MICHIGAN DEPARTMENT OF NATURAL RESOURCES & ENVIRONMENT

INTEROFFICE COMMUNICATION

TO: File for 2-Diethylaminoethanol (CAS # 100-37-8)

FROM: Doreen Lehner, Toxics Unit, Air Quality Division

DATE: July 10, 2015

SUBJECT: Screening Level for 2-Diethylaminoethanol (CAS # 100-37-8)

The initial threshold screening level (ITSL) for 2-diethylaminoethanol is 4 µg/m^3 based on an annual averaging time.

This ITSL was originally set as 4 µg/m^3 based on a 24-hour averaging time and established on 2/27/1996 based on a well conducted 14 week inhalation study by Hinz et al., (1992) using male and female Fischer 344 rats (20/sex/dose) exposed to 0, 10, 25, or 75 ppm (0, 49, 122, or 365 mg/m^3). The most sensitive effect of mixed inflammatory cell infiltration (upper respiratory irritation) gave a NOAEL of 53.6 mg/m^3. As this study was a 14 week study, an RfC was derived and given a 24-hour averaging time, according to Rule 232(2)(b). This study is considered a sub-chronic 14-week study and according to Rule 229(2)(b), it would be more appropriate to utilize a longer averaging time, which would be an annual averaging time. Therefore, the ITSL for 2-diethylaminoethanol is 4 µg/m^3 based on an annual averaging time.

References:

Act 451 of 1994, Natural Resources and Environmental Protection Act and Air Pollution Control Rules, Michigan Department of Environmental Quality

Hinz JP, Thomas JA, and Ben-Dyke R. 1992. Evaluation of the inhalation toxicity of diethylethanolamine (DEEA) in rats. *Fund appl Toxicol* 18(3):418-424.

MICHIGAN DEPARTMENT OF ENVIRONMENTAL QUALITY

INTEROFFICE COMMUNICATION

February 27, 1996

TO: File for 2-Diethylaminoethanol (2-DEAE) [CAS # 100-37-8]

FROM: Dan O'Brien

SUBJECT: Initial Threshold Screening Level for 2-DEAE

The initial threshold screening level (ITSL) for 2-diethylaminoethanol is 4 $\mu g/m^3$ based on a 24 hour averaging time.

The following references or databases were searched to identify data to determine the ITSL: AQD chemical files, IRIS, HEAST, ACGIH TLV Booklet, NIOSH Pocket Guide to Chemical Hazards, RTECS, NTP Management Status Report, EPB Library, IARC Monographs, CAS On-line and NLM/Toxline (1967 -November 6, 1995), CESARS, Handbook of Environmental Data on Organic Chemicals, Patty's Industrial Hygiene and Toxicology, Merck Index and Condensed Chemical Dictionary.

2-DEAE is a colorless, hygroscopic liquid combining the properties of amines and alcohols (Hawley, 1981). It is corrosive with a sharp, ammoniacal odor; the odor threshold lies in the range from 0.01-0.25 ppm (Benya and Harbison, 1994). It is used in water soluble salts; fatty acid derivatives; textile softeners; pharmaceuticals; antirust compositions; emulsifying agents in acid media; derivatives containing tertiary amine groups; as a curing agent for resins; and for extraction of hydrogen sulfide and carbon dioxide from natural gas (ACGIH, 1994).

With respect to acute toxicity, 2-DEAE is considered slightly toxic orally for laboratory animals (Deichmann, 1969), with a rat LD_{50} listed as 1.3 g/kg (RTECS, 1995). Neutralizing the agent increases the LD_{50} to 5.6 g/kg (Cornish, 1965). An inhalation LC_{50} of Russian origin is listed at 5 g/m3 for mice (RTECS, 1995). It is a severe eye and skin irritant, a skin sensitizer in the guinea pig and is capable of causing permanent eye damage. (Deichmann, 1969).

Two longer term rodent inhalation studies were located in our search of the noted references, a recent 14 week study by Hinz *et al.* (1992), and a somewhat older work by Cornish (1965). The Cornish study used only one exposure concentration, 200 ppm (974 mg/m^3), a level at which there was frank toxicity, including mortality in 7/50 male Sprague-Dawley rats during the first month of exposure. Animals were exposed 6 hours per day, 5 days per week. Necropsy of the deceased demonstrated bronchopneumonia with no other abnormalities among the examined tissues. Among those that survived, there was "mild eye and nasal irritation', as well as "considerable depression of growth rate" after 1 month of exposure, but other monitored toxicity endpoints were not markedly

different from those in controls. By the end of 6 months of exposure, the exposed animals were considered comparable to controls with respect to body and organ weights, hematology, serum protein, aspartate transaminase (AST) and histopathology. The lack of detail in the experimental protocol and the lack of exposure levels which might have defined a toxicity threshold limit the usefulness of this study in derivation of a screening level. Nonetheless, it notes two potentially important points: 1) Respiratory irritation in the absence of systemic toxicity as a critical effect and 2) The potential for acclimation to 2-DEAE-induced irritation over time.

