

NMA/NBMA ASSOCIATION

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SRA International
1920 L Street, NW, Suite 420, Washington, DC 20036
(202) 728-1400 – (202) 331-3393 fax

September 30, 2002

VIA COURIER-HAND DELIVERED

U.S. Environmental Protection Agency
HPV Challenge Program
EPA East Building Room 6428
1200 Constitution Avenue, NW
Washington, DC 20460

Attn: Response to EPA Comments on the Robust Summaries/Test Plan

Dear Dr. Hernandez:

The NMA/NBMA Association (Consortium Registration No. _____, consisting of Cytec Industries Inc. and National Starch (ICI), has taken responsibility for addressing the EPA requests for two chemicals, acrylamide, N-(hydroxymethyl) [NMA], CAS No. 924-42-5, and acrylamide, N-(butoxymethyl) [NBMA], CAS No. 1852-16-0, under the EPA HPV Challenge Program.

Previously (7 September 2001), the Association submitted to EPA Robust Summaries and a Test Plan for these substances (i.e. NMA and NBMA) and in addition, Robust Summaries on acrylamide, a closely related chemical on which there is a plethora of available scientific information. Subsequently (4 June 2002), the EPA issued comments on that submission. The current submission is the Association's response to the EPA comments.

Format of this Submission

Given the large amount of material submitted to and reviewed by the EPA, the subsequent comments from the Agency were relatively few in number. To facilitate review of our responses to the issues raised by the Agency, this document includes the Agency's original comment followed by the Association response.

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ISSUE #1: CATEGORY JUSTIFICATION

EPA COMMENT:

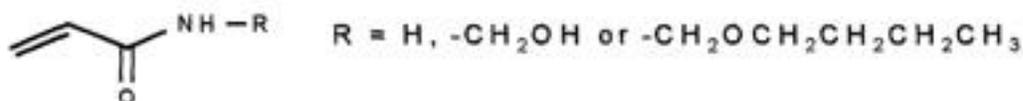
The proposed category includes NMA and NBMA, and the submitter proposed that these can be grouped together with the structural analog acrylamide (AMD). However, the submitter needs to discuss similarities in metabolism, distribution, and biological activities (including modes of action) of AMD, NMA, and NBMA that are likely to provide important support for grouping these chemicals together and for using AMD as an analog for NMA and NBMA.

ASSOCIATION RESPONSE¹:

There are a number of general principles that underlie comparisons of these compounds; two of them are easy to state and are repeatedly supported by the available data. First, these compounds have similar physical, chemical, physiological and biological properties. For example, their metabolism is interrelated, their distribution very similar, and their biological effects are *qualitatively* nearly identical. In many instances, the *quantitative* aspects of their biological effects are similar; however when they do differ, it is generally AMD that is the most biologically active of the group. Second, because of the similarities in structure, reactivity and manufacturing, AMD is generally a component of closely related products. To wit, it is known that AMD accounts for $\leq 5\%$ of products containing NMA. Thus, given the greater potency of AMD compared to its structural analogs, and its presence in related products, AMD is likely to play a significant role in the overall toxicity of those related products. These principles should be kept in mind when reviewing the information provided below.

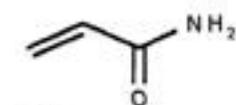
Similarity in Structure

The three substances identified above (AMD, NMA, and NBMA) can be grouped together because of their close structural similarities and relatively minor structural differences. The similarity is based upon the fact that the structure of all three substances contains the "propanamide" or the "acrylamide" moiety. The generic molecular structure of all category members is shown below:

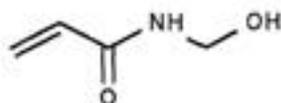


¹ Much of the information presented herein was obtained from the National Toxicology program which employed these data in reaching its decision to test structural analogs of AMD based upon similarities in structure, metabolism, distribution, genetic/reproductive/developmental and neuro-toxicity (NTP Tech. Report No. 352, 1989).

