of Atmospheric CO_2 to Convert all NaOH to Na_2CO_3

Particle diameter (μm)	CO_2 diffusion rate (mole/s)	Dry NaOH particle Na content (moles)	Dry NaOH minimum diffusion time (s)
0.5	0.33×10^{-2}	3.48×10^{-15}	0.0053
1.0	0.66 "	2.78×10^{-14}	0.021
2.0	1.32 "	2.23×10^{-13}	0.084
4.0	2.64 "	1.78×10^{-12}	0.34
8.0	5.28 "	1.42×10^{-11}	1.35
16.0	10.56 "	1.14×10^{-10}	5.40
	(proportional to diameter)	(proportional to volume)	(proportional to area)

To calculate the time for enough carbon dioxide to diffuse to the surface of a sodium hydroxide droplet, one can determine the droplet size (d_d) from an expression we developed, patterned after that of Neiburger and Wurtele(34), which correlates the computed results of Clough and Garland(4):

$$d_d/d_p = 0.87/(1-H)^{1/3} \text{ for } 0.35 < H < 0.99 \qquad \text{(B.5}$$

then interpolate from Table B.1 to obtain the carbon dioxide diffusion rate (note its linear dependence on diameter). Finally to obtain the amount of CO_2 needed, calculate the moles of Na present and divide by two. A final check must be made to compare the mole concentration of the sodium in the aerosol with the mole fraction of carbon dioxide in the air ($330\text{x}10^{-6}$). At 100 mg/m^3 of NaOH there are about four times as many moles of CO_2 as there are moles of Na in the same volume, thus only about one-eight of the available carbon dioxide will be consumed, and y_∞ will not be very different from 330 ppm, even assuming no carbon dioxide diffuses into the aerosol cloud from without.

DIFFUSION OF A REACTING SPECIES WITHIN THE DROPLET

Bird, et al.(34) presented the following expression for the mass transfer rate W for a species having molar concentration c_s at the surface of a sphere of radius R, diffusing with an effective diffusion constant D* and undergoing a first order reaction:

$$W = -4\pi R D^* c_s \ (1 - z \coth z) \qquad \text{(B.6}$$

where $z = (t_d/t_r)^{1/2}$

$t_d = R^2/D^*$, characteristic diffusion time

t_c = characteristic reaction time.

The characteristic diffusion time for droplets of CO_2 in water can be obtained from the CO_2 diffusivity (ignoring some possible circulation in the droplet), which we calculated to be $1.8\text{x}10^{-5}$ cm^2/s (see reference 35). For particles of R = 10 μm = 10^{-3}/cm, this time is only 0.06 sec; it obviously decreases rapidly with decreasing particle size. Limiting cases for z coth z are (36) $1 + z/3$ for $z \ll 1$ and z for $z \gg 1$, but we lack t_c.

The effect of the reaction is to accelerate the diffusion process(35), so that we can make a lower limit estimate of the diffusion rate from equations developed for the non-reacting case. The solution of the diffusion equation:

$$\partial c/\partial t = D\nabla^2 c$$

in spherical coordinates produces an infinite series of exponential functions the coefficients of which are determined by the boundary conditions. In this case the initial concentration of carbon dioxide in the drop is zero and the surface concentration of carbon dioxide is in question. If diffusion into the droplet is much lower than diffusion to its surface (and the diffusion coefficient in the liquid is four orders of magnitude smaller

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here), then one can assume that the surface of the droplet effectively comes into equilibrium with the gas instantaneously. Without reaction, the concentration at the surface of a droplet (c_1) could be estimated from Henry's law, relating the equilibrium carbon dioxide concentration in the liquid to that in the gas. The various concentrations are related by(35):

$$(\bar{c}-c_1)/(c_o-c_1) = f(D^*t/R^2) = f(t/t_d) \qquad (B.7)$$

in which $\bar{c}$ = average concentration

c_1 = constant surface concentration

c_o = uniform initial concentration

This fractional difference between the mean and the surface concentrations falls to 1×10^{-2} at $(D^*t/R^2) = 0.4$, so that by the time t is equal to t_d, the concentration would be very nearly uniform in the absence of reaction. Diffusion within the droplet should take place (for $R \lesssim 10$ μm) in times much less than a second.

Diffusion to the droplets should provide enough carbon dioxide to neutralize the NaOH droplets in seconds, and the concentration in the liquid droplet surface should diffuse throughout in much less than a second. The limiting factor may be the relationship between the equilibrium concentration of carbon dioxide in the solution compared with that in air. Clough and Garland(4) used the Henry's law constant for the equilibrium between carbon dioxide and water:

$$p_o = H\, c_o \qquad (B.8)$$

in which H is the constant, p_o the carbon dioxide mole fraction in the air, and c_o its equilibrium mole fraction in the liquid. This Henry's law approach leads to the conclusion that the rate of transfer into the droplet will be the rate given above for diffusion, but with c_o given by p_o/H; H is quite large ($\sim10^5$ for gases in water at 20°C). The Henry's law analysis by Clough and Garland led them to conclude this to be the rate-limiting step, delaying conversion to the carbonate for times from 10^{-4} to 10^2s for particles between 0.1 and 10 μm in radius at humidities from 40 to 99% RH.

