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INTERNATIONAL UNION, UNITED AUTOMOBILE, AEROSPACE & AGRICULTURAL IMPLEMENT WORKERS OF AMERICA—UAW

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September 5, 2002

RECEIVED
SEP 10 2002

Dr. C. W. Jameson
National Toxicology Program
79 Alexander Drive, Room 3118
PO Box 12233
Research Triangle Park, NC 27709

Re: UAW Comments in support of listing diethanolamine as "reasonably anticipated to be carcinogenic to humans."

Dear Dr. Jameson:

Enclosed please find UAW comments in support of listing diethanolamine as "reasonably anticipated" to cause cancer in humans in the **11th Report on Carcinogens.**

These comments are submitted past the deadline. However, the UAW requests the period be reopened to consider the two recently published mortality studies identifying excess mortality from liver cancer among workers likely exposed to diethanolamine through metal working fluids, and recent toxicology studies of showing liver and testicular toxicity in mice given topically applied semi-synthetic metal working fluids likely containing diethanolamine.

I would hope these comments would be provided to the members of the report review committee, and posted to the website.

Sincerely,

Franklin E. Mirer

Franklin E. Mirer, Ph. D.
Director
UAW Health and Safety Department

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opeiu494
Attachment
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cc: NTP File

Comments

National Toxicology Program
Board of Scientific Counselors
Report on Carcinogens Review Sub-Committee

submitted by

Health and Safety Department
International Union, UAW
8000 East Jefferson Avenue
Detroit, MI 48214

regarding

Proposed Listing of Diethanolamine as "Reasonably Anticipated to Cause Cancer in Humans" in the Report on Carcinogens

September 4, 2002

The UAW believes that diethanolamine should be listed as "Reasonably Anticipated to Cause Cancer in Humans." These comments past the deadline are prompted by recent publication of two mortality studies identifying increased mortality from liver cancer among workers likely exposed to diethanolamine in metalworking fluids.

The UAW believes that the recommendation of RG1 not to list diethanolamine constitutes nullification of the rules for listing and abandonment of scientific judgment in the listing process.

Our key points are:

a. Substantial human exposure to diethanolamine occurs through the use of diethanolamine contaminated materials and direct addition to metalworking fluids. **2**
b. Liver tumors from bioassays in mice provide clear evidence for carcinogenicity in males and females **3**
c. Kidney tumors from bioassay in mice provide clear evidence for carcinogenicity in males **4**
d. The carcinogenic potential for diethanolamine in rats was not fully evaluated because of dose limitations arising from study design **5**
e. Increased cancer, liver cancer and liver disease among people exposed to metalworking fluids provides limited and indirect evidence for carcinogenicity of diethanolamine, but still provides evidence which adds to that in the bioassay information **6**

f. The IARC working group conclusions regarding diethanolamine were based on incomplete information ***9***
g. Diethanolamine clearly meets the criteria for listing as "reasonably anticipated to be a human carcinogen," based on bioassay data alone. Human data adds extra weight for this determination ***9***
Appendix ***11***

Full Text

a. Substantial human exposure to diethanolamine occurs through the use of diethanolamine contaminated materials and direct addition to metalworking fluids.

Diethanolamine (DEA) is most frequently encountered by people as it contaminates commercial grades of fatty acid-diethanolamide condensates. These are frequent ingredients in cosmetics, skin and hair care products. Diethanolamine is a plausible contaminant of technical grade triethanolamine, and may be a microbial degradation product of triethanolamine and a hydrolysis product of the condensates as well.

Diethanolamine fatty acid condensates contaminated by DEA, and triethanolamine and its condensates, are currently important and widely used components of soluble, synthetic and semi-synthetic metalworking fluids. Diethanolamine itself was in the past a major intended ingredient of these materials, and despite recent declines in use, is still be present in some formulations.

NIOSH estimates that up to 1,000,000 American workers have occupational exposure to metalworking fluids and lubricants. Mono-, di- and tri-ethanolamines and ethanolamides may also be components of drawing compounds and die lubricants in metal stamping and possibly die casting.

By contrast to presence of DEA in personal care products, where exposure is primarily by skin contact, in the occupational environment DEA exposure includes both skin contact and inhalation of aqueous aerosols containing DEA, and possibly vapor.

Metalworking fluids were reviewed by a recent NIOSH criteria document.[1] NIOSH concluded that:

> "before the mid-1970's, substantial evidence indicated that at least some MWF's were associated with increased cancer risks at

[1] NIOSH, *Criteria for a Recommended Standard for Occupational Exposure to Metalworking Fluids,* DHHS Publication No. 98-102 (1998).

> several organ sites (larynx, rectum, pancreas, skin, scrotum and bladder)... The evidence is equivocal for an association between MWF exposure at several other sites, including stomach, esophagus, lung, prostate, brain, colon and hematopoietic system."

The UAW believes, contrary to NIOSH, that there is clear evidence for an association between exposure to water based MWF and stomach cancer. As discussed below, evidence has since emerged regarding liver toxicity and liver cancer. [Additional evidence has emerged regarding stomach cancer and cancer at other sites as well.]

Metalworking fluids have been addressed by an OSHA Standards Advisory Committee, which recommended a new standard based in part on carcinogenicity information, and by ACGIH for revision of the Threshold Limit Value.

b. Liver tumors from bioassays in mice provide clear evidence for carcinogenicity in males and females

Diethanolamine has actually been bio-assayed four times, once as the pure compound[2], and three other times as an impurity in the matrix of DEA-fatty acid condensates of varying degrees of contamination[3,4,5].

Although NTP does not make a call of a level of evidence in a sex-species experiment based on a particular tumor site, the liver tumors by themselves would have been the basis for a call of "clear evidence" in the two higher dose studies.

NTP noted a remarkably consistent exposure response relationship for liver tumors in female mice across the four studies.

The UAW notes that a statistically significant increase of liver tumors is observable at a lower risk rate attributable to treatment in female mice than male mice, because the female mice have a lower background rate of these tumors and less variability in the control population. The UAW further notes that combining of liver adenomas and carcinomas is scientifically appropriate, consistent with other studies and past practice of NTP.