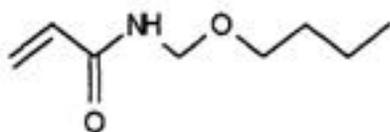
The structural difference between these three compounds results from a substitution on acrylamide to form the other two compounds. Thus, when one of the hydrogens on the nitrogen atom is replaced with either methylol (-CH₂OH) or butoxymethyl (-CH₂OCH₂CH₂CH₂CH₃) group, NMA or NBMA are formed, respectively.



AMD



NMA



NBMA

Similarity in Comparative Toxicity and Mechanism of Action

When comparing the toxicity of these chemical substances, a pattern of qualitatively similar effects with AMD being quantitatively more toxic is repeatedly observed across a wide range of toxicological endpoints.

The rat LD₅₀ values of NMA and AMD are in the same range, with the latter being slightly more toxic, 563 versus 203 mg/kg (Hashimoto and Aldridge, 1970). Injections, i.p., of up to 100 mg/kg of AMD or NMA twice per week resulted in onset of ataxia and weight loss at 4 weeks in the AMD-dosed groups, but no effects were observed after 10 weeks in the NMA groups. However, the close interaction of these compounds was documented by feeding rats a diet containing 1,400 ppm NMA for 1 week preceding AMD injections and 700 ppm during the week injections. This regimen resulted in an earlier onset of AMD toxicity than that observed in AMD-injected rats fed control diets. Furthermore, AMD and NMA have similar rate constants for all conditions, when tested *in vitro* with glutathione, protein sulfhydryl groups, and binding to hemoglobin.

The extent of depletion of non-protein sulfhydryl groups in the brain, spinal cord, and liver was similar after gavage administration of equal amounts of AMD and NMA to rats, and the pattern of tissue and sub-cellular organelle distribution of the carbon-14 label was similar after administration of equal doses of [1-¹⁴C]-AMD or [1-¹⁴C]-NMA. Radioactivity was found in all tissues examined, with high counts in the blood; protein binding was observed. Radioactivity was found in all sub-cellular fractions of brain and liver in amounts related to the protein content of the fractions. Little binding to nucleic acids was found.

Edwards (1974) reported that NMA produced neurotoxic effects similar to those of AMD, but was about one-fifth as potent, and that the neurotoxicity was probably not a result of conversion to AMD *in vivo*. Rats given diets containing 1,800 ppm NMA for 1 week and then diets containing 900 ppm for 5 weeks demonstrated slight ataxic effects that worsened when four additional intraperitoneal injections of NMA at 50 mg/kg were given over the next 2 weeks (Edwards, 1975a). Tani and Hashimoto (1983) gave rats drinking water containing up to 13.8 mM NMA for 90 days. They observed decreased weight gain and deficits in performance on a neurobehavioral test (rotarod). Examination of tibial and sural nerves showed microscopic evidence of shrinkage and loss of myelinated fibers, myelin retraction, and corrugated myelin sheaths. [³H]Colchicine binding (a measure of neurotubulin content) was reduced by 50% in the sciatic nerve after 60 days of dosing with drinking water containing 13.8 mM NMA. Similar effects on rotarod performance were observed for mice given doses of NMA at one-fifth to one-half the LD₅₀ value by gavage twice per week (Hashimoto et al., 1981). Neurobehavioral effects were seen after 4 weeks. Simultaneous intraperitoneal administration of 50 mg/kg phenobarbital, 5 days per week, lessened the signs of neuropathy, presumably through stimulation of drug-metabolizing enzymes.

Similarity in Reproductive and Developmental Toxicity

These chemicals are known to produce alterations in the male reproductive organs. Based upon the degeneration of the testicular tubules in rats given AMD (McCollister et al., 1964), Hashimoto et al. (1981) examined effects of NMA dosing on the mouse testis and demonstrated degeneration of the epithelia of the seminiferous tubules including the spermatids and spermatocytes, reduced spermatozoa, and reduced testicular weight after 8 weeks of gavage administration.