We have our doubts about this aspect of the analysis by Clough and Garland: the formation of carbonate may be more rapid than they indicate for droplets but slower for dry NaOH particles. In the limit of extremely low hydroxide concentrations, their model of having carbon dioxide dissolve in water then react with the hydroxide might be appropriate, with the equilibrium constant between phases for CO_2 being the Henry's law constant. For concentrated solutions, however, the equilibrium constant which would be more appropriate would be that between carbon dioxide and sodium hydroxide solution with the formation of the carbonate and a net free energy change of -30.4 kcal/mole. This has an equilibrium given by:

$$\frac{[CO_2]\,[NaOH]}{[Na_2CO_3]} = \exp\left[\frac{(-30.4 \times 10^3 \text{cal/mole})}{586 \text{ cal/mole}}\right] \doteq 10^{-23}$$

which means that virtually all the CO_2 influx will be converted to the carbonate (if the rate of reaction is not limiting). Thus, the process will be one of diffusion of CO_2 to the surface, passage through the "skin" of the droplet caused by orientation of molecules at its surface, and reaction to form the carbonate, which will then diffuse away as the hydroxide diffuses to the surface.

Our qualitative statement that the rate of mass transfer will be increased can be made quantitative. The mass transfer flux can be expressed as the product of a mass transfer coefficient k_L and the difference between the concentration in the interior of the liquid and that in the liquid at the surface. For the case in which gaseous species A (carbon dioxide) reacts with liquid species B (sodium hydroxide) essentially irreversibly as:

$$A + B = C \qquad (B.9)$$

the ratio of the liquid mass transfer coefficient k_L to what it would be without the reaction is(35):

$$k_L/k_L^o = (1 + (D_B/D_A)(B_o/vA_i))\,(D_A/D_B)^{1/2}$$ (B.1

in which expression the diffusivity of A in the gas is D_A and of B in the liquid is D_B; the initial concentration of the reactant B is B_o; the gas concentration at the interface is A_i; the number of moles of B which react with A is v. We calculated the diffusivity for NaOH from equations provided by Perry and Chilton(35) for very dilute electrolyte solutions (thus introducing some error). For $D_B \doteq 2 \times 10^{-5}$ cm²/s, $D_A = 0.16$ cm²/s, $B_o = 1M$, $v = 2$, $A_i = 3.3 \times 10^{-4}$, the result was that the expression $k_L/k_L^o \sim 100$, meaning a very large increase in the transfer in the liquid due to the reaction. This would lower the times predicted by Clough and Garland(4) to be needed for the formation of sodium carbonate.

Our analysis, in contrast to that of Clough and Garland(4), suggests that the rate-limiting step for the hydroxide droplets to become the less toxic carbonate will be the diffusion of gas to the surface of the droplets (see Table B.1) or the diffusion of the gas through the solid carbonate (at < 35% RH), as discussed next.

DIFFUSION OF CARBON DIOXIDE INTO SOLID PARTICLES

If the humidity is below 35% RH, a solid NaOH particle will remain a solid rather than become a droplet(4). In this case, the reaction with CO_2 will occur first on the surface, forming sodium carbonate and water, the latter evaporating. For further reaction to occur, the carbon dioxide will have to diffuse through the solid carbonate layer to react with the hydroxide below. Bird, et al.(34) showed some examples for diffusivities of various substances from which one can note that diffusivity at room temperature and pressure is ∿ 10^{-1} cm²/s for gases in gases, ∿ 10^{-5} for gases in liquids, and

$\sim 10^{-9}$ for gases in solids, thus the diffusion time for a 20 µm diameter solid particle of NaOH might be on the order of an hour, much longer than that for a droplet. Because this characteristic diffusion time changes with the square of the radius, it should be shorter than a minute for respirable dry NaOH particles (d $\overline{<}$ 3 µm). The reaction rate in the solid form remains a question. Experimentation should indicate the rate at which these dry particles are transformed to carbonate.

For a droplet of NaOH, the reaction to form the carbonate might leave the droplet locally saturated at the surface, possibly forming a carbonate film which would impede diffusion of the carbon dioxide to the interior hydroxide. This seems unlikely, however, because the droplet will have circulation of liquid in its interior(37), the diffusion times for the water and carbonate molecules are short, and even supersaturation may not immediately produce a solid phase, so that one expects the droplet to remain a droplet at least until nearly wholly saturated with carbonate (as the decahydrate form).

APPENDIX C

PRELIMINARY MODEL OF NaOH AEROSOL DEPOSITION ON THE EYE AND THE RESULTING pH OF TEAR FLUID

INTRODUCTION

In this appendix is presented a preliminary model for determining the pH of the fluid (tears) in contact with the surface of the eye while NaOH aerosol particles are striking that surface. Several pessimistic assumptions are made which lead to over-estimating the pH of the fluid. The model is useful in that it shows roughly the relationship between the pH (which is related to eye damage) and the other parameters of the problem; it is useful, too, in that it might have ruled out high pH as a potential problem, although this was not the case for the parameter values used.

