

**ENVIRONMENTAL PROTECTION
AGENCY**

-42036; TSH-FRL 2382-1]

**4,4'-Methylenedianiline; Response to
the Interagency Testing Committee**
AGENCY: Environmental Protection
Agency (EPA).

ACTION: Notice.

SUMMARY: This notice is EPA's response to the Interagency Testing Committee's recommendation that EPA consider requiring health and environmental effects testing of 4,4'-Methylenedianiline (MDA) under section 4(a) of the Toxic Substances Control Act. EPA is not initiating rulemaking at this time under section 4(a) to require testing of MDA for health effects because: (1) EPA has received data from a recently completed National Toxicology Program (NTP) 2-year carcinogenicity study which show MDA to be a carcinogen in both rats and mice, and (2) EPA has initiated evaluation of the need to control exposure to MDA on the basis of the NTP test data and does not believe that data obtained from testing for other health effects are likely to significantly change the regulatory decisions that will be based on the NTP data. EPA is not initiating rulemaking at this time to require epidemiological studies of MDA because a suitable study population has not been identified. EPA is not initiating rulemaking for environmental effects testing because MDA is not anticipated to enter the environment in substantial quantities, and if it does enter the environment it is not expected to persist sufficiently to attain levels likely to lead to toxicity.

FOR FURTHER INFORMATION CONTACT:
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SUPPLEMENTARY INFORMATION:
I. Background

Section 4(a) of the Toxic Substance Control Act (TSCA) (Pub. L. 94-469, 90 Stat. 2003 *et seq.*, 15 U.S.C. 2601 *et seq.*) authorizes EPA to promulgate regulations requiring testing of chemical substances and mixtures to develop data relevant to determining the risks that such chemicals may present to health and the environment.

Section 4(e) of TSCA established an Interagency Testing Committee (ITC) to recommend a list of chemicals for EPA

to consider for promulgation of testing rules under section 4(a) of TSCA. The ITC may designate up to 50 of its recommendations at any one time for priority consideration.

The ITC designated 4,4'-methylenedianiline (MDA) for priority consideration in its Fourth Report published in the *Federal Register* of June 1, 1979 (44 FR 31866). It recommended that MDA be considered for testing for carcinogenicity, mutagenicity, teratogenicity, other chronic effects, environmental effects and epidemiology. The ITC's recommendations were based upon: (1) High production levels of MDA; (2) a National Institute for Occupational Safety and Health (NIOSH) estimate of 5,000 people potentially exposed to MDA in the workplace; (3) toxicological effects in animals, including indications of tumorigenic potential, (4) carcinogenic activity of structurally similar compounds, mutagenic activity in two strains of *Salmonella*, teratogenic effects on chicks, and retinotoxic effects on cats, (5) liver toxicity to humans, (6) development of contact dermatitis by humans working with MDA, (7) sensitivity of *Daphnia* to MDA and lack of other environmental effects data, and (8) potential widespread environmental exposure to MDA (Ref. 21).

This notice provides EPA's response to the ITC's designation of MDA for testing as required by TSCA section 4(e).

II. Decision Not To Initiate Rulemaking

EPA has decided not to initiate rulemaking at this time to require testing of MDA for health effects under section 4 of TSCA because the results of a recently completed 2-year National Toxicology Program (NTP) carcinogenesis bioassay indicate MDA is a carcinogen in both rats and mice and provide sufficient information to assess the carcinogenic risk of MDA. Because the potential carcinogenic risk to humans exposed to MDA is projected to be significant, EPA believes that an exposure level that is acceptable for control of the carcinogenicity risk should provide an acceptable margin of safety for the other health effects of concern listed by the ITC and that no significant additional regulatory information will be gained from requiring further testing for mutagenicity, teratogenicity, or other chronic effects.

On the basis of existing data on MDA's carcinogenicity and indications of potentially significant cancer risks to exposed workers, EPA and the Occupational Safety and Health Administration (OSHA) are exploring

the type of regulatory action that might be taken to provide protection against the risk of cancer from exposure to MDA. EPA is conducting its review of the control of MDA exposure under the accelerated schedule prescribed by section 4(f) of TSCA. The Agency's designation of MDA for such priority consideration was published in the *Federal Register* of April 27, 1983 (48 FR 19078).