Sakamoto and Hashimoto (1986) reported the results from reproductive toxicity (dominant lethal) and sperm morphology tests with NMA, AMD, and two other structurally related compounds (N-methylacrylamide and N-isopropylacrylamide) in mice. Significant increases in resorptions and decreases in fetuses in females mated 1-8 days after exposure to males administered 4.3 mM NMA for 6 weeks or 1.2 mM AMD for 4 weeks in drinking water were observed. Administration of AMD also caused a reduction in the fertility of dosed males perhaps related to the decreases in sperm count and increases in abnormal sperm morphology observed immediately after exposure.

Similarity in Distribution and Metabolism

Edwards (1975b) demonstrated that blood concentration of NMA decreased, with a half-life of 1.55 hours after a single 140 mg/kg intravenous dose to rats. Extrapolation to zero time gave a concentration close to the theoretical value for distribution in total body water. No studies on the excretion of NMA have been reported, but with [vinyl-¹⁴C]-AMD, Miller et al. (1982) showed similar kinetics for removal from plasma, with an initial half-life of approximately 2 hours. Elimination of radioactivity from most tissues was biphasic, with a first-phase half-life of less than 5 hours and a second phase of about 8 days, although unmetabolized AMD could no longer be isolated from any tissue after day 1. The radioactive label was distributed as follows: muscle, 48%; skin, 15%; blood, 12%; liver, 7%; and neural tissues, less than 1%. Only erythrocytes concentrated radioactivity. Within 24 hours, 62% of the radioactivity was excreted in the urine; 71% was excreted within 7 days by this route. No [¹⁴C]carbon dioxide was observed in expired air. Fecal excretion was minimal (6% by 7 days), but within 6 hours, 15% of the radioactivity was found in the bile, suggesting enterohepatic circulation. The major labeled material found in the urine was N-acetyl-S-(3-amino-3-oxopropyl)cysteine, a product of glutathione conjugation. The only organ that showed a somewhat delayed uptake was the testis (Miller et al., 1982). This was confirmed in whole body autoradiography studies (Marlowe et al., 1986). With AMD (with the vinyl moiety labeled), accumulation in the male reproductive tract peaked in the testis within 3 hours of oral dosing, and the label appeared to move subsequently to the seminiferous tubules, to the head of the epididymis, and by 9 days to the crypts of the epithelium of the glans penis. This is not consistent with labeling of spermatogonia but could represent binding to spermatids or large molecules within the seminiferous tubules. As detailed below, Shelby et al. (1986) performed a dominant lethal test with acrylamide in various strains of mice and reported increased percentages of dead implants in a time pattern consistent with effects on late spermatids and early spermatozoa.

Similarities in Genetic Toxicity

NMA was not mutagenic in several strains of *S. typhimurium* in either the presence or absence of exogenous metabolic activation (Hashimoto and Tanii, 1985; Zeiger et al., 1988). These results, coupled with the positive dominant lethal test in mice (Sakamoto and Hashimoto, 1986) indicate an activity profile similar to AMD, which is an *in vitro* and *in vivo* eukaryotic mutagen whose clastogenic effect *in vivo* appears more pronounced in germ cells than in somatic cells (Dearfield et al., 1988). Salmonella tests with AMD generally indicate no mutagenic activity (Lijinsky and Andrews, 1980; Hashimoto and Tanii, 1985; Knaap et al., 1988), with one exception. Zeiger et al. (1988) reported a weakly positive response for AMD in *S. typhimurium*, a finding that was not reproduced in a second laboratory.

Carlson and Weaver (1985) demonstrated *in vivo* binding of AMD to DNA of lung, testis, stomach, and skin of mice, 6 hours after oral or dermal administration of radiolabeled chemical. This observation is consistent with the uniformly positive *in vivo* genotoxicity test results with AMD. Acrylamide has been shown to induce dominant lethal mutations in both rats (Smith et al., 1986) and mice (Shelby et al., 1986), inherited translocations in mice (Shelby et al., 1986).