DEPOSITION ON THE EYE

In this section we calculate the approximate deposition of particulates onto the eyeball. The major mechanisms considered are: diffusion, interception, impaction, sedimentation. We seek the effective migration velocity to the eye, v_m, so that the rate of mass transfer, $\dot{M}$ (g/s), can be determined from the equation:

$$\dot{M} = c\, v_m\, A_e \qquad \text{(C.1}$$

in which $\dot{M}$ is the product of the concentration (c), the effective migration velocity (v_m) and the cross-sectional area (A_e) of the eye normal to the air flow.

Air velocities are of the order of miles per hour; 10 mph corresponds to 447 cm/s.

Diffusion is characterized by effective velocities on the order of the diffusivity of the particles divided by the significant obstacle dimension; for particles larger than 0.1 μm the diffusivities divided by the dimension of the eye (a 2.4 cm diameter sphere, approximately) give effective migration velocities less than 10^{-5} cm/s.

Interception is approximately proportional to the gas velocity times the ratio of the particle size to the obstacle size, and this ratio will be $\sim 10^{-2}$ or less for particles smaller than 200 μm diameter.

Settling velocities for unit density spheres in air at normal temperature and pressure are 0.08, 0.3, 7, cm/s for particles 5, 10, and 50 μg in diameter, but for those particles for which settling velocity is comparable to air velocity, sedimentation from the air will tend to remove them before they reach the subject.

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Virtually all the aerosol mass is expected to be in particles in the size range from a few tenths to a few tens of micrometers, in which range impaction will generally dominate.

"Impaction" is used to describe the situation in which an airborne particle is captured by an obstacle in the flow due to the inability of the particle to follow the gas streamlines because of its inertia. For particles of a single size, in a flow of uniform velocity (v) far away from the obstacle, the rate at which particle mass will be captured by the obstacle is

$$\dot{M} = c\ v\ A_p\ \eta \tag{C.2}$$

in which η is the impaction collection efficiency of the target (including material caught at front, sides, and rear of the target) and A_p is the cross-sectional area of the target perpendicular to the flow.

In this study an empirical expression for impaction efficiency has been used which has been obtained from measurements with spheres in uniform flow. One expects this to overestimate this form of collection a few ways: first, the flow change of direction is not as abrupt as this assumes (a case could be made for using the head diameter as a more appropriate dimension, which would be greatly lower collection) and second, the efficiencies include particles caught behind the obstacle, whereas none would be captured by the rear side of the eye; third, the eyeball is only partially exposed (due to eyelids); fourth, the eyelashes may help form a stagnation zone for the gas, a region of low flow, another factor inhibiting impaction.

To calculate the collection efficiency of the eyeball due to impaction, one must calculate the dimensionless impaction parameter, ψ, then use an expression which is a function of that parameter that has been found to correlate experimental data successfully. The impaction parameter is the distance the particle would travel in still air before stopping, given an initial velocity v, divided by a dimension which is characteristic of the obstacle. Here,

$$\psi = vC\ \rho_p\ d_p^{\,2}/18\mu\ D_e \tag{C.3}$$

where C = Cunningham slip correction for gas mean free path

ρ_p = particle density, g/cm^3

v = velocity of the gas far from the obstacle (the free steam velocity), cm/s

d_p = particle diameter, cm

D_E = obstacle diameter, cm

μ = gas viscosity, g/cm-s (poise)

The Cunningham correction at room temperature and pressure is $1 + (0.16 \times 10^{-4}\ \mathrm{cm}/d_p)$. The air viscosity ($\mu$) is 1.81×10^{-4} poise. For the diameter of the eyeball, we have used 2.4 cm.

Values were calculated for the impaction parameter, ψ, and for the collection efficiency of the eye due to impaction, η. The latter was determined using the empirical expression:

$$\eta = \psi^2/(\psi+0.32)^2 \qquad (C.4)$$

The predicted collection efficiencies for different velocities and particle aerodynamic diameters ($d_p = d_{ae}$) are shown in Fig. C.1 and C.2. Both gas velocity and particle size strongly affect collection.

DYNAMIC CHEMICAL EQUILIBRIUM

As the NaOH particles strike the liquid surface of the eye, they will dissolve. The eye fluid will have an increasing concentration of NaOH until the rate of addition of NaOH is balanced by the removal of NaOH due to tear flow out of the eye. The rate of such tear flow is in doubt. In Section IV was cited the value for normal flow of 0.5-1.25 g (or cm^3) per day; de Roetth(3) has cited the work of Ahlstrom in which a tube was inserted near the lacrimal gland, with a flow of 4 g/day measured, which was interpreted as having caused sensory stimulation that increased the flow beyond the normal rate. This was the only such study de Roetth found. If the aerosol stimulus induces unusual tear flow, then the resulting pH will be lower than calculated below, where (1/16) cm^3/hr was used as the flow rate value.