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[2] NTP, TR-478 Toxicology and Carcinogenesis Studies of Diethanolamine (CAS No. 111-42-2) in F344/N Rats and B6C3F1 Mice (Dermal Studies)
[3] NTP, TR-479 Toxicology and Carcinogenesis Studies of Coconut Oil Acid Diethanolamine Condensate (CAS No. 68603-42-9) in F344/N Rats And B6C3F1 Mice (Dermal Studies)
[4] NTP, TR-480 Toxicology and Carcinogenesis Studies of Lauric Acid Diethanolamine Condensate (CAS NO. 120-40-1) in F344/N Rats and B6C3F1 Mice (Dermal Studies)
[5] NTP, TR-481 Toxicology and Carcinogenesis Studies of Oleic Acid Diethanolamine Condensate (CAS No. 93-83-4) in F344/N Rats and B6C3F1 Mice (Dermal Studies)

Compound	Diethanolamine content	Male Mice Liver	Female Mice Liver
Diethanolamine	100%	Clear evidence	clear evidence
Coconut oil acid diethanolamine condensate	18.2%	Clear Evidence	clear evidence
Lauric acid diethanolamine condensate	0.83%	no evidence	some evidence
Oleic acid diethanolamine condensate	0.19%	no evidence	no evidence

The UAW believes that the more appropriate manner to express results is the mortality adjusted incidence rate, rather than the overall rate. In this regard, we believe the call for the lauric acid diethanolamine condensate should have been "clear evidence" for female mice. The female mice maxed out their adjusted incidence rate at about 80% at the middle dose, and had little room for further exposure response at the high dose. The most appropriate statistical test is the mortality adjusted test for trend, which was marginally significant for adenomas and carcinomas separately, and highly significant for the two combined in this bioassay.

c. Kidney tumors from bioassay in mice provide clear evidence for carcinogenicity in males

We now turn our attention to kidney tumors in male mice. The UAW notes that while kidney tumors in male rats have been denigrated as evidence for carcinogenicity, incorrectly in our view, there is no similar attack on the validity of mouse kidney tumors in either sex.

For the bioassay of diethanolamine in male mice, the life table test for trend was significant, while the Poly-3 test was marginally significant for kidney adenomas, and kidney adenomas and carcinomas combined. This is at least "some evidence," a positive study.

For coconut oil acid diethanolamine condensate, every test for trend and pairwise comparison at the high dose were highly significant for kidney adenomas and adenoma and carcinoma combined, while kidneycarcinomas showed an exposure response trend but were too few in number to achieve statistical significance in any test. This is "clear evidence," although some might argue to downgrade to "some" based on the predominance of adenomas in the

statistical weight. We note that for the lauric acid DEA condensate, which contained the next highest dose, there were two kidney adenomas in the treatment groups and none in the controls. For male mice exposed to oleic acid diethanolamine condensate, there were no adenomas in either treated or control animals. The treated animals were essentially unexposed to diethanolamine. Therefore, the control rate for all studies combined should be the rate for adenomas and carcinomas combined among the 6 groups unexposed to diethanolamine: 3/50, 4/50, 0/50, 0/50, 0/50, 0/50 = 7/300 = 2.3% unadjusted.

The UAW recommends some effort to combine the studies for statistical analysis. However, we have two bioassays which individually provide at least some, and more likely one providing some and one providing clear piece of evidence for carcinogenicity at the kidney site in male mice.

d. The carcinogenic potential for diethanolamine in rats was not fully evaluated because of dose limitations arising from study design

The UAW understands that failure to find evidence for carcinogenicity in a second species raises some concern for a species-specific mechanism that may undermine the evidence of risk to humans. However, the lack of effect in the rat can readily be explained by the lack of sensitivity of the rat bioassay (compared to the mouse) to find an effect, if it were there, because of dose and lifespan.

We will address the bioassay of diethanolamine alone, because this was the highest dose study of the four bioassays and thus the most likely to detect carcinogenicity if it were there. Among female rats, the highest dose tested, 32 mg/kg, was less than the lowest dose tested among male and female mice, which was 40 mg/kg. Thus, carcinogenicity would have been seen only if rats were less resistant than mice. Among male rats, the highest dose tested was 64 mg/kg. Kidney tumors were not significantly elevated in mice at 40 mg/kg, although liver tumors were.

Perhaps most important, the dose in both rats and mice was limited by the amount of skin damage the animals could tolerate. Such an effect is irrelevant to mechanisms of systemic toxicity. This may have limited the dose which the animals could have been given, had exposure been by inhalation or ingestion. Thus, the full carcinogenic potential of diethanolamine in rats has not been tested.

The bioassay in rats was inherently less sensitive than that in mice to observe carcinogenicity because of poor survival. Less than half the male rats survived to the end of the study. Thus, less than half the test animals would have been able to exhibit a late appearing tumor, if it were there, so the sensitivity of the bioassay was compromised. This poor survival also was observed in the control animals.

e. Increased cancer, liver cancer and liver disease among people exposed to metalworking fluids provides limited and indirect evidence for carcinogenicity of diethanolamine, but still provides evidence which adds to that in the bioassay information

The most studied group of people with exposure to diethanolamine are workers exposed to water reduced metalworking fluids. As noted above, metalworking fluids were reviewed by a recent NIOSH criteria document[1] and journal publication:[6]

NIOSH concluded that:

> "before the mid-1970's, substantial evidence indicated that at least some MWF's were associated with increased cancer risks at several organ sites (larynx, rectum, pancreas, skin, scrotum and bladder)... The evidence is equivocal for an association between MWF exposure at several other sites, including stomach, esophagus, lung, prostate, brain, colon and hematopoietic system."

The problem with attributing some of these findings to diethanolamine is the presence of other potential carcinogens in the mix, notably polynuclear aromatic hydrocarbons (from petroleum oils), nitrosamines (notably N-nitroso-diethanolamine, formed from the reaction of nitrite with diethanolamine and therefore evidence of the presence of diethanolamine), formaldehyde (from formaldehyde release biocides, which themselves might be carcinogenic but never tested), and chlorinated paraffins. In addition, liver cancer was not among the target sites highlighted.

Since the release of the criteria document, two additional studies have been published which noted an apparent work-related increase in liver cancer[7,8]. These were updates of cohorts examined in the NIOSH criteria document at an earlier time. One of these studies, Eisen et al, is recognized as the strongest of the epidemiological studies. Earlier reports by the Eisen group noted excesses of cancer at other sites, and exposure response relationships, but did not identify liver cancer. In this regard, the NCI cohort, also updated, may be among the weaker studies. The updates both noted excess mortality from liver cancer which was not found earlier. The UAW concurs that the Eisen cohort is the best

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[6] Calvert GM, Ward E, Schnorr TM, Fine LJ. Cancer risks among workers exposed to metalworking fluids: a systematic review. Am J Ind Med. 1998 Mar;33(3):282-92.

[7] Eisen EA, Bardin J, Gore R, Woskie SR, Hallock MF, Monson RR. Exposure-response models based on extended follow-up of a cohort mortality study in the automobile industry. Scand J Work Environ Health. 2001 Aug;27(4):240-9.