EPA is not initiating rulemaking at this time to require epidemiologic studies of MDA because a suitable study population has not been identified. EPA has decided not to initiate rulemaking for environmental effects because: (1) There are no data that document MDA's presence in the environment, (2) if any release occurs it is predicted to be low, and (3) MDA is not predicted to persist in the environment so as to pose a threat to aquatic or terrestrial species if the release does occur.

A. Release and Exposure

From 200 to 400 million pounds of MDA is produced annually. Approximately 90 percent of the animal production is used as an on-site intermediate in the manufacture of methylenediphenyl diisocyanate (MDI), which is used to manufacture rigid polyurethane foams. About 9 percent of the total MDA is transferred to other locations for MDI production (Ref. 22). The remaining MDA (about 1 percent, or 2 to 4 million pounds annually) is purified and used as an intermediate in the manufacture of specialty products such as epoxy resins, a corrosive preventive, a footwear antioxidant, and the chemical *trans, trans*-bis(*para*-aminocyclohexyl) methane (PACM) which is in turn used in the manufacture of elastomeric fibers (Refs. 22, 28).

Human exposure to MDA appears to be principally in the workplace as a result of uses other than MDI manufacture. In 1976, NIOSH estimated worker exposure to be 2500 people (Ref. 30). In 1979, the same agency (Ref. 31) estimated 5000 workers were exposed to MDA. NIOSH (Ref. 36) currently estimates 12,000-13,000 people may be exposed to MDA in the workplace. The NIOSH estimates are generated for the use of MDA in the fabrication of finished products in non-captive use. The Chemical Manufacturers Association (CMA), on the other hand, estimates that approximately 600 people are potentially exposed to MDA in the workplace during the onsite manufacture as an MDI intermediate (Ref. 3).

In 1980, The American Conference of Government Industrial Hygienists (ACGIH) recommended a Threshold Limit Value (TLV) of 0.1 ppm (0.8 mg/m³) (8-hour time-weighted average) and a Short Term Exposure Limit (STEL) of 0.5 ppm (4 mg/m³) for MDA (Ref. 1). TLV and STEL are nonenforceable recommendations for protection of workers in the workplace.

Data from industrial monitoring studies indicate that airborne concentrations of MDA can be as high as 3.8 ppm during the transfer of molten MDA and during grinding and packaging operations (Ref. 1). With proper housekeeping and good work practices, MDA levels have been controlled to ambient levels as low as 0.0064 ppm during the above operations (Ref. 13).

Except for data submitted under TSCA section 8(a) (Ref. 29) which indicate small quantities of MDA were released into the environment in 1981 to unspecified environmental compartments, there are no data that show any release of MDA to the environment. Mathematical modeling predicts that MDA's bioconcentration factor (log BCF) would range from 1-16, its octanol-water partition coefficient (log Kow) from 1.76-2.52 and its organic carbon distribution coefficient (log Koc) from 1.79-2.62 (Ref. 26). These calculated values suggest that uptake and subsequent concentration by the biota would have little impact on the fate of MDA in the environment.

The Agency's conclusion that MDA has little potential for general population exposure is based upon data on the behavior in landfill situations of other monomers used in polyurethane manufacture, data on the behavior of MDA when subjected to treatment in manufacturing effluent, and on the results of mathematical modeling. There are no monitoring data available showing MDA in the environment; however, indirect negative evidence is provided in an EPA-sponsored study designed to detect pollutants in surface waters. Two hundred four water samples from 14 heavily industrialized river basins were collected (Ref. 37). MDA-producing plant sites were included in the water systems which were sampled. In control experiments, amines could be detected reproducibly at levels as low as 50 ppb. No aromatic amines were detected in the ambient water samples.