Assuming steady flow and perfect mixing in the fluid volume in the eye, the concentration therein would be described by the equation:

$$c = (\dot{M}/\dot{V})(1 - e^{-\dot{V}t/V}) \qquad (C.5)$$

in which c = concentration of NaOH in solution in the tear fluid, $\mathrm{g/cm}^3$

$\dot{M}$ = mass rate of NaOH aerosol into the eye, g/s

$\dot{V}$ = volume rate of tear fluid flow, cm^3/s

V = volume of the fluids in contact with the eye surface, cm^3

V was estimated as 0.006 cm^3 by multiplying the tear thickness (7 μm) times the hemispherical surface area of the eye. If that is the case, the time constant for the approach to equilibrium, $V/\dot{V}$, is 12 minutes. Our experience is that one extra drop of saline solution can be put into an eye and will not be lost when blinked, but a second drop is lost, so that crying, if induced, might increase the value of V up to about that of a drop of water ($\sim 0.05\ \text{cm}^3$), but $\dot{V}$ will also be increased by crying, so the time constant should still be $\sim$ 10 minutes. This means the equilibrium concentration would not be reached except for exposures much longer than this duration.

The pH is the negative of the logarithm of the hydrogen ion concentration (moles/liter); it can also be given in terms of the dissociation constant of water (K_w) and the hydroxyl ion concentration $[OH^-]$:

$$pH = -\log(K_w/[OH^-]) \tag{C.6a}$$

or

$$pH = -\log(3.3 \times 10^{-9}/(\dot{M} + 0.033)) \tag{C.6b}$$

in which we have used $K_w = 1.0 \times 10^{-14}$ for water at 25°C. (The eye fluids will be at a temperature somewhere between that of the body, 37°C, and that of the tear solution, the wet bulb temperature.) It is assumed that all the NaOH dissociates to form OH^-, a reasonable assumption for a strong base except at unusually high concentrations (see Table 5).

By using equation (A.3) for NaOH particle growth with humidity and the equations for deposition and pH given in this appendix, the expected pH values were calculated, given the assumptions stated above.

Figure C.3 shows the expected pH for an aerosol of 1 mg/m^3 concentration NaOH at 50% relative humidity; for one curve, the aerosol is assumed to be monodisperse with a dry diameter of 0.5 µm and for the other curve, to be monodisperse with a dry diameter of 16 µm. For the 0.5 µm aerosol, pH = 11 is not expected until the air velocity reaches 40 mph; for the 16 µm aerosol, this velocity is about a factor of 100 lower.

Figure C.4 shows the expected pH for the tear fluids for the same aerosols at 99 per cent RH. The velocities required for the pH to reach 11 have been cut by a factor of three due to the growth in size of the particles as they absorb moisture.

For 1 mg/m^3 of NaOH aerosol, less than the ACGIH and NIOSH limits for occupational exposure, the calculations show some conditions for which pH of the eye solution using this model would have exceeded 11, the pH at which eye injury has been reported to accelerate to the point of being appreciable after seconds or minutes. This disagreement suggests that further work in this area is needed.