[8] Kazerouni N, Thomas TL, Petralia SA, Hayes RB. Mortality among workers exposed to cutting oil mist: update of previous reports. Am J Ind Med. 2000 Oct;38(4):410-6

studied, given the size of the cohort and the quantitative exposure assessment. However, the ability to see an effect, if it were there, is also impacted by the types of metalworking fluids used, exposure patterns and levels, duration of exposure and cohort characteristics.[9] Two earlier mortality studies among workers exposed to metal working fluids found excess mortality from liver disease (cirrhosis)[10], and from medical examiner-diagnosed alcoholism [which was likely liver disease][11]. We note that during the time period of these studies, there were no identified liver poisons among the chemicals in metal working fluids. Therefore, even the rare personal physician who would consider occupational exposures in relation to liver disease would have attributed liver disease to a personal cause, such as alcohol. In addition, to attribute increased liver disease *in a cohort* to personal factors requires a conclusion that the *cohort as a whole* consumed more alcohol than the general population.

During the earlier period when most of the studies reviewed by NIOSH were published, causes of death which were elevated but not significantly were not published, and liver disease was also not listed as a separate cause of death but instead reported as non malignant digestive disease.

The specific identity and composition of the metalworking fluids used in the period of exposure related to these effects was unknown. However, soluble oils, semi-synthetic and synthetic MWF's have many common ingredients, and ethanolamines and ethanolamine fatty acid condensates were very common. The most prominent liver toxin and laboratory liver carcinogen likely present in MWF is diethanolamine. Some MWF's also contain chlorinated paraffins, which have similar liver toxic properties but much less potency.[12]

Recent toxicological studies have demonstrated liver toxicity of MWF's applied to the skin of mice are working concentrations. NIOSH laboratory scientists confirmed the possibility of liver toxicity from metalworking fluids, and found previously unknown effects on the male reproductive system.[13] The investigators noted their preliminary results showed a significant increase in weights of the liver of both sexes, following dermal application of 50% unused (neat) semisynthetic MWF to the unshaved backs of mice, twice a week for 6 wk.

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[9] Eisen EA, Tolbert PE, Monson RR, Smith TJ. Mortality studies of machining fluid exposure in the automobile industry I: A standardized mortality ratio analysis. Am J Ind Med. 1992; 22(6):809-24.

[10] Silverstein M, Park R, Marmor M, Maizlish N, Mirer F. Mortality among bearing plant workers exposed to metalworking fluids and abrasives. J Occup Med. 1988 Sep;30(9):706-14.

[11] Park RM, Wegman DH, Silverstein MA, Maizlish NA, Mirer FE. Causes of death among workers in a bearing manufacturing plant. Am J Ind Med. 1988;13(5):569-80.

[12] National Toxicology Program, Toxicology and Carcinogenesis Studies of Chlorinated Paraffins (C12, 60% Chlorine) (CAS No. 108171-26-2*) in F344/N Rats and B6C3F$_1$ Mice (Gavage Studies), TR-308

[13] Al-Humadi NH, Shvedova AA, Batteli L, Diotte N, Castranova V, Kommineni C., "Dermal and systemic toxicity after application of semisynthetic metal-working fluids in B6C3F1 mice."J Toxicol Environ Health A. 2000 Dec 15;61(7):579-89

In this study, mice of both sexes were exposed to MWFs following the protocol just described, except that the skin application was with 5% MWFs with or without adding 5% ethanol to their drinking water (7 d/wk) for 13 wk. Skin biochemicals and cell types related to allergic reactions were significantly increased in the female group. The biochemistry of the liver in both sexes, and testes were significantly changed in unhealthy directions. The investigators concluded that "These results suggest that MWFs are absorbed through the skin and produce toxicity in the liver of both sexes and in the male gonads. This may represent an important health risk to MWF-exposed industrial workers, and ethanol may exacerbate this risk."

Another line of evidence for the presence of diethanolamine in MWF's is the series of papers seeking N-nitroso-diethanolamine in MWF's. Some investigators analyzed fluids [14,15,16,17], while others sought the nitrosamines in urine of exposed workers.[18,19,20] The weight of this literature suggests first, that N-nitroso-diethanolamine concentration is relatively low compared to levels causing cancer and liver toxicity in laboratory studies. More important for this review, it demonstrates that diethanolamine, from which the N-nitroso-diethanolamine came, is very common in metalworking fluids.

Although this is not yet consensus view, the UAW believes that there is sufficient evidence to conclude that the "exposure circumstance" of working with water based metalworking fluids is known to be carcinogenic to humans. However, because there are at least five competing carcinogens, and one a liver toxin as well, we are not able to rule out co-exposures and confounding with those co-exposures as a cause. Therefore, the evidence that exposure to diethanolamine is "known" to be carcinogenic to humans is limited.

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[14] Jarvholm B, Zingmark PA, Osterdahl BG N-nitrosodiethanolamine in commercial cutting fluids without nitrites. Ann Occup Hyg. 1991 Dec;35(6):659-63.

[15] Monarca S, Scassellati Sforzolini G, Spiegelhalder B, Pasquini R, Fatigoni C. Monitoring nitrite, N-nitrosodiethanolamine, and mutagenicity in cutting fluids used in the metal industry. Environ Health Perspect. 1993 Jun;101(2):126-8.

[16] Keefer LK, Goff U, Stevens J, Bennett EO. Persistence of N-nitrosodiethanolamine contamination in American metal-working lubricants. Food Chem Toxicol. 1990 Jul;28(7):531-4.

[17] Jarvholm B, Zingmark PA, Osterdahl BG. High concentration of N-nitrosodiethanolamine in a diluted commercial cutting fluid. Am J Ind Med. 1991;19(2):237-9.

[18] Ducos P, Gaudin R, Francin JM Determination of N-nitrosodiethanolamine in urine by gas chromatography thermal energy analysis: application in workers exposed to aqueous metalworking fluids. Int Arch Occup Environ Health. 1999 Jul;72(4):215-22.

[19] Monarca S, Scassellati-Sforzolini G, Donato F, Angeli G, Spiegelhalder B, Fatigoni C, Pasquini R. Biological monitoring of workers exposed to N-nitrosodiethanolamine in the metal industry. Environ Health Perspect. 1996 Jan;104(1):78-82.

[20] Spiegelhalder B, Muller J, Drasche H, Preussmann R N-nitrosodiethanolamine excretion in metal grinders. IARC Sci Publ. 1987;(84):550-2.

This conclusion depends in part on connecting the dots between the presence of diethanolamine generally in water based metalworking fluids, and the likely exposure of the cohorts studied to diethanolamine. The listing criteria state:

> Conclusions regarding carcinogenicity in humans or experimental animals are based on scientific judgment, with consideration given to all relevant information.