In the study by Hinz and coworkers (Hinz *et al.*, 1992; EPA-OTS, 1990), groups of 20 male and 20 female Fischer 344 rats, 10 weeks old at the start of dosing, were exposed to target concentrations of 0, 10, 25 or 75 ppm (0, 49, 122 or 365 mg/m^3), 6 hours per day, 5 days per week for up to 14 weeks. Dose levels for this trial were chosen based on the results of a 2 week study utilizing the same experimental protocol and also described in this publication. The actual daily mean (± standard deviation) chamber concentrations during exposure (as determined by gas chromatographic analysis) were 0, 11 (± 2.1), 25 (± 2.1) and 76 (± 3.4) ppm, respectively. Half of the animals in each exposure group were terminated during the 14th week of exposure, while those remaining were sacrificed following a 4-week postexposure recovery period. Endpoints monitored included observations for clinical signs and recording of body weights (weekly); food and water consumption determination; ophthalmologic exams; neurobehavioral evaluations (monthly); comprehensive urinalysis (obtained during the period from 16 hours prior to termination and the time of euthanasia); hematology and serology; and assays for brain, plasma and erythrocyte cholinesterase (from samples obtained at termination). A full complement of tissues were obtained for histopathology at necropsy; all of these tissues from the control and 75 ppm groups were subjected to histopathological examination, along with the nasal turbinates and respiratory tracts of the 10 and 25 ppm groups. Statistical analyses consisted of evaluations for homogeneity of variances (Bartlett's test), followed by 1) ANOVA (with multiple comparisons by Dunnett's test) for group comparisons and linear regression for assessment of dose-response trend in cases where variances were homogeneous, or 2) Kruskal-Wallis (with multiple comparisons by Dunn's summed rank test) for group comparisons and Jonckheere's test for dose-response trend where variances were not homogeneous.

With one notable exception, clinical observations, neurobehavioral assessment, body weights and food and water consumption did not show a pattern of exposure-related differences. The exception was that signs of respiratory irritation (rales, "sneeze-like" noises) were noted in all exposed animals, with time to onset inversely related to exposure concentration. These signs were transient, persisting less than 1 hour generally, but sometimes overnight. There was no mortality. There was a high incidence of calcified corneal opacities. These also developed sooner in the higher exposure concentrations, with all of the 76 ppm group rats effected after 1 month of exposure, most of the 25 ppm group effected after 2 months, and all animals (including controls) effected to some degree by the end of the study. None of the rats exhibited

corneal lesions at the start of the study. Rats in the highest exposure group exhibited statistically significantly slower growth compared to controls through the seventh week of the study, and this body weight decrement was never recovered. Urinalysis, hematology, serology and cholinesterase assays showed no biologically significant exposure-related differences between exposed animals and controls. In those animals necropsied after 14 weeks, there were no gross lesions; however, in the high dose males, absolute and relative kidney weights were significantly elevated. After 4 weeks of recovery period, there was no pattern of exposure-related macroscopic findings nor organ weights at any dose level. Exposure-related histopathologic lesions were limited to the upper respiratory tract, where lesions were seen only in the 25 and 76 ppm exposure groups (with the exception of inflammatory cell infiltration, seen in all groups), and included epithelial hyperplasia, squamous metaplasia, goblet cell hypertrophy, inflammatory cell infiltration, focal mucosal necrosis and exudates in the lumen of the passages. The most sensitive effect was mixed inflammatory cell infiltration, which was noted in 4/10 males and 3/10 females in the 25 ppm group, and 5/10 males and 10/10 females in the 76 ppm group; it was also present in 3/10 control and 4/10 10 ppm females. After 4 weeks of recovery, inflammatory cell infiltrations were present in all exposure groups of both sexes, but otherwise, the incidences of lesions showed a tendency toward a slight decline. Incidences of squamous metaplasia, for example, declined from 3/10 males and 2/10 females in the 25 ppm group at 14 weeks to 1/10 males and 2/10 females after 4 weeks recovery time. The authors note that "the lack of any systematic toxicity suggests that the initial and principal site of action of diethylaminoethanol is limited to its site of contact" and "based on the lack of respiratory lesions, the no-observed effect level (NOAEL) in these studies was 10 ppm (49 mg/m^3)".