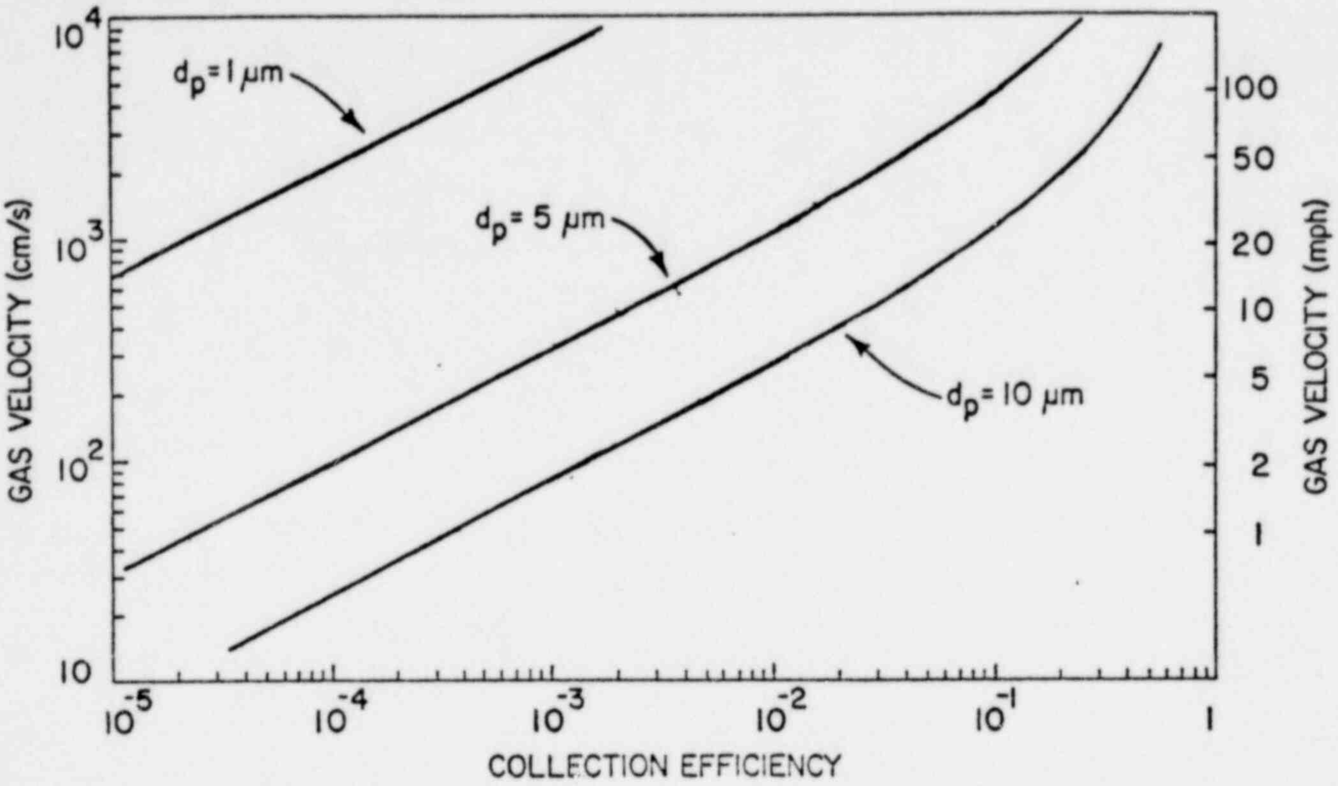

FIGURE C.1 - Predicted collection efficiency of 2.4 cm sphere as a function of gas velocity for particles of aerodynamic diameter 1 μm, 5 μm, and 10 μm.

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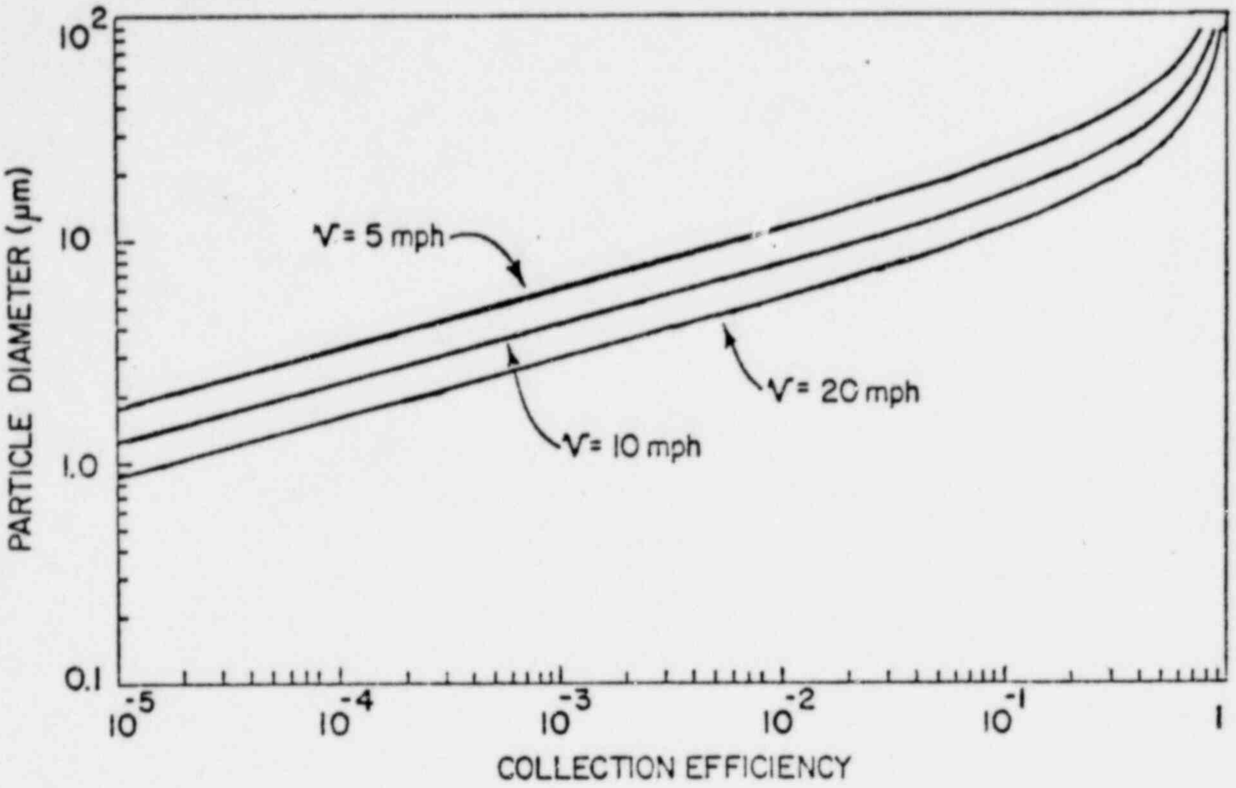

FIGURE C.2 - Predicted collection efficiency of 2.4 cm sphere as a function of particle aerodynamic diameter for gas velocities of 5, 10, 20 mph.