In this case, the judgment involves deriving conclusions about the presence of diethanolamine in water based metalworking fluids generally.

f. The IARC 1999 working group conclusions regarding diethanolamine were based on incomplete information

Prior to the meeting of the working group, only the bioassay of diethanolamine had completed publication. The other three bioassays were available only in draft form, and were not considered. One of the additional bioassays had been published at the time of the meeting, and was provided to the carcinogenicity group at the time of the meeting. It received only cursory consideration.

The carcinogenicity group declined to consider any of the condensate bioassays as bioassays of diethanolamine, although NTP itself had concluded that the observed effects were due to residual diethanolamine, thus exonerating the condensates themselves as pure compounds.

Possibly in nullification of IARC criteria, the carcinogenicity group treated the results in male mice and female mice as a single study rather than two separate experiments. They concluded then that a single positive study left the evidence for carcinogenicity in animals at "limited evidence" and therefore the rating for diethanolamine as "not classifiable."

The working group did not have available to it the recent mortality updates. In addition, the working group declined inferring diethanolamine exposure where not explicitly mentioned in a peer reviewed article.

g. Diethanolamine clearly meets the criteria for listing as "reasonably anticipated to be a human carcinogen," based on bioassay data alone. Human data adds extra weight for this determination.

The criteria for listing as "reasonably anticipated for be a human carcinogen" include:

> There is sufficient evidence of carcinogenicity from studies in experimental animals which indicates there is an increased incidence of malignant and/or a combination of malignant and

> benign tumors: (1) in multiple species or at multiple tissue sites, or (2) by multiple routes of exposure, or (3) to an unusual degree with regard to incidence, site or type of tumor, or age at onset;

The UAW notes no dispute about clear evidence in male and female mice, based on liver tumors alone. The UAW argues that the observation of increased kidney tumors, adenomas and carcinomas combined, provides some evidence in one study and clear evidence in a second study. Thus, a second site of action was identified.

The UAW participated in the original efforts to define both the levels of evidence from individual studies, and the criteria for listing. During the discussion of criteria for listing, there was no discussion of the issue of whether observation of tumors repeated studies in males and females of the same species should be given any greater weight than repeated studies showing greater resistance in one sex of a species. In our memory, the paradigm of a single species carcinogen being considered was the male rat exposed to limonene.

In addition, the UAW believes that observation of tumors at a site remote from the application site in a skin absorption bioassay should be interpreted as "an unusual degree."

In addition, in both sexes of mice, the incidence of liver tumors was virtually 100% at the lowest dose tested. This also meets the definition of "unusual degree."

Finally, we believe that the repeated observation of increased cancers among workers likely exposed to diethanolamine in metal working fluids, and the recent observation of increased liver cancers among such workers, should be given some weight in the rating of diethanolamine.

Appendix

Terminal Sacrifice at 104 weeks

Dose	Males 0 MG/KG	Males 40 MG/KG	Males 80 MG/KG	Males 160 MG/KG	Females 0 MG/KG	Females 40 MG/KG	Females 80 MG/KG	Females 160 MG/KG
Kidney: Renal Tubule Adenoma								
TUMOR RATES								
OVERALL (a)	1/50 (2%)	4/50 (8%)	6/50 (12%)	6/50 (12%)	0/50 (0%)	0/50 (0%)	0/50 (0%)	0/50 (0%)
POLY-3 ADJUSTED (b)	2.2%	8.3%	13.1%	13.3%	0.0%	0.0%	0.0%	0.0%
TERMINAL (d)	1/40 (3%)	3/43 (7%)	3/34 (9%)	2/30 (7%)	0/44 (0%)	0/33 (0%)	0/33 (0%)	0/23 (0%)
FIRST INCIDENCE	729 (T)	692	654	540	---	---	---	---
STATISTICAL TESTS								
LIFE TABLE	P=0.028 *	P=0.207	P=0.047 *	P=0.042 *	(e)	(e)	(e)	(e)
POLY 3	P=0.049 *	P=0.196	P=0.056	P=0.053	(e)	(e)	(e)	(e)
POLY 1.5	P=0.051	P=0.189	P=0.057	P=0.054	(e)	(e)	(e)	(e)
POLY 6	P=0.047 *	P=0.202	P=0.054	P=0.053	(e)	(e)	(e)	(e)
LOGISTIC REGRESSION	P=0.058	P=0.197	P=0.058	P=0.061	(e)	(e)	(e)	(e)
COCH-ARM / FISHERS	P=0.056	P=0.181	P=0.056	P=0.056	(e)	(e)	(e)	(e)

Dose	Males 0 MG/KG	Males 40 MG/KG	Males 80 MG/KG	Males 160 MG/KG	Females 0 MG/KG	Females 40 MG/KG	Females 80 MG/KG	Females 160 MG/KG
Kidney: Renal Tubule Carcinoma								
TUMOR RATES								
OVERALL (a)	2/50 (4%)	1/50 (2%)	0/50 (0%)	2/50 (4%)	0/50 (0%)	0/50 (0%)	0/50 (0%)	0/50 (0%)
POLY-3 ADJUSTED (b)	4.4%	2.1%	0.0%	4.6%	0.0%	0.0%	0.0%	0.0%
TERMINAL (d)	2/40 (5%)	1/43 (2%)	0/34 (0%)	2/30 (7%)	0/44 (0%)	0/33 (0%)	0/33 (0%)	0/23 (0%)
FIRST INCIDENCE	729 (T)	729 (T)	---	729 (T)	---	---	---	---
STATISTICAL TESTS								
LIFE TABLE	P=0.482	P=0.475N	P=0.275N	P=0.588	(e)	(e)	(e)	(e)
POLY 3	P=0.574	P=0.483N	P=0.240N	P=0.680	(e)	(e)	(e)	(e)
POLY 1.5	P=0.580	P=0.489N	P=0.238N	P=0.684	(e)	(e)	(e)	(e)
POLY 6	P=0.566	P=0.476N	P=0.245N	P=0.674	(e)	(e)	(e)	(e)
LOGISTIC REGRESSION	P=0.482	P=0.475N	(e)	P=0.588	(e)	(e)	(e)	(e)
COCH-ARM / FISHERS	P=0.591	P=0.500N	P=0.247N	P=0.691N	(e)	(e)	(e)	(e)