Once MDA is converted into polyurethane materials, there is very little likelihood that MDA will be released into the environment from the plastics in significant amounts. An example of the behavior of aromatic amine monomers used as starting

materials in the manufacture of plastics was submitted in response to EPA's ANPR on phenylenediamines (Ref. 24). The International Isocyanate Institute (Ref. 25) submitted the results of a research effort to determine whether ether-based polyurethane flexible foams would biodegrade under the conditions of sanitary landfills and whether 2,4- and 2,6-toluenediamines (TDA) would be released. Polyurethane foam made with ¹⁴C-labeled toluenediisocyanates was subjected to three experimental media of different bacterial activity for three months. The sanitary fill medium and the refuse compost medium were subjected to temperatures of 22°C and 50°C. At 22°C no TDA could be detected and no release of ¹⁴CO₂ was identified from any experiments done with sanitary fill medium, but after three months at 50°C, 0.04 percent of the ¹⁴C-tagged starting activity in foam extracts was identified as 2,4- and 2,6-TDA. In refuse compost medium and parabrown earth medium, no detectable TDA was formed, but at 22°C and at 50°C, 0.01 percent and 0.1 percent of the starting activity of the labeled foam was detectable as ¹⁴CO₂. The paper concludes that polyurethane is very resistant to microbial degradation. The Agency believes that polyurethanes based on MDA could be expected to behave in a manner similar to those based on TDA. Therefore, very little regeneration and release of MDA would be expected in landfill situations. Any MDA released is expected to be degraded chemically or microbially as suggested by the occurrence of radiolabeled carbon primarily as CO₂ rather than as TDA in the experiment discussed above.

The potential mode of release of MDA into the aquatic environment is in effluent from MDA manufacturing plants or from the user plants. The Bendix Corporation sponsored a study that was designed to determine the most efficient method for removing MDA from the plant's effluent (Ref. 32). Treatment of the waste water from the treatment plant with activated charcoal filters or sodium nitrite reduced effluent concentrations of MDA from 62.6 mg/L to less than the detection limit of 1.0 mg/L. No other data were located which indicate that other MDA manufacturers or users treat their waste effluent specifically to remove MDA.

Data submitted in response to the TSCA section 8(a) (Ref. 29) rule indicate that only small quantities of MDA were released into the environment in 1981 into unspecified environmental compartments during the manufacturing process. The Agency is also aware that MDA may be present in MDI

manufacturing plant waste streams which enter these plants' waste treatment facilities. Based upon existing data for water solubility (1000 mg/L), melting point (91-92°C), boiling point (398-399°C) and heat of vaporization (22.8 Kcal/mole) and estimates of equilibrium constants and second-order kinetic rate data based on data for structurally related compounds (toluene and aniline), maximum exposure levels of MDA under normal conditions are estimated to range from 10⁻¹-10⁻⁴ mg/L in the water column of the river systems receiving effluent from MDA manufacturing plants. MDA levels (ug/g) estimated for the sediment of these aquatic ecosystems, by EPA's EXAMS modeling system were of the same order of magnitude as predicted for the water column. The EXAMS model predicts that MDA would persist in the river systems 0.2 to 6.5 days ("best case-worst case") and that oxidation would have the greatest impact on MDA in these environments (Ref. 26). MDA is therefore unlikely to be found in the environment at high enough concentrations to lead to concern.

B. Health Effects

The NTP carcinogenesis bioassay (Ref. 16) subjected groups of 50 F344/N rats and 50 B6C3F1/N mice of each sex to 150 or 300 ppm 4,4'-methylene dianiline dihydrochloride (MDA.2HCl) in the drinking water for 103 weeks. Concurrent control groups of 50 rats and 50 mice of each sex received drinking water adjusted with 0.1 N HCl to the pH of the 300 ppm formulation. Under the conditions of the NTP bioassay, MDA.2HCl caused statistically significant (P<0.05) increased incidences of thyroid follicular-cell carcinomas in male rats, follicular-cell adenomas in female rats and mice of each sex, C-cell adenomas in female rats, neoplastic nodules in the livers of male rats, hepatocellular carcinomas in mice of each sex, malignant lymphomas in female mice, and adrenal pheochromocytomas in male mice. In addition, the appearance of rare bile duct adenomas in male rats and ovarian granulosa-cell tumors and urinary bladder transitional-cell papillomas in female rats may also have been related to the administration of MDA.2HCl. All of the tumors except for the hepatocellular carcinomas in male mice were increased in a dose-related fashion.

EPA has concluded that the data from the NTP bioassay are sufficient to characterize the oncogenic potential of MDA in rats and mice and, therefore,

further oncogenicity testing is unnecessary.