ACGIH (1994) cites chronic oral studies conducted by Atochem (1990), a 1-yr exposure to small (3 males and 3 females at each dose level) groups of beagle dogs, and a 2-yr exposure to rats. Both were feeding studies; dose levels in the dog study were 500, 1000, 5000 (lowered to 2000 after 134 days) and 10,000 ppm, while dose levels in the rat study were 0, 200, 500 and 1000 ppm. Chief effects in the canine study were mortality (2/6 and 6/6 in the 5000 and 10,000 ppm groups, resp.) and "severe cases of weakness, tremors, convulsions, and ataxia". All dogs in the 1000 ppm group exhibited "tremors and/or shaking of the head at various times". The only reported effect in the rat study was testicular atrophy (incidences were 0/34, 3/18, 2/17 and 4/15 in the 0, 200, 500 and 1000 ppm groups, resp.). No statistical analyses are reported, but it is questionable whether the differences between the exposed groups would be significant. The NOAEL for the dog study was listed as 500 ppm; no NOAEL is listed for the chronic rat study.

No data were located which suggested carcinogenic effects due to 2-DEAE exposure. Although RTECS cites tumorigenic effects in the reproductive system associated with the Cornish (1965) and Hinz *et al.* (1992) studies, neither study reports such effects. In the former, reproductive organs were not obtained at necropsy; in the latter, the only histopathological abnormalities reported were confined to the upper

respiratory tract. The compound has tested negative in the *Salmonella* mutagenicity assay (NTP, 1995; Zeiger *et al.*, 1987).

Some accounts of the toxicity of 2-DEAE to humans following acute exposures were found in the literature, all documented as episodes of accidental exposure. The earliest is reported by Cornish (1969), and involved a laboratory worker who experienced nausea and vomiting within five minutes of exposure to concentrations ≤ 200 ppm (960 mg/m^3); the worker was removing test animals from an exposure chamber from which the test agent had been inadequately evacuated. Cornish, using an unspecified method, determined that the likely exposure concentration was approximately 100 ppm (487 mg/m^3). The second account described rapid onset of headache, nausea, vomiting, dizziness, and eye, nose and throat irritation in 77 of 121 workers at an Ohio electronics manufacturer following the release of boiler steam to indoor air for humidification (Hills *et al.*, 1990; MMWR, 1990). Employees reported an odor coinciding with the steam release and preceding the onset of clinical signs. 2-DEAE and cyclohexylamine had been added to the boiler water at four times normal strength three months earlier as corrosion inhibitors. Eleven workers were sickened severely enough to be sent to hospital, though none were admitted. A NIOSH investigation was unable to detect 2-DEAE in boiler steam released the following day, although previous investigations of three similar incidents had reportedly recorded air concentrations of 0.04-0.05 mg/m^3. NIOSH speculated that dermal exposure to 2-DEAE condensate on surfaces may have been a significant route of toxicity, since air concentrations were quite low. This hypothesis was supported by reports of dermatitis in intoxicated workers in previous incidents, although dermatitis was not noted in the Ohio disease outbreak. A third account (Gadon *et al.*, 1994) consists of a case series of workers exposed to 2-DEAE from a heating system steam leak in a large government office building. "Within hours", symptoms of irritation were experienced by most of the 2500 employees. Forty-nine were referred to local emergency rooms. Over the next three months, asthma (according to the NIOSH surveillance case definition for occupational asthma) was diagnosed for the first time in fourteen employees. Seven of these were considered confirmed cases and another seven suspect (as diagnosed by peak flow testing (positive in 10/14), spirometry (positive in 4/14), and/or occurrence of work-related, post-exposure respiratory symptoms alone (3/14)). Air monitoring using Hydrazine Drager tubes for non-specific amines was carried out, but no specific 2-DEAE concentrations were reported, only that "air monitoring did not detect a high level of ambient chemical", perhaps because monitoring was not initiated until three days after the initial leak. Additionally, 5/7 of the confirmed asthmatics were cigarette smokers, as were 4/7 of the suspects, and the design of the study did not allow for assessment of the effect of smoking on the observed respiratory signs. So, while this study is not in itself adequate for quantitative use in screening level development, the authors nonetheless validly point out that 2-DEAE could plausibly have been a factor in these new-onset cases of occupational asthma because 1) it is an established respiratory irritant and dermal sensitizer (at least in animals); 2) it is quite similar in structure to dimethylethanolamine, monoethanolamine and aminoethylethanolamine, all of which have been associated with cases of occupational asthma and 3) a plausible mechanism (occupational asthma

due to irritant-induced bronchial hyperreactivity) has been previously established. This evidence, while not definitive, must still be born in mind when considering potential human health risks from 2-DEAE exposure.