Smith et al. (1986) observed an increase in post-implantation losses in untreated female rats mated to males that had received 30 or 60 ppm AMD in drinking water for 72 days; matings with males that had received 60 ppm also resulted in increased pre-implantation losses. No significant increase in chromosomal aberrations in spermatocytes was observed in males analyzed immediately or 12 weeks after completion of the breeding studies. The results indicate that AMD is less effective in producing clastogenic effects in spermatogenic cells than in post-meiotic sperm cells.

Shelby et al. (1986) reported induction of dominant lethal mutations in male mice mated to females. Their studies demonstrated a peak response for an increased incidence of dead implants from matings 4.5-11.5 days after males were given a single i.p. injection of 125 mg/kg. These results indicate that late spermatids and early spermatozoa are the stages most susceptible to clastogenic damage by AMD. Injection of males with AMD at 50 mg/kg per day for 5 days (total dose of 250 mg/kg) induced approximately twice the dominant lethal effect of the single 125 mg/kg dose. Using the same injection scheme, Shelby et al. (1987) reported positive results with acrylamide in the mouse heritable translocation test. They detected a high frequency of translocations in the offspring derived from matings performed 7-10 days post-injection between untreated female mice and male mice injected once per day for 5 days with 50 mg/kg AMD.

The induction of chromosomal aberrations in the germ cells of mice fed 500 ppm AMD in feed for 3 weeks is further evidence of the *in vivo* genetic effects of AMD (Shiraishi, 1978). Shiraishi was not able to demonstrate induction of chromosomal aberrations in the bone marrow cells of these same mice. However, aneuploidy and polyploidy were observed in both the bone marrow and spermatogonial cells, leading the author to suggest that AMD exerts at least some of its effect through disruption of cytoplasmic microtubules and spindle formation. No increase in the SCE frequency was observed in either tissue.

Similarity in Carcinogenicity

AMD has been reported to have carcinogenic activity in a variety of non-lifetime models using a number of routes of administration (Bull et al., 1984). However, it is the availability of 2-year toxicity and carcinogenicity studies of both AMD and NMA that provide another opportunity to evaluate their biological activity in a side-by-side comparison.

AMD was administered to F344 rats in drinking water at doses of 0, 0.01, 0.1, 0.5, or 2 mg/kg/day (Johnson et al., 1986). NMA was administered in water *but by gavage* to F344 rats and B6C3F1 mice at doses of 0, 6, or 12 mg/kg/day and 0, 25 or 50 mg/kg/day, respectively, 5 days per week (NTP, 1989).

AMD treated female rats, were observed to have increased tumor incidences in the mammary gland, central nervous system, thyroid gland follicular epithelium, oral tissues, uterus, and clitoral gland. Male rats treated with AMD showed increased tumors of the scrotal mesothelium, thyroid gland follicular epithelium, and central nervous system (Johnson et al., 1986).

NMA treated rats displayed no biologically important non-neoplastic or neoplastic lesions compared to the concurrent controls (NTP, 1989). This is in spite of the fact that the high dose of NMA was 24 times higher than the dose of AMD (0.5 mg/kg/day) at which a carcinogenic effect was clearly detected and 120 times higher than the AMD dose (0.1 mg/kg/day) at which tumor incidences exceeded historical and concurrent control values. Furthermore, the highest NMA dose (12 mg/kg/day) produced no effects whatsoever (over a 2 yr. period) while the same dose of AMD (12.5 mg/kg/day) produced significant neurotoxicity after only 13 wk. of administration. Part of the explanation of these findings may be related to the relative purity of the NMA employed. In this study, the test article was determined to be at least 98.1% NMA and the largest impurity (approximately 0.1%) appeared to be a NMA polymer. Therefore, the concentration of AMD had to be <0.1% (compared to $\leq 5\%$ in commercially available NMA products) and its potential contribution to the overall biological effect had to be extremely small.