Date: 06/19/97 EXPERIMENT: 05162 TEST: 10 Page 5

Statistical Analysis of Primary Tumors in Mice(B6C3F1) - DIETHANOLAMINE

Terminal Sacrifice at 104 weeks

	Males				Females			
Dose	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG
Kidney: Renal Tubule Carcinoma or Adenoma								
TUMOR RATES								
OVERALL (a)	3/50 (6%)	5/50 (10%)	6/50 (12%)	8/50 (16%)	0/50 (0%)	0/50 (0%)	0/50 (0%)	0/50 (0%)
POLY-3 ADJUSTED (b)	6.6%	10.4%	13.1%	17.8%	0.0%	0.0%	0.0%	0.0%
TERMINAL (d)	3/40 (8%)	4/43 (9%)	3/34 (9%)	4/30 (13%)	0/44 (0%)	0/33 (0%)	0/33 (0%)	0/23 (0%)
FIRST INCIDENCE	729 (T)	692	654	540	---	---	---	---
STATISTICAL TESTS								
LIFE TABLE	P=0.030 *	P=0.400	P=0.193	P=0.056	(e)	(e)	(e)	(e)
POLY 3	P=0.064	P=0.386	P=0.242	P=0.093	(e)	(e)	(e)	(e)
POLY 1.5	P=0.067	P=0.375	P=0.244	P=0.095	(e)	(e)	(e)	(e)
POLY 6	P=0.061	P=0.398	P=0.237	P=0.092	(e)	(e)	(e)	(e)
LOGISTIC REGRESSION	P=0.068	P=0.396	P=0.238	P=0.096	(e)	(e)	(e)	(e)
COCH-ARM / FISHERS	P=0.073	P=0.357	P=0.243	P=0.100	(e)	(e)	(e)	(e)
	Males				Females			
Dose	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG

Liver
Hemangiosarcoma

TUMOR RATES								
OVERALL (a)	0/50 (0%)	1/50 (2%)	2/50 (4%)	0/50 (0%)	0/50 (0%)	0/50 (0%)	0/50 (0%)	0/50 (0%)
POLY-3 ADJUSTED (b)	0.0%	2.1%	4.4%	0.0%	0.0%	0.0%	0.0%	0.0%
TERMINAL (d)	0/40 (0%)	1/43 (2%)	0/34 (0%)	0/30 (0%)	0/44 (0%)	0/33 (0%)	0/33 (0%)	0/23 (0%)
FIRST INCIDENCE	---	729 (T)	583	---	---	---	---	---
STATISTICAL TESTS								
LIFE TABLE	P=0.651N	P=0.514	P=0.237	(e)	(e)	(e)	(e)	(e)
POLY 3	P=0.634N	P=0.510	P=0.238	(e)	(e)	(e)	(e)	(e)
POLY 1.5	P=0.628N	P=0.506	P=0.238	(e)	(e)	(e)	(e)	(e)
POLY 6	P=0.642N	P=0.514	P=0.235	(e)	(e)	(e)	(e)	(e)
LOGISTIC REGRESSION	P=0.562N	P=0.514	P=0.206	(e)	(e)	(e)	(e)	(e)
COCH-ARM / FISHERS	P=0.616N	P=0.500	P=0.247	(e)	(e)	(e)	(e)	(e)

Date: 06/19/97 EXPERIMENT: 05162 TEST: 10 Page 6
Statistical Analysis of Primary Tumors in Mice(B6C3F1) - DIETHANOLAMINE
Terminal Sacrifice at 104 weeks

	Males				Females			
Dose	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG
Liver Hepatoblastoma								
TUMOR RATES								
OVERALL (a)	0/50 (0%)	2/50 (4%)	8/50 (16%)	5/50 (10%)	0/50 (0%)	2/50 (4%)	1/50 (2%)	1/50 (2%)
POLY-3 ADJUSTED (b)	0.0%	4.2%	17.5%	11.3%	0.0%	4.8%	2.3%	2.3%
TERMINAL (d)	0/40 (0%)	2/43 (5%)	4/34 (12%)	2/30 (7%)	0/44 (0%)	1/33 (3%)	1/33 (3%)	1/23 (4%)
FIRST INCIDENCE	---	729 (T)	633	684	---	421	731 (T)	731 (T)
STATISTICAL TESTS								
LIFE TABLE	P=0.010 *	P=0.254	P=0.004 **	P=0.023 *	P=0.370	P=0.206	P=0.443	P=0.371
POLY 3	P=0.018 *	P=0.249	P=0.004 **	P=0.028 *	P=0.446	P=0.207	P=0.482	P=0.478
POLY 1.5	P=0.019 *	P=0.244	P=0.004 **	P=0.029 *	P=0.471	P=0.218	P=0.490	P=0.488
POLY 6	P=0.016 *	P=0.253	P=0.003 **	P=0.027 *	P=0.407	P=0.196	P=0.472	P=0.461
LOGISTIC REGRESSION	P=0.021 *	P=0.254	P=0.005 **	P=0.030 *	P=0.509	P=0.451	P=0.443	P=0.371
COCH-ARM / FISHERS	P=0.026 *	P=0.247	P=0.003 **	P=0.028 *	P=0.500	P=0.247	P=0.500	P=0.500

Dose	Males 0 MG/KG	Males 40 MG/KG	Males 80 MG/KG	Males 160 MG/KG	Females 0 MG/KG	Females 40 MG/KG	Females 80 MG/KG	Females 160 MG/KG
Liver Hepatocellular Adenoma								
TUMOR RATES								
OVERALL (a)	31/50 (62%)	42/50 (84%)	49/50 (98%)	45/50 (90%)	32/50 (64%)	50/50 (100%)	48/50 (96%)	48/50 (96%)
POLY-3 ADJUSTED (b)	65.0%	86.5%	98.0%	93.5%	66.1%	100.0%	96.4%	96.4%
TERMINAL (d)	28/40 (70%)	40/43 (93%)	33/34 (97%)	28/30 (93%)	30/44 (68%)	33/33 (100%)	33/33 (100%)	23/23 (100%)
FIRST INCIDENCE	411	641	445	386	674	418	474	522
STATISTICAL TESTS								
LIFE TABLE	P<0.001 **	P=0.037 *	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 3	P<0.001 **	P=0.009 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 1.5	P<0.001 **	P=0.009 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 6	P<0.001 **	P=0.010 *	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
LOGISTIC REGRESSION	P<0.001 **	P=0.022 *	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
COCH-ARM / FISHERS	P<0.001 **	P=0.012 *	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **

Date: 06/19/97 EXPERIMENT: 05162 TEST: 10 Page 7
Statistical Analysis of Primary Tumors in Mice(B6C3F1) - DIETHANOLAMINE
Terminal Sacrifice at 104 weeks