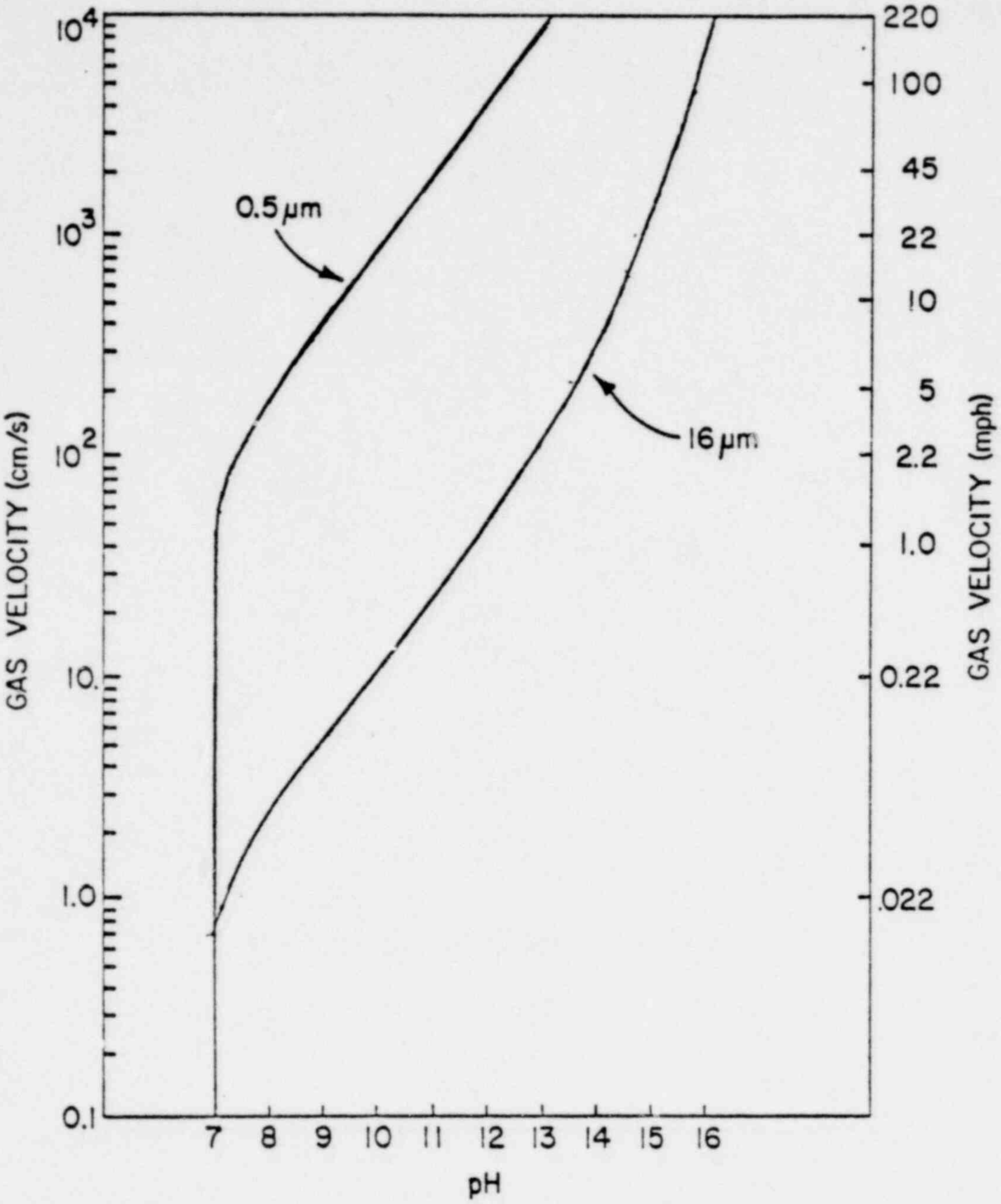

FIGURE C.3 - Predicted relationship between ocular fluid pH and gas velocity for NaOH aerosols of 0.5 μm and 16 μm dry aerodynamic diameter, 1 mg/m^3 in concentration, at 50% RH, using assumptions noted in text.