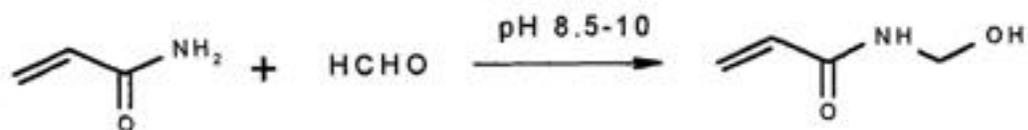
Mice treated with NMA at levels 50 or 100 times the AMD levels administered to rats showed an increased incidence of adenomas (but not carcinomas) of the mouth (Harderian gland), hepatocellular adenomas and carcinomas (individually or combined), alveolar/bronchiolar adenomas or carcinomas (although this finding is confounded by the presence of Sendai virus), neoplasms of the ovary, and adenomas of the pars distalis.

Similarity in Chemical Reactivity and Catabolism

AMD is a crystalline solid, which is chemically stable at room temperature. Aqueous solutions of AMD are generally stable at room temperature. However, in the presence of free radicals it undergoes polymerization. Therefore, inhibitors are added to aqueous solutions to stabilize acrylamide.

Aqueous solutions of AMD hydrolyze in the presence of strong acids or strong bases.

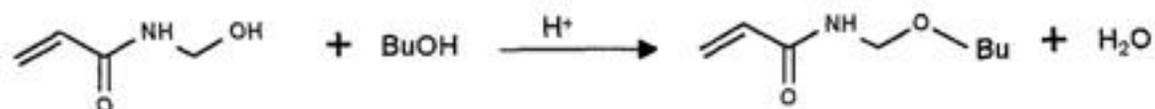
NMA is derived from AMD by reaction with formaldehyde at alkaline pH.



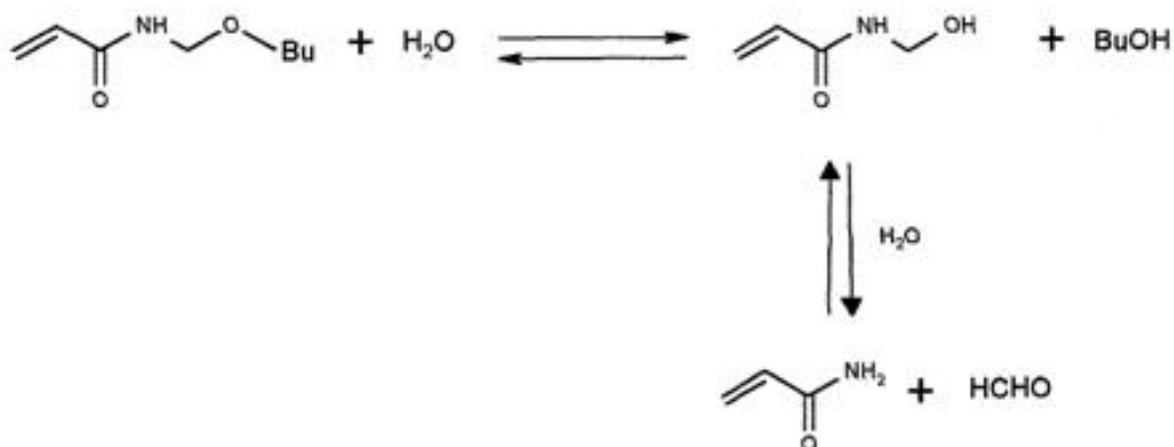
Dilute aqueous solutions of NMA are unstable at neutral pH conditions and undergo de-methylolation to AMD and formalin.



Alkylation of NMA with butanol under acidic conditions yields NBMA as shown. The reaction is driven to the right by employing a large excess of butanol and by removing the by-product water.