Teratogenic effects data for MDA include data on histopathological and cytogenetic effects on laboratory animals and follow-up observations on workers exposed to MDA. Female beagle dogs fed 70 mg/day, 3 times/week, for 3-7 years showed general necrotic changes in liver, kidneys, and lung on post mortem examination (Ref. 6).

MDA has been extensively studied for its mutagenic activity in bacteria. It is a positive mutagen in *Salmonella* strains TA 100 and TA 98 and is non-mutagenic in strains TA 1535, 1537, and 1538 (Refs. 2, 5, 7, 10, 12, 15, 17, 18, 19 and 20).

Reports have also become available which indicate that MDA is not a mutagen in the *Drosophila* sex-linked recessive tests (Ref. 9), mammalian cell transformation assay (Ref. 10), sister chromatid exchange assay and chromosome aberration test (Ref. 8). The *Drosophila* gene mutation and the cytogenetic data on MDA suggest that MDA lacks mutagenic activity in a number of important test systems. Data received from Upjohn (Ref. 20) indicate significant single strand breaks in DNA of V-79 cells exposed to MDA *in vitro*. Parodi et al. (Ref. 17) also reported significant DNA fragmentation induced *in vivo* by MDA administered i.p. to

rats. Taken as a whole, the data suggest that although sufficient research has not yet well identify an upper-level test system in which MDA is mutagenic, it is not a broadly active mutagen.

Only one study has addressed MDA's teratogenic potential. Five mg of MDA in ethanol was injected into the yolk sac of fertile White Leghorn chicken eggs prior to incubation (Ref. 14). Only 30 percent of the MDA-injected eggs hatched. Eggs injected with 5 mg boiled water or 5 mg undiluted ethanol had 95 percent hatching success. This was described as being the same rate as found in non-treated controls. Beak and skeletal abnormalities were observed in the MDA-injected embryos that did not hatch. These data are at best weakly suggestive of potential MDA teratogenicity in other species.

An unpublished report submitted in response to the TSCA section 8(d) health and safety data reporting rule (Ref. 33) presents the results of oral toxicity studies in rats and dogs. Rats were fed MDA for 90 days on diets equivalent to 30 mg/kg-day and 100 mg/kg-day. The dogs were given MDA orally in two dose regimes: (1) 12, 8, or 5 mg/kg-day or (2) 33 doses in 44 days of 2.5, 1.25 or 0.65 mg/kg-day. Gross and microscopic examination of body organs showed some reduction in liver weight and bile duct proliferation at all doses in

the dog and only at the 100 mg/kg-day dose in rats. Hemoglobin levels were reported low in both species, no methemoglobin formation was found, and urinalysis was within the normal range (Ref. 34).

Prechronic tests to the carcinogenicity study by NTP (Ref. 16) showed increases, compared to controls, in bile duct hyperplasia, adenomatous goiter and thyroid follicular cell hyperplasia in male and female rats at 400 ppm and 800 ppm. In mice, the only histopathological effect noticed was an increase in bile duct hyperplasia over controls at 400 ppm in both males and females (Ref. 16).

McGill and Moto (Ref. 13) examined 12 workers exposed to 0.1 ppm MDA during the manufacture of epoxy resins. Within 2 weeks of initial exposure to the MDA, these individuals developed acute hepatitis. Examinations 9 months to 5.5 years after the occurrence of the hepatitis indicated no residual liver toxicity in any of the individuals. Private communications supplied by Dow Chemical Co. for the preparation of an ACGIH documentation of TLV (Ref. 1) were reported to indicate no MDA-attributable morbidity findings at exposure levels ranging from 0.03 to 0.4 ppm during the 26-year period covered by the data. Specific information on the medical criteria used to determine morbidity are not available.

Two of the 84 humans who consumed MDA-contaminated bread in Epping, England (0.26% or 2600 ppm in bread) complained of visual problems in addition to acute hepatitis (Ref. 11). Neither the visual nor the hepatitis-related symptoms were observed in a 2-year follow-up examination.

Retinotoxic effects have also been observed in cats given MDA at various dose levels by stomach tube. One animal received single doses of 25 and 50 mg/kg, another animal received a dose of 200 mg/kg, two animals received 3 doses of 25 mg/kg and 3 doses of 50 mg/kg and one animal received one dose of 100 mg/kg. The MDA administration resulted in blindness of all 5 animals by causing disintegration of the rods and cones accompanied by proliferation of the pigment epithelium and atrophy of the nuclei of the outer granular layer. The doses required to cause blindness were close to the lethal levels of MDA in cats (Ref. 4).