Dose	Males 0 MG/KG	Males 40 MG/KG	Males 80 MG/KG	Males 160 MG/KG	Females 0 MG/KG	Females 40 MG/KG	Females 80 MG/KG	Females 160 MG/KG
Liver Hepatocellular Carcinoma								
TUMOR RATES								
OVERALL (a)	12/50 (24%)	17/50 (34%)	33/50 (66%)	34/50 (68%)	5/50 (10%)	19/50 (38%)	38/50 (76%)	42/50 (84%)
POLY-3 ADJUSTED (b)	25.1%	34.9%	66.9%	72.3%	10.4%	43.4%	77.9%	84.9%
TERMINAL (d)	6/40 (15%)	13/43 (30%)	20/34 (59%)	20/30 (67%)	4/44 (9%)	12/33 (36%)	26/33 (79%)	18/23 (78%)
FIRST INCIDENCE	485	576	445	446	729	423	474	522
STATISTICAL TESTS								
LIFE TABLE	P<0.001 **	P=0.275	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 3	P<0.001 **	P=0.206	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 1.5	P<0.001 **	P=0.201	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 6	P<0.001 **	P=0.208	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
LOGISTIC REGRESSION	P<0.001 **	P=0.147	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **

COCH-ARM / FISHERS	P<0.001 **	P=0.189	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
	Males				Females			
Dose	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG
Liver								
Hepatocellular Carcinoma or Hepatoblastoma								
TUMOR RATES								
OVERALL (a)	12/50 (24%)	18/50 (36%)	34/50 (68%)	34/50 (68%)	5/50 (10%)	20/50 (40%)	39/50 (78%)	43/50 (86%)
POLY-3 ADJUSTED (b)	25.1%	36.9%	68.9%	72.3%	10.4%	44.9%	79.9%	86.9%
TERMINAL (d)	6/40 (15%)	14/43 (33%)	20/34 (59%)	20/30 (67%)	4/44 (9%)	12/33 (36%)	27/33 (82%)	19/23 (83%)
FIRST INCIDENCE	485	576	445	446	729	421	474	522
STATISTICAL TESTS								
LIFE TABLE	P<0.001 **	P=0.217	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 3	P<0.001 **	P=0.151	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 1.5	P<0.001 **	P=0.147	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 6	P<0.001 **	P=0.152	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
LOGISTIC REGRESSION	P<0.001 **	P=0.106	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
COCH-ARM / FISHERS	P<0.001 **	P=0.138	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **

Date: 06/19/97 EXPERIMENT: 05162 TEST: 10 Page 8

Statistical Analysis of Primary Tumors in Mice(B6C3F1) - DIETHANOLAMINE

Terminal Sacrifice at 104 weeks

	Males				Females			
Dose	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG
Liver Hepatocellular Carcinoma or Hepatocellular Adenoma								
TUMOR RATES								
OVERALL (a)	39/50 (78%)	47/50 (94%)	50/50 (100%)	49/50 (98%)	33/50 (66%)	50/50 (100%)	50/50 (100%)	50/50 (100%)
POLY-3 ADJUSTED (b)	79.0%	95.3%	100.0%	99.9%	68.2%	100.0%	100.0%	100.0%
TERMINAL (d)	31/40 (78%)	41/43 (95%)	34/34 (100%)	30/30 (100%)	31/44 (71%)	33/33 (100%)	33/33 (100%)	23/23 (100%)
FIRST INCIDENCE	411	576	445	386	674	418	474	522
STATISTICAL TESTS								
LIFE TABLE	P<0.001 **	P=0.208	P=0.003 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 3	P<0.001 **	P=0.014 *	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 1.5	P<0.001 **	P=0.015 *	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 6	P<0.001 **	P=0.014 *	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
LOGISTIC REGRESSION	P<0.001 **	P=0.025 *	P<0.001 **	P=0.002 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
COCH-ARM / FISHERS	P<0.001 **	P=0.020 *	P<0.001 **	P=0.002 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **

	Males				Females			
Dose	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG	0 MG/KG	40 MG/KG	80 MG/KG	160 MG/KG
Liver Hepatocellular Carcinoma, Hepatocellular Adenoma, or Hepatoblastoma								
TUMOR RATES								
OVERALL (a)	39/50 (78%)	47/50 (94%)	50/50 (100%)	49/50 (98%)	33/50 (66%)	50/50 (100%)	50/50 (100%)	50/50 (100%)
POLY-3 ADJUSTED (b)	79.0%	95.3%	100.0%	99.9%	68.2%	100.0%	100.0%	100.0%
TERMINAL (d)	31/40 (78%)	41/43 (95%)	34/34 (100%)	30/30 (100%)	31/44 (71%)	33/33 (100%)	33/33 (100%)	23/23 (100%)
FIRST INCIDENCE	411	576	445	386	674	418	474	522
STATISTICAL TESTS								
LIFE TABLE	P<0.001 **	P=0.208	P=0.003 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 3	P<0.001 **	P=0.014 *	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 1.5	P<0.001 **	P=0.015 *	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
POLY 6	P<0.001 **	P=0.014 *	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
LOGISTIC REGRESSION	P<0.001 **	P=0.025 *	P<0.001 **	P=0.002 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **

COCH-ARM / FISHERS	P<0.001 **	P=0.020 *	P<0.001 **	P=0.002 **	P<0.001 **	P<0.001 **	P<0.001 **	P<0.001 **
Kidney: Renal Tubule Adenoma								
TUMOR RATES								
OVERALL (a)	1/50 (2%)	1/50 (2%)	7/50 (14%)		0/50 (0%)	0/50 (0%)	1/50 (2%)	
POLY-3 ADJUSTED (b)	2.2%	2.3%	15.3%		0.0%	0.0%	2.4%	
TERMINAL (d)	1/41 (2%)	1/37 (3%)	4/36 (11%)		0/35 (0%)	0/36 (0%)	0/26 (0%)	
FIRST INCIDENCE	727 (T)	727 (T)	597		---	---	621	
STATISTICAL TESTS								
LIFE TABLE	P=0.009 **	P=0.739	P=0.027 *		P=0.257	(e)	P=0.481	
POLY 3	P=0.009 **	P=0.751	P=0.029 *		P=0.274	(e)	P=0.481	
POLY 1.5	P=0.009 **	P=0.754	P=0.029 *		P=0.273	(e)	P=0.487	
POLY 6	P=0.009 **	P=0.748	P=0.029 *		P=0.277	(e)	P=0.471	
LOGISTIC REGRESSION	P=0.010 *	P=0.739	P=0.033 *		P=0.305	(e)	P=0.545	
COCH-ARM / FISHERS	P=0.010 *	P=0.753N	P=0.030 *		P=0.269	(e)	P=0.500	

Date: 09/26/97 EXPERIMENT: 55312 TEST: 04 Page 4
Statistical Analysis of Primary Tumors in Mice(B6C3F1) - COCONUT OIL ACID DIETHANOLAMINE CONDENSATE
Terminal Sacrifice at 103 weeks