Dilute aqueous solutions of NBMA undergo hydrolysis slowly to give a mixture of NMA, AMD and formaldehyde.



ISSUE #2: PHYSIOCHEMICAL AND ENVIRONMENTAL FATE DATA

EPA COMMENT:

- (a) The submitter needs to correct the reported water solubility estimate for NBMA.
- (b) The submitter needs to address inconsistent statements regarding biodegradation of NMA.
- (c) The submitter needs to provide input values for transport and distribution (fugacity) estimations.

ASSOCIATION RESPONSE:

- (a) The Agency is correct; there is a typographical error in the original document; the water solubility was reported as 1.2×10^{-4} when it really is 1.2×10^4 ; thank you for noting this error (see Attachment 1, revised HPV Test Plan, Section 3.1).
- (b) This has been addressed in Attachment II (revised HPV Test Plan, Section 3.2.3)
- (c) The input values for Transport and Distribution (fugacity) are those that are recommended in the EPA EPIWIN model. These are presented in Attachment III.

ISSUE #3: HEALTH ENDPOINTS

EPA COMMENT:

- (a) The submitter proposed that no additional testing is needed for NMA and NBMA, because adequate data for AMD are available that could be used to address the health effects endpoints for NMA and NBMA. However, as stated above, the submitter needs to provide a stronger justification for using AMD as a structural analog.
- (b) Until this information is provided, EPA considers the developmental toxicity endpoint unaddressed and additional testing may be necessary.

ASSOCIATION RESPONSE

- (a) The requested information has been provided (see Issue #1 above)
- (b) The requested information has been provided (see Issue #1 above)

ISSUE #4: ECOTOXICITY

EPA COMMENT:

The submitter considered the existing data for AMD, NMA, and NBMA adequate to address ecotoxicity endpoints for NMA and NBMA. However, given the absence of aquatic invertebrate and algae testing for NMA and NBMA, and the failure of the submitter to demonstrate the relevance of the AMD data, EPA recommends that additional acute toxicity testing is necessary on aquatic invertebrate and algae for at least NBMA.

ASSOCIATION RESPONSE

The information submitted in this document demonstrates the relevance of the AMD data.

Bernard to Hernandez
EPA HPV Program
NMA/NBMA Response
30 September 2002 p. 10

ADDITIONAL ISSUE (#5): TYPOGRAPHICAL ERROR

EPA COMMENT:

None

ASSOCIATION RESPONSE:

In reviewing the test plan, an error was noted in Section 3.4.1 wherein the AMD acute dermal LD50 in rabbits was stated to be 1.88 mg/kg; it should have been 1.88 g/kg. This has been revised in Section 3.4.1 of the test plan (see Attachment IV) and in the AMD IUCLID submission, Section 5.1.3 (see Attachment V).

Sincerely,

1

Bruce K. Bernard, Ph.D.
President, SRA International Inc.
Authorized Representative for the
NMA/NBMA Association

ATTACHMENT I

3. Test Plan

As part of this HPV submission, the Agency will find three sections containing robust summaries of data related to the three chemicals (AMD, NMA and NBMA) which are the basis of this submission. The data are provided in the IUCLID format as suggested by the Agency. The following test plan summarizes those studies that the submitter believes provide the most significant information. It is not the intent of the test plan to refer to all studies listed in the data sets. However, summaries of additional studies not employed in the test plan are available to the reviewer in the data summaries.

3.1 Chemical and Physical Properties

Chemical/physical properties are summarized in Table 1.