Occupational Exposure Limits (OELs) for 2-DEAE have been established both by the American Conference of Governmental Industrial Hygienists [ACGIH] (ACGIH, 1994) and by the National Institute for Occupational Safety and Health [NIOSH] (NIOSH, 1992). The ACGIH Threshold Limit Value (TLV) is 2 ppm (9.6 mg/m^3) on a time-weighted average (TWA) and carries a "skin" designation, indicating that there is a potentially significant contribution to overall exposure *via* the cutaneous route. Indeed, two fairly recent publications in which potential dermal toxicity in humans was modeled both concluded, using different methods, that 2-DEAE had significant potential for dermal toxicity (Guy and Potts, 1993; Fiserova-Bergerova *et al.*, 1990). The NIOSH Recommended Exposure Limit (REL) is 10 ppm (50 mg/m^3), also TWA and carrying a "skin" designation. NIOSH has also set an Immediately Dangerous to Life or Health (IDLH) concentration of 100 ppm (487 mg/m^3). The current TLV is a recent revision of previous TLV of 10 ppm (48 mg/m^3) that was in effect from 1991 to 1994. The principal difference between the current TLV documentation and its predecessor is the citation of the chronic studies conducted by Atochem (1990) which were not available for our review. In addition, although the revision lowered the TLV from 10 to 2 ppm, the draft revised documentation places conspicuously less emphasis on irritation as a critical effect than did the 1991 documentation. This is somewhat puzzling, considering that the available data suggest sensory and upper respiratory irritation is the most sensitive health endpoint. The revised documentation also states that "this threshold for DEAE has to be seen as preliminary because the toxicological profile of DEAE is not well established".

With respect to selection of a key concentration to drive the derivation of the screening level, either the TLV of 9.6 mg/m^3 or the NOAEL from Hinz *et al.* (1992) could be used. While the use of the OEL theoretically has the advantage of being based on human occupational exposure experience, in this case the draft TLV documentation cites Hinz *et al.* (1992) as "the only study that can be used for an evaluation" of longer term exposures at low dose levels. Moreover, the draft TLV documentation does not discuss the health effects exhibited by humans who were exposed acutely in the more recent reports of accidental releases (Gadon *et al.*, 1994; Hills *et al.*, 1990; MMWR, 1990). Consequently, the use of the TLV does not appear to hold any particular advantage with respect to minimizing the uncertainty due to interspecies extrapolation, since the TLV does not appear to be based on human data. The Hinz *et al.* report, on the other hand, appears to have been rigorously conducted, and at 14 weeks duration, satisfies the minimum criteria necessary for derivation of an inhalation RfC (EPA, 1990)[1].

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[1] The 1990 Interim RfC Methods are used in this case, even though the more recent Final Methods (EPA, 1994) are currently available. Methodological inconsistencies have become apparent during application of the 1994 guidelines to inhalation risk assessments for other chemicals. These inconsistencies have been communicated to EPA, which

Since the derivation of an ITSL based on an inhalation RfC is given precedence over one based on an OEL, the Hinz study is used here for development of an ITSL for 2-DEAE.

Human Equivalent Concentration (HEC) Calculation:

a) The key study NOAEL, based on the actual chamber concentration of 11 ppm, is converted to mg/m^3, using the chemical-specific conversion factor (1 ppm = 4.87 mg/m^3) of Verschueren (1983). Thus, the NOAEL = 53.6 mg/m^3.

b) Dose adjustment is necessary to account for discontinuous exposure regimens used in the key study. Per EPA (1990), section 4.1.1.2, p. 4-13:

$$NOAEL_{[ADJ]}\ (mg/m^3) = 53.6\ mg/m^3 \times \frac{6\ hrs/day}{24\ hrs/day} \times \frac{5\ days/week}{7\ days/week}$$

$$= 53.6\ mg/m^3 \times 0.25 \times 0.71 = 9.57\ mg/m^3$$

c) 2-DEAE is absorbed dermally and orally, penetrates to the bloodstream and is capable of causing systemic signs at higher exposure concentrations, suggesting that it is soluble enough in tissues to exert at least some systemic effects. However, review of the available data suggests that the most sensitive health endpoint associated with both animal and human exposure is eye and upper respiratory irritation. The prominence of irritative signs in the upper respiratory tract at lower vapor concentrations (Hinz *et al.*, 1992), and the limitation of lesions to the respiratory tract even at concentrations which caused overt mortality [200 ppm] (Cornish, 1965) suggests that the systemic toxic effects of 2-DEAE following inhalation exposure are not prominent, or may be secondary to respiratory compromise. This fact, in conjunction with the chemical's water solubility, suggests classification as a rapidly reactive gas. In addition, although lower respiratory effects clearly can occur if exposure concentrations are sufficiently high, the results of Hinz and coworkers suggest that they do not occur at low 2-DEAE concentrations. Therefore, an RfC based on prevention of upper respiratory irritation should also be protective of lower respiratory injury. Consequently, the NOAEL Human