A human skin sensitization study sponsored by Dow concluded that MDA was a skin sensitizer but not a primary irritant nor a fatiguing agent (Ref. 35).

While the MDA data raise the possibility of its posing mutagenic, cytogenetic, teratogenic, and other chronic risks, EPA's analysis indicates significant carcinogenic risk to humans,

a fact which EPA believes must be taken into consideration in reaching an MDA testing decision. In the case of MDA, EPA's judgment is that exposure levels which are predicted to minimize worker risk of cancer are lower than the exposure levels at which teratogenic, reproductive or other effects are likely to occur. EPA concludes, therefore, that any exposures low enough to afford adequate protection against unreasonable risk of cancer would also protect workers against other health risks. EPA has issued a notice under section 4(f) of TSCA (Ref. 23) indicating its concern about carcinogenic risks and its investigation of control options to reduce these risks to workers. In view of this investigation, EPA has concluded that testing for other health effects is not necessary at this time.

C. Environmental Effects.

The ITC recommended MDA for environmental effects testing because of its known toxicity to certain organisms and lack of information on its behavior in the environment.

A single study has been identified which demonstrates the toxicity of MDA to aquatic organisms (Bringmann & Meinick 1964). This study indicates the minimum levels of MDA at which four organisms were unable to function. They are: *Daphnia magna* = 0.25 mg/L; *Pseudomonas fluorescens* (bacterium) = 15 mg/L; *Scenedesmus quadricauda* (alga) = 30 mg/L; and *Microcystis heterostoma* (protozoan) = 124 mg-L. This study does not present LC₅₀ values.

The only information documenting any release of MDA to the environment is from the TSCA section 8(a) (Ref. 29) reports which indicated that only small quantities were released to unidentified environment compartments in 1981. There is no indication which compartments were affected or the amount released to each. However, in light of other information available to EPA and the EXAMS modeling results, the Agency has concluded that only very small amounts of MDA might be released to the environment or persist for any period in the environment.

The only environmental effects data for MDA are for aquatic organisms. Release of MDA to water has not been documented. Any release of MDA to water that might occur would likely be in very low concentrations from waste treatment facilities and would be rapidly diluted. MDA is predicted to oxidize fairly rapidly in water. Thus EPA is unable to conclude from available data that the very limited expected release of MDA to water will present a risk to aquatic organisms. In

the absence of any data on environmental effects outside the aquatic environment and any data on release to nonaquatic compartments, EPA is unable to conclude that there is any risk of adverse environmental effects in those compartments.

D. Epidemiology

The Agency is not proposing epidemiologic studies at this time, because it has been unable to adequately identify the specific manufacturer or processor populations at risk from exposure to MDA.

The Agency realizes that 5,000-13,000 people are potentially exposed during MDA use. The Agency also realizes that epidemiologic studies of user populations, if feasible, would be beneficial in establishing regulatory measures under sections 6 of TSCA and the Occupational Safety and Health Act. The Agency has been able to identify the potential, generic use categories for non-MDI production, and is in the process of identifying specific companies which use MDA in their manufacturing process. When the appropriate cohorts can be identified, epidemiologic studies will be considered.