Table 1. Chemical/physical properties of AMD, NMA, and NBMA

Endpoint	AMD (CAS # 79-06-1)	NMA (CAS # 924-42-5)	NBMA (CAS # 1852-16-0)
Melting point	83.75 °C * 84.5° + 0.3 °C	69.5 °C * 74-75 °C	79.9 °C *
Boiling point	87 °C @ 2.03mmHg 103 °C@ 5.03mmHg 116 °C@ 10.5mmHg 136 °C@ 24.8mmHg	276.5 °C * 100 °C ***	296.53 °C * 118-143 °C **
Vapor pressure	0.007mmHg @ 25°C 0.033mmHg @ 40°C 0.08mmHg @ 50°C	0.00023mmHg @ 25 °C * 23.76mmHg @ 25 °C ***	0.000704mmHg @ 25 °C *
Partition coefficient (Log Pow or Kow)	-0.9	-1.81*	.92*
Water solubility	2155 g/l at 30° C 4260 g/l at 50° C	1,220 g/l @ 10° C 1,880 g/l @ 20° C 3,540 g/l @ 40° C 7,550 g/l @ 60° C	12 g/l @ 25°C*

* Values obtained by EPIWIN

** Values obtained using a 50% solution in butanol

*** Values obtained using a 48% aqueous solution

3.1.1 Melting Point

Melting point determinations by modeling (Klimisch et al., 1997; Syracuse Research Corporation 1998) suggest that all these compounds have moderate melting points (70° C - 85° C). Experimental studies in AMD (Van der Burg 1922, Kirk-Othmer 1991, Carpenter and Davis 1957, The Merck Index, 12th Ed.) and NMA (Feurr and Lynch 1953) confirm the accuracy of the modeling predictions.

ATTACHMENT II

3.2.2 Stability in Water

The EPWIN model predicts that these compounds are stable in water (i.e. resistant to hydrolysis) with half-lives estimated at greater than one year (Table 2). This is substantiated by experimental results on AMD (Brown et al., 1980; Jung et al., 1980). Data is also available for a wide variety of pH's in an aqueous environment (Moens and Smets, 1957).

3.2.3 Biodegradation

The conclusion reached by modeling studies indicates that all three compounds will biodegrade rapidly in water (Klimisch et al., 1997; Syracuse Research Corporation 1998). Experimentally derived data support this conclusion (AMD: USTC 1991, Birdie et al., 1979, Winter and Wolff 1982, Brown et al., 1982, Lande et al., 1979, Yamada et al., 1979, Brown et al., 1980b, Arai et al., 1981, Dow 1975, Brown et al., 1980c, Batchelder 1975, and Croll et al., 1974; NMA and NBMA: Wang, 1991). The results in water are consistent with evidence that AMD rapidly degrades in soils under various conditions (Abdelmagid and Tabatabai, 1982). NMA degraded only 51.9% after 28-days when tested using activated sludge in the closed bottle model; not meeting the definition of readily biodegradable. Under the same conditions, NBMA was found to be readily biodegradable achieving 98.1% degradation in 28-days.

3.2.4 Fugacity

Estimation of relative distribution of a chemical released into various environmental compartments can be estimated using the Mackay Level III fugacity model (Klimisch et al., 1997; Syracuse Research Corporation 1998). This model cannot be employed to predict actual environmental concentrations. One of the key assumptions underlying this model, is the assumption of zero loss of material through degradation or dispersion out of the environmental system. When applied to AMD, NMA and NBMA, the model predicts that all three compounds partition primarily to soil and to a slightly lesser degree to water. Partition to sediment and air is negligible (Table 3).

Table 3. MacKay Level III fugacity model

Medium	AMD, (CAS # 79-06-1)	NMA, (CAS # 924-42-5)	NBMA, (CAS# 1852-16-0)
	Concentration %	Concentration %	Concentration %
Air	0.032	0.000307	0.177
Water	45.3	45.3	44.5
Soil	54.3	54.6	55.3
Sediment	0.07571	0.0755	0.0794

3.2.5 New Testing Required

All endpoints have been met by experimentation or use of EPIWIN. No further testing is required.