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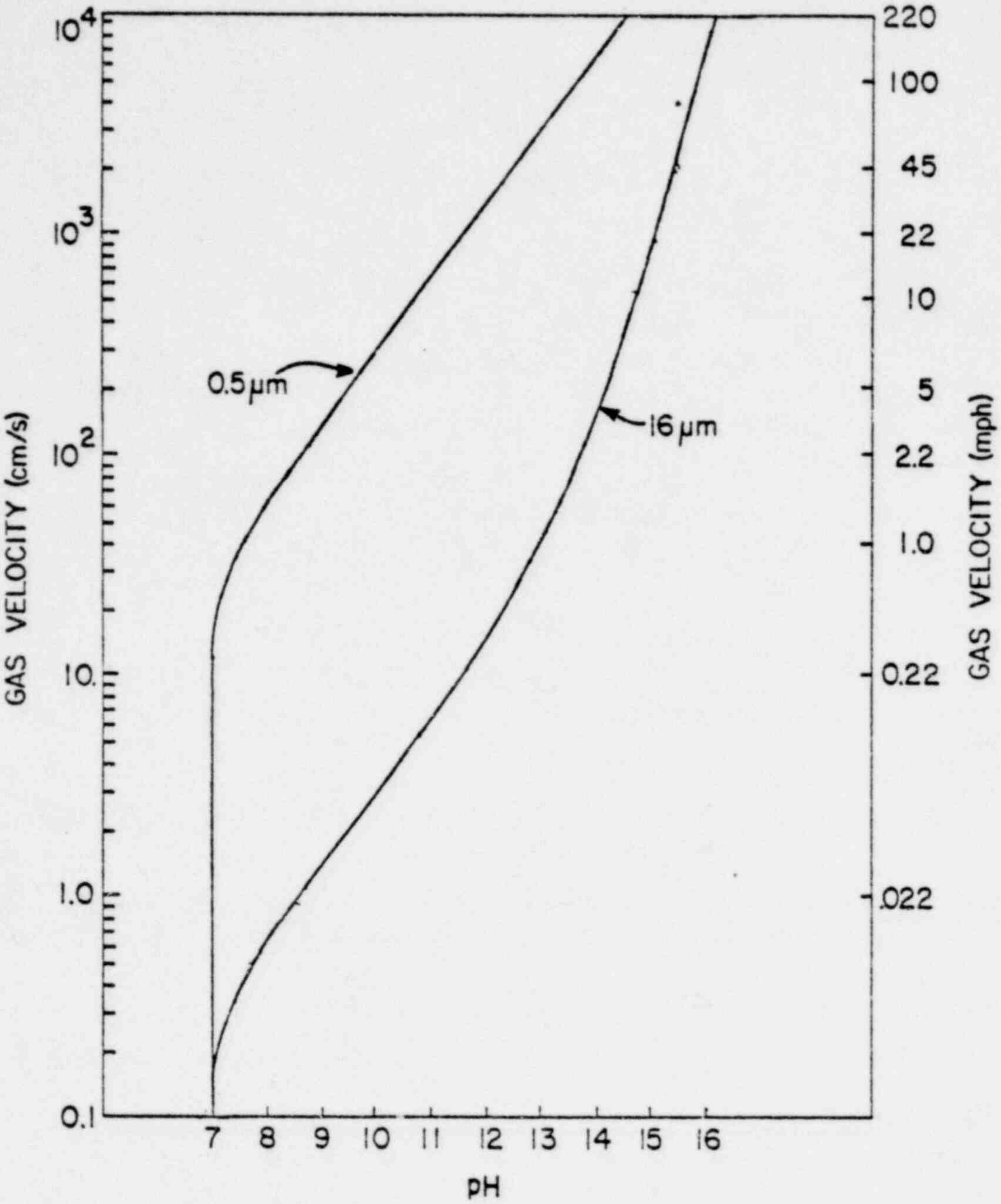

FIGURE C.4 - Predicted relationship between ocular fluid pH and gas velocity for NaOH aerosols of 0.5 μm and 16 μm dry aerodynamic diameter, 1 mg/m^3 in concentration, at 99% RH, using assumptions noted in text.

REFERENCES

1. Hamilton, A. and Hardy, H.L. Industrial Toxicology, 3rd ed., Publishing Sciences Group, Acton, MA, 1974.

2. U.S. Department of Health, Education and Welfare. Criteria for Recommended Standard...Occupational Exposure to Sodium Hydroxide, NIOSH Publication (NIOSH)76-105, 1975.

3. American Conference of Governmental Industrial Hygienists. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1975, ACGIH, Cincinnati, OH, 1975.

4. Clough, W.S. and Garland, J.A. "The Behavior in the Atmosphere of the Aerosol from Sodium Fire," J. Nucl. Energy, 25, 425-435, 1971.

5. Linke, W.F. Solubilities of Inorganic and Metal Organic Compounds, 4th ed., American Chemical Society, 1958.

6. McCoy, H.N. and Smith J. Am. Chem. J., 29, 437, 1903.

7. Murata, M., Naritomi, M., Yoshida, Y. and Kokubu, M. "Behavior of Sodium Aerosol in Atmosphere," J. Nucl. Sci. Technol., 11(2), 65-71, 1974.

8. Amdur, M.O. "Industrial Toxicology," in The Industrial Environment - Its Evaluation and Control, NIOSH, Washington, D.C., 1973.

9. American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values for Substances In Workroom Air, 3rd ed., ACGIH, Cincinnati, OH, 1971.