III. References

- (1) ACCII Documentation of TLV. 1980. 4,4'-Methylenedianiline. pg. 278.
- (2) Andersen M, Binderup M, Keil P, Larsen H, Maxild J. 1980. Mutagenic action of isocyanates used in the production of polyurethanes. *Scand. J. Work Environ. Health* 6:221-226.
- (3) CMA (Chemical Manufacturers Association). 1982. Letter summarizing current information on 4,4'-methylenedianiline. Submitted to U.S. Environmental Protection Agency. November 4, 1982.
- (4) Constatt BS, Hofmann H, Oettel H, Zeiler H. 1966. Retinal changes in cats intoxicated with perioral or percutaneous doses of two chemicals. *Verh. Deut. Ges. Pathol.* 50:429-435.
- (5) Darby TD, Johnson HJ, Northup SJ. 1978. An evaluation of a polyurethane for use as a medical grade plastic. *Toxicol. Appl. Pharmacol.* 46(2):449-453.
- (6) Deichmann WB, MacDonald WE, Coplar M, Woods F, Blum-E. 1978. Di-(4-aminophenyl)methane (MDA) 4-7 year dog feeding study. *Toxicology* 11(2):185-188.
- (7) DuPont. 1976. Unpublished studies on the mutagenic activity of MDA. Submitted to U.S. Environmental Protection Agency, Washington, D.C. by the Chemical Manufacturers Association. September 17, 1982.
- (8) Ho T, Tipton SC, Epler JL. 1978. Cytogenetic effects of m-phenylene diamine (MPDA) and methylene dianiline (MDA) on human leukocytes *in vitro*. *J. Tissue Culture Association* 14:350. Abstract.
- (9) Ho T, Hardigree AA, Larimer FW, Nix CE, Rao TK, Tipton SC, Epler JL. 1979. Comparative mutagenicity study of potentially carcinogenic industrial compounds. *Environmental Mutagenesis* 1:167. Abstract.
- (10) ICI (Imperial Chemical Industries), Central Toxicology Laboratory. 1979. Unpublished studies on the mutagenic activity of MDA. Submitted to U.S. Environmental Protection Agency, Washington, D.C. by the Chemical Manufacturers Association. September 17, 1982.
- (11) Kopelman H, Scheuer PJ, Williams R. 1965. The liver lesion of the Epping Jaundice. *Quart. J. Medicine.* 35(140):553-568.
- (12) Lavoie E, Tulley L, Fow E, Hoffman D. 1979. Mutagenicity of aminophenyl and nitrophenyl ethers, sulfides and disulfides. *Mutation Research* 67: 123-131.
- (13) McGill DB, Moto J. 1974. Industrial outbreak of toxic hepatitis due to methylenedianiline. *New England Journal of Medicine.* 291(6):278-82.
- (14) McLaughlin J, Marliac JP, Verrett MJ, Mutchler MK, Fitzhugh OG. 1963. The injection of chemicals into the yolk sacs of fertile eggs prior to incubation as a toxicity test. *Toxicol. Appl. Pharmacol.* 5:760-771.
- (15) Morgott D, Hooberman B, Sinsheimer J, Cornish H, Weber W. 1982. Mutagenicity of putative metabolites of 4,4'-methylenedianiline and benzidine. *The Toxicologist* 2: 188. Abstract.
- (16) NTP (National Toxicology Program). 1982. NTP technical report on the carcinogenesis bioassay of 4,4'-methylenedianiline dihydrochloride (CAS No. 13552-44-8) in F344/N rats and B6C3F1/N mice (Drinking Water Study). Draft. James Lamb (Chemical Manager).
- (17) Parodi S, Taninger M, Russo P, Pala M, Tamaor M, MontiBradigan C. 1981. DNA damaging activity *in vivo* and bacterial mutagenicity of sixteen aromatic amines and azo-derivatives, as related quantitatively to their carcinogenicity. *Carcinogenesis* 2, 1317-26.
- (18) Rao TK, Dorsey GF, Allen BE, Epler JL. 1982. Mutagenicity of 4,4'-methylenedianiline derivatives in the *Salmonella* histidine reversion assay. *Archives of Toxicology* 49, 185-190.
- (19) Takemura N, Shimizu H. 1978. Mutagenicity of some aromatic amino- and nitro-compounds. *Mutat. Res.* 54(2):256-257.
- (20) Upjohn Company. 1976. Unpublished studies on mutagenicity activity of MDA. Submitted by the Chemical Manufacturers Association to U.S. Environmental Protection Agency, Washington, D.C., September 17, 1982.
- (21) U.S. Environmental Protection Agency (USEPA). 1979. Fourth report of the TSCA Interagency Testing Committee to the Administrator, Environmental Protection Agency. (44 FR 31866).
- (22) Mathtech, Inc. 1982. Level I economic evaluation, 4,4'-methylenedianiline (MDA). Draft Report. Washington, DC: Office of Pesticides and Toxic Substances, U.S. Environmental Protection Agency. Contract 68-01-5884.