ATTACHMENT III (pg. 1/3)

The AMD Level III Fugacity Model values are:

	<u>Mass Amount (percent)</u>	<u>Half-Life (hr)</u>	<u>Emissions (kg/hr)</u>
Air	0.032	20	1000
Water	45.3	360	1000
Soil	54.5	360	1000
Sediment	0.0757	1.44e+003	0
Persistence Time: 418 hr			

	<u>Mass Amount (percent)</u>	<u>Half-Life (hr)</u>	<u>Emissions (kg/hr)</u>
Air	0.09	20	1000
Water	27.9	360	0
Soil	72	360	0
Sediment	0.0465	1.44e+003	0
Persistence Time: 446 hr			

	<u>Mass Amount (percent)</u>	<u>Half-Life (hr)</u>	<u>Emissions (kg/hr)</u>
Air	4.1e-007	20	0
Water	99.8	360	1000
Soil	0.000328	360	0
Sediment	0.167	1.44e+003	0
Persistence Time: 342 hr			

	<u>Mass Amount (percent)</u>	<u>Half-Life (hr)</u>	<u>Emissions (kg/hr)</u>
Air	0.000104	20	0
Water	22	360	0
Soil	77.9	360	1000
Sediment	0.0368	1.44e+003	0
Persistence Time: 466 hr			

ATTACHMENT III (pg. 2/3)

The NMA Level III Fugacity Model values are:

	<u>Mass Amount</u> <u>(percent)</u>	<u>Half-Life</u> <u>(hr)</u>	<u>Emissions</u> <u>(kg/hr)</u>
Air	0.000307	8.59	1000
Water	45.3	360	1000
Soil	54.6	360	1000
Sediment	0.0755	1.44e+003	0

Persistence Time: 421 hr

	<u>Mass Amount</u> <u>(percent)</u>	<u>Half-Life</u> <u>(hr)</u>	<u>Emissions</u> <u>(kg/hr)</u>
Air	0.000853	8.59	1000
Water	28	360	0
Soil	71.9	360	0
Sediment	0.0467	1.44e+003	0

Persistence Time: 453 hr

	<u>Mass Amount</u> <u>(percent)</u>	<u>Half-Life</u> <u>(hr)</u>	<u>Emissions</u> <u>(kg/hr)</u>
Air	3.73e-011	8.59	0
Water	99.8	360	1000
Soil	3.15e-006	360	0
Sediment	0.166	1.44e+003	0

Persistence Time: 342 hr

	<u>Mass Amount</u> <u>(percent)</u>	<u>Half-Life</u> <u>(hr)</u>	<u>Emissions</u> <u>(kg/hr)</u>
Air	9.7e-009	8.59	0
Water	22.2	360	0
Soil	77.8	360	1000
Sediment	0.0369	1.44e+003	0

Persistence Time: 466 hr

ATTACHMENT III (pg. 3/3)

The NBMA Level III Fugacity Model values are:

	<u>Mass Amount (percent)</u>	<u>Half-Life (hr)</u>	<u>Emissions (kg/hr)</u>
Air	0.177	4.93	1000
Water	44.5	360	1000
Soil	55.3	360	1000
Sediment	0.0794	1.44e+003	0

Persistence Time: 380 hr

	<u>Mass Amount (percent)</u>	<u>Half-Life (hr)</u>	<u>Emissions (kg/hr)</u>
Air	0.62	4.93	1000
Water	24.2	360	0
Soil	75.1	360	0
Sediment	0.0433	1.44e+003	0

Persistence Time: 324 hr

	<u>Mass Amount (percent)</u>	<u>Half-Life (hr)</u>	<u>Emissions (kg/hr)</u>
Air	1.45e-005	4.93	0
Water	99.8	360	1000
Soil	0.00176	360	0
Sediment	0.178	1.44e+003	0

Persistence Time: 342 hr

	<u>Mass Amount (percent)</u>	<u>Half-Life (hr)</u>	<u>Emissions (kg/hr)</u>
Air	0.00271	4.93	0
Water	18.3	360	0
Soil	81.7	360	1000
Sediment	0.0327	1.44e+003	0

Persistence Time: 474 hr