10. Stokinger, H.E. "Criteria and Procedures for Assessing Toxic Response to Industrial Chemicals," in Permissible Levels of Toxic Substances in the Working Environment, International Labor Office, 1960.

11. Patty, Frank A., ed. Industrial Hygiene and Toxicology, Vol. II, Interscience Publishers, Inc., New York, 1949.

12. Dluhos, M., Sklensky, B. and Vyskocil, J. "Effect of Aerosol Inhalation of Sodium Hydroxide on the Respiratory Tract of Rats," (in Czech), Vnitrni Lekarstvi, 15(1), 38-42, 1969.

13. Vyskocil, J., Tuma, J. and Dluhos, M. "The Effect of Aerosol Inhalations of Sodium Hydroxide on the Elimination of Quartz Dust from the Lungs of Rats," Scripta Medica, 39(1), 25-29, 1966.

14. Elkins, H.B. "Maximum Allowable Concentrations: A Comparison of USSR and USA," *Arch. Env. Hlth.*, *2*, 45, 1961.

15. Ryazanov, V.A. "Sensory Physiology as Basis for Air Quality Standards: Approach of the USSR," *Arch. Env. Hlth.*, *5*, 480, 1962.

16. Patty, Frank A, ed. *Industrial Hygiene and Toxicology*, Vols. I and II, 2nd ed., Interscience Publishers, Inc., New York, 1958 (Vol. I), 1963 (Vol. II).

17. Sax, N.L. *Dangerous Properties of Industrial Materials*, Reinhold, New York, 1968.

18. Caujolle, F., Chanh, P.H., Gelade, M.C., and Patte, J. "Study of the Effects of Alkaline Aerosols on the Isolated Lung of the Guinea Pig," *Med. Pharmacol. Exp.* (Basel), *14*(4), 371-378, 1966.

19. Page, L.B. "Epidemiological Evidence on the Etiology of Human Hypertension and its Possible Prevention," *Am. Heart J.*, *91*(4), 527-534, 1976.

20. Goodman, L.S. and Gilman, A. *Pharmacological Basis of Therapeutics*, 5th ed., MacMillan, New York, 1975.

21. Tadmor, J. "Consequences of an Accidental Release of Sodium to the Environment from an LMFBR," *Nucl. Safety*, *14*, 324-340, 1973.

22. Grant, W.M. *Toxicology of the Eye*, Charles C. Thomas, Springfield, IL, 1962.

23. Hughes, W.F. "Alkali Burns of the Eye. I. Review of the Literature and Summary of Present Knowledge," *Arch. Ophthamol.*, *35*, 423-449, 1946.

24. Potts, A.M. and Gonasun, L.M. "Toxicology of the Eye," Chapter 13 in Casarett, L. J. and Doull, J., eds. *Toxicology: The Basic Science of Poisons*, Academic Press, New York, 1975.

25. Moses, R.A., ed. *Adler's Physiology of the Eye*, C.V. Mosby, St. Louis, 1975.

26. Fox, S.L. *Industrial and Occupational Ophthalmology*, Charles C. Thomas, Springfield, IL, 1973.

27. Grant, W.M. and Kern, H.L. "Action of Alkalies on the Corneal Stroma," *Arch. Ophthalmol.*, *54*, 931-939, 1955.

28. Brown, S.I., et al. "Pathogenesis of Ulcers of the Alkali-Burned Cornea," *Arch. Ophthalmol.*, *83*, 205, 1970.

29. Carpenter, C.D. and Smyth, H.F. "Chemical Burns of the Rabbit Cornea," Am. J. Ophthamol., 29, 1363, 1946.

30. McLaughlin, R.S. "Chemical Burns of the Human Cornea," Am. J. Ophthamol., 29, 1355-1362, 1946.

31. Weast, R.C. Handbook of Chemistry and Physics, Chemical Rubber Co., Cleveland, OH, 1970.

32. Orr, C., Jr., Hurd, F.K., and Corbett, W.J. "Aerosol Size and Relative Humidity," J. Coll. Sci., 13, 472-482, 1958.

33. Neiburger, M. and Wurtele, M.G. "On the Nature and Size of Particles in Haze, Fog and Stratus of the Los Angeles Region," Chem. Rev., 44, 321-335, 1949.

34. Bird, R.B., Stewart, W.E. and Lightfoot, E.N. Transport Phenomena, John Wiley and Sons, Inc., New York, 1960.

35. Perry, R.H. and Chilton, C.H., eds. Chemical Engineer's Handbook, 5th ed., McGraw-Hill, New York, 1973.

36. Selby, S.M. CRC Standard Mathematical Tables, 16th ed., Chemical Rubber Co., Cleveland, OH, 1968.

37. Fuchs, N.A. The Mechanics of Aerosols, Pergamon Press - The MacMillan Company, New York, 1964.

38. de Roetth, A., Sr. "Lacrimation in Normal Eyes," Arch. Ophthalmol., 59, 325, 1953.

39. Hughes, G. W. and Anderson, N. R., "Visibility in Sodium Fume," IAEA Working Group on Fast Reactors (1971).

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