- (23) USEPA. 1983. U.S. Environmental Protection Agency. 4,4'-methylenedianiline: initiation of review (48 FR 19078).
- (24) USEPA. 1982. U.S. Environmental Protection Agency. Phenylenediamines: Response to Interagency Testing Committee. (47 FR 973).
- (25) International Isocyanate Institute, Inc. 1979. Bulletin 1. Study on microbial degradation of polyurethane flexible polyether foams under waste disposal conditions. New Canaan, Conn. International Isocyanate Institute, Inc.
- (26) Newman JR, Hendy CD, Hart R, Pollamn C, Altpeter LL, Ou L, Auwarter AG, Rao S. 1981. Environmental Science and Engineering, Inc. Environmental assessment for 4,4'-methylenedianiline (MDA). Test rule support documents, Chapters I, II, IV, V. Final Report. Washington, DC: U.S. Environmental Protection Agency, Contract 68-01-6153.
- (27) Bringmann VG, Meinek F. 1964. *Wassertoxikologische Beurteilung von Industrieabwassern (Aquatic Toxicological study of industrial waste waters)*. *Gesundheits-Ingenieur*, 85:229-260.
- (28) LaFlamme P. 1982. Springborn Laboratories, Inc. Use analysis of 4,4'-methylenedianiline (Non-Isocyanate use). Draft Report. Washington, DC: U.S. Environmental Protection Agency. Contract 68-01-6601.
- (29) USEPA. 1982. U.S. Environmental Protection Agency. Chemical Information Rules; Manufacturers reporting: Preliminary Assessment Information. (47 FR 26992).
- (30) NIOSH. 1976. National Institute for Occupational Safety and Health. Current Intelligence Bulletin, 8, 4,4'-Diaminodiphenylmethane (DDM).
- (31) NIOSH. 1979. National Institute for Occupational Safety and Health. Computer Printout: projected numbers by industry to 4,4'-methylenedianiline. Washington DC: Office of Pesticides and Toxic Substances.
- (32) Young DA, Parker BC. 1978. Removal of methylenedianiline from chemical plant wastewater. Conference on treatment, Houston, TX, 18 Apr. 1978. pp. 28.
- (33) USEPA. 1982. U.S. Environmental Protection Agency. Protection of the Environment Chapter I. Environmental Protection Agency. Subchapter R—Toxic Substance Control. Part 716—health and safety data reporting. Submission of lists and copies of health and safety studies. (47 FR 38780).
- (34) Hodge HC, Downs W, Coze RD, John A, Chen P. 1955. Short term oral toxicity of methylene dianiline in rats and dogs. Submitted by Dow Chemical Company to U.S. Environmental Protection Agency in response to TSCA section 8(d) rule (47 FR 36780).
- (35) Shelanski MV. 1954. Repeated insult patch test study. Shelanski method with Dow Methylene Dianiline (5% in Dowinal 50 B). Submitted by Dow Chemical Company to U.S. Environmental Protection Agency in response to TSCA section 8(d) (47 FR 38780).
- (36) NIOSH. 1980. National Institute for Occupational Safety and Health, Computer Printout: Projected numbers by employment. Washington, DC: Office of Pesticides and Toxic Substances.
- (37) Ewing BB, Chain ESK, Cook JC, Evans CA, Hopke PK, Perkins EC. 1977. Monitoring to detect previously unrecognized pollutants in surface waters. Report. Washington DC: Office of Pesticides and Toxic Substances.

U.S. Environmental Protection Agency.
Contract 68-01-3234.

Public Record

EPA has established a public record for this testing decision (docket number OPTS-42036) which includes:

1. Federal Register notice containing the designation of 4,4'-methylenedianiline to the Priority List and public comments thereon.
2. Communications (public).
 - a. Non-confidential letters.
 - b. Confidential letters (separately held).
 - c. Contact reports of telephone conversations.
 - d. Meeting summaries.
3. Published and unpublished data.

This record, which includes basic information considered by the Agency in developing this decision, is available for inspection in the OPTS Reading Room from 8:00 a.m. to 4:00 p.m. on working days in Rm. E-107, 401 M St., SW., Washington D.C. 20460.

(Sec. 4, 90 Stat. 2003; (15 U.S.C. 2061))

Dated June 30, 1983.

William D. Ruckelshaus,

Administrator.

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