	Males			Females		
Dose	0 MG/KG	100 MG/KG	200 MG/KG	0 MG/KG	100 MG/KG	200 MG/KG
Kidney: Renal Tubule Carcinoma						
TUMOR RATES						
OVERALL (a)	0/50 (0%)	0/50 (0%)	2/50 (4%)	0/50 (0%)	0/50 (0%)	0/50 (0%)
POLY-3 ADJUSTED (b)	0.0%	0.0%	4.4%	0.0%	0.0%	0.0%
TERMINAL (d)	0/41 (0%)	0/37 (0%)	2/36 (6%)	0/35 (0%)	0/36 (0%)	0/26 (0%)
FIRST INCIDENCE	---	---	727 (T)	---	---	---
STATISTICAL TESTS						
LIFE TABLE	P=0.085	(e)	P=0.210	(e)	(e)	(e)
POLY 3	P=0.094	(e)	P=0.231	(e)	(e)	(e)
POLY 1.5	P=0.094	(e)	P=0.233	(e)	(e)	(e)
POLY 6	P=0.094	(e)	P=0.228	(e)	(e)	(e)
LOGISTIC REGRESSION	(e)	(e)	P=0.210	(e)	(e)	(e)
COCH-ARM / FISHERS	P=0.095	(e)	P=0.247	(e)	(e)	(e)

Dose	Males 0 MG/KG	Males 100 MG/KG	Males 200 MG/KG	Females 0 MG/KG	Females 100 MG/KG	Females 200 MG/KG
Kidney: Renal Tubule Carcinoma or Adenoma						
TUMOR RATES						
OVERALL (a)	1/50 (2%)	1/50 (2%)	9/50 (18%)	0/50 (0%)	0/50 (0%)	1/50 (2%)
POLY-3 ADJUSTED (b)	2.2%	2.3%	19.6%	0.0%	0.0%	2.4%
TERMINAL (d)	1/41 (2%)	1/37 (3%)	6/36 (17%)	0/35 (0%)	0/36 (0%)	0/26 (0%)
FIRST INCIDENCE	727 (T)	727 (T)	597	---	---	621
STATISTICAL TESTS						
LIFE TABLE	P=0.002 **	P=0.739	P=0.007 **	P=0.257	(e)	P=0.481
POLY 3	P<0.001 **	P=0.751	P=0.007 **	P=0.274	(e)	P=0.481
POLY 1.5	P<0.001 **	P=0.754	P=0.008 **	P=0.273	(e)	P=0.487
POLY 6	P<0.001 **	P=0.748	P=0.007 **	P=0.277	(e)	P=0.471
LOGISTIC REGRESSION	P=0.002 **	P=0.739	P=0.009 **	P=0.305	(e)	P=0.545
COCH-ARM / FISHERS	P=0.002 **	P=0.753N	P=0.008 **	P=0.269	(e)	P=0.500

SYNTILO 9154 HONING FLUID

```
     TDS: 0071173                                      Cons Prod/Haz Sub: YES
    Part: 470502455        Standard: N/AV                   Hazwoper Haz: YES
Supplier: 94044           Prep Date: 03/21/1995          OSHA Hazardous: YES
Manufctr: 94044
*******************************************************************************
********************** SECTION 01 - PRODUCT INFORMATION ***********************
*******************************************************************************
 MFG BY: CASTROL INDUSTRIAL CENTRAL INC     CASTROL INDUSTRIAL INC
         149 GRANT STREET     NORTH AURORA   IL     60542
         EMERGENCY PHONE: 312-454-1000                AFTER HOURS: 312-454-1000
DIST BY: CASTROL INDUSTRIAL CENTRAL INC     CASTROL INDUSTRIAL INC
         149 GRANT STREET     NORTH AURORA   IL     60542
         EMERGENCY PHONE: 312-454-1000                AFTER HOURS: 312-454-1000
DAIMLERCHRYSLER INDUSTRIAL HYGIENE: 248-512-8260     AFTER HOURS: 248-576-8888
BRAND NAME: SYNTILO 9154 HONING FLUID
MFG ID     : 39-154D
DESCRIPTION: CUTTING OIL CASTROL INDUS  SYNTILO 9154 HONING  OIL
*******************************************************************************
************************** SECTION 02 - INGREDIENTS ***************************
*******************************************************************************
HAZARDOUS INGREDIENTS:              PERCENT
COMM NAME / CAS NO & CHEM NAME:     BY WGT    OSHA ACGIH CHRYS UNITS NOTATIONS
                          ***EXISTING INGREDIENTS***
DIETHANOLAMINE                      10-30  W     3 0.460 0.460 PPM   S
000111-42-2  ETHANOL, 2,2'-IMINOB                                     T

GENERIC DESC: CUTTING SOLUTION MIXTURE OF AMINES, SURFACTANT AND WATER.
  FOR EXPLANATION OF "NOTATIONS", SEE THE HAZARD COMMUNICATION SHEET
EXPLANATIONS PAGE.
*******************************************************************************
************************* SECTION 03 - PHYSICAL DATA **************************
*******************************************************************************
BOILING POINT :     212 F            SOLUBILITY IN WATER: N/AV
VAPOR PRESSURE: N/AV                   EVAP. RATE: < 1.000  REF: BUTYLACETATE
VAPOR DENSITY :  N/AV                  SPECIFIC GRAVITY:   1.070 AT N/AV
PH AT FULL STRENGTH:    9.5           PH AT REC. DILUT:   N/AV
%VOLATILE BY VOL    :   50.0           VOLATILE ORGANIC COMP: N/AV     N/AV
ODOR THRESHOLD:   N/AV       PPM FOR     % POPULATION
FREEZING POINT:     N/AV                 COEFF. OF WATER/OIL DIST: N/AV
APPEARANCE & ODOR: APPEARANCE: CLEAR..... STATE: LIQUID.....-ODOR: SLIGHT......
*******************************************************************************
******************** SECTION 04 - FIRE AND EXPLOSION DATA *********************
*******************************************************************************
FLASH POINT:  N/AP      N/AV   IGN TEMP:  N/AP      LEL: N/AV     UEL: N/AV
SPECIAL FIRE & EXPLOSION HAZARDS: MAY PRODUCE TOXIC THERMAL DECOMPOSITION
   PRODUCTS.
EXTINGUISHING MEDIA:   NON-FLAMMABLE-USE MEDIA APPROPRIATE FOR MATERIALS
   ACTUALLY INVOLVED IN FIRE.
SPECIAL FIREFIGHTING PROCEDURES: USE WATER TO FLUSH SPILL AWAY FROM FIRE.
SENSITIVE TO MECHANICAL IMPACT?: NO       SENSITIVE TO STATIC DISCHARGE?: NO
HAZARDOUS COMBUSTION PRODUCTS: CARBON MONOXIDE, CARBON DIOXIDE, NITROGEN OXIDES

FLAME PROJECTION:  N/AP
```

********** SECTION 05 - HEALTH HAZARD DATA **********

EFFECTS OF OVEREXPOSURE - ACUTE & CHRONIC: <<---SKIN CONTACT--->> MIST-CONCENTRATED SOLUTIONS MAY PRODUCE THE FOLLOWING: IRRITATION OF UPPER RESPIRATORY TRACT AND MUCOUS MEMBRANE IRRITATION. DIRECT SKIN CONTACT WITH THIS PRODUCT MAY CAUSE TEMPORARY HAIR LOSS AT THE SITE OF CONTACT. <<---SKIN CONTACT--->> LIQUID-CONCENTRATED SOLUTIONS MAY PRODUCE THE FOLLOWING: SEVERE IRRITATION. PROLONGED CONTACT-DILUTE CONCENTRATIONS MAY PRODUCE THE FOLLOWING: MINOR IRRITATION. <<---EYE CONTACT--->> DIRECT CONTACT-CONCENTRATED SOLUTIONS MAY PRODUCE THE FOLLOWING: SEVERE IRRITATION. DILUTE CONCENTRATIONS MAY PRODUCE THE FOLLOWING: IRRITATION AND TEARING. <<---INGESTION--->> CONCENTRATED SOLUTIONS MAY PRODUCE THE FOLLOWING: MUCOUS MEMBRANE IRRITATION AND MAY CAUSE UPSET STOMACH, NAUSEA, VOMITING AND DIARRHEA.

EMERGENCY FIRST AID PROCEDURES: <<---INHALATION--->> REMOVE TO FRESH AIR AT ONCE. CONTACT A PHYSICIAN. <<---SKIN CONTACT--->> REMOVE CONTAMINATED CLOTHING. WASH WITH SOAP AND WATER. IF CONDITION PERSISTS, CONSULT A PHYSICIAN. <<---EYE CONTACT--->> RINSE EYES IMMEDIATELY WITH LARGE AMOUNTS OF WATER, OCCASIONALLY LIFTING BOTH UPPER AND LOWER LIDS. CONTINUE FOR 15 MINUTES. IF CONDITION PERSISTS, CONSULT A PHYSICIAN. <<---INGESTION--->> DO NOT INDUCE VOMITING-CALL PHYSICIAN IMMEDIATELY.

********** SECTION 06 - REACTIVITY DATA **********

STABILITY: STABLE

CONDITIONS TO AVOID: NONE KNOWN.

MATERIALS TO AVOID: STRONG OXIDIZING AGENTS.

HAZARDOUS POLYMERIZATION: WILL NOT OCCUR

POLYMERIZATION CONDITIONS TO AVOID: POLYMERIZATION WILL NOT OCCUR.

HAZARDOUS DECOMPOSITION PRODUCTS: CARBON MONOXIDE, CARBON DIOXIDE, NITROGEN OXIDES, AND HYDROGEN CHLORIDE GAS.

********** SECTION 07 - SPILL OR LEAK PROCEDURES **********

STEPS TO BE TAKEN IF MATERIAL IS RELEASED OR SPILLED: CONTAIN WITH DIKE. PUMP INTO STORAGE CONTAINER. RESIDUE-FLUSH AREA WITH WATER.

WASTE DISPOSAL METHODS: DISPOSE OF IN A MANNER CONSISTENT WITH STATE, PROVINCIAL, LOCAL, AND FEDERAL DISPOSAL REGULATIONS. CHEMICALLY SPLIT EMULSIONS WITH STRONG ACID. ALLOW TO SEPARATE. SKIM OFF OIL WHICH FLOATS TO THE TOP AND HAUL AWAY OR BURN. NEUTRALIZE REMAINING ACIDIC LIQUID WITH ALKALINE MATERIAL.

********** SECTION 08 - SPECIAL PROTECTION **********

RESPIRATORY PROTECTION: NO SPECIAL PROTECTION NEEDED UNDER NORMAL CONDITIONS. DUST AND MIST RESPIRATOR. THIS EQUIPMENT MAY BE NEEDED IF THE ALLOWABLE LIMIT OF THE PRODUCT OR ANY COMPONENT IS EXCEEDED. OR. THIS EQUIPMENT MAY BE NEEDED IF THE PRODUCT IS MISTED..

VENTILATION TYPE: LOCAL EXHAUST VENTILATION.

PROTECTIVE GLOVES: RECOMMENDED. BUTYL RUBBER GLOVES OR NEOPRENE GLOVES.. THIS EQUIPMENT MAY BE NEEDED TO AVOID PROLONGED SKIN CONTACT..

EYE PROTECTION: RECOMMENDED. CHEMICAL GOGGLES OR FULL FACE SHIELD..

OTHER PROTECTIVE EQUIPMENT: APRON (SAME MATERIAL AS GLOVES). THIS EQUIPMENT MAY BE NEEDED TO AVOID PROLONGED SKIN CONTACT.. AN EYE WASH MAY BE NEEDED FOR EMERGENCY SITUATIONS.. THIS EQUIPMENT MAY BE NEEDED FOR EMERGENCY SITUATIONS..

********** SECTION 09 - SPECIAL PRECAUTIONS **********

PRECAUTIONS TO BE TAKEN IN HANDLING & STORAGE: STORE SEPARATE FROM INCOMPATABLE MATERIALS. REACTS WITH OXIDIZERS. ALLOW MATERIAL TO WARM UP TO ROOM TEMPERATURE BEFORE USING. KEEP CONTAINER TIGHTLY CLOSED WHEN NOT IN USE. THIS PRODUCT CONTAINS AN AMINE COMPOUND. DO NOT LET THIS PRODUCT CONTACT OR MIX WITH PRODUCTS WHICH CONTAIN NITRITES, BECAUSE HAZARDOUS NITROSAMINES MAY BE FORMED.

OTHER PRECAUTIONARY MEASURES: AVOID BREATHING MIST. AVOID SKIN CONTACT. AVOID EYE CONTACT. AVOID INGESTION. WASH THOROUGHLY AFTER HANDLING. REMOVE CONTAMINATED CLOTHING AND LAUNDER BEFORE REUSE. MAINTAIN GOOD HOUSEKEEPING AND HYGIENIC PRACTICES.

********** SECTION 10 - WASTE LABELING INFORMATION **********

DOT LABELING INFORMATION (49 CFR 100-199)

ID#: N/AP ERG#: HAZARD CLASS - PRMY: N/AP PACKING GROUP: N/AP

NOT HAZARDOUS PER DOT REGULATIONS

********** SECTION 11 - PREPARATION INFORMATION **********

PREPARATION DATE: 03/21/1995 DATA EFFECTIVE DATE: 04/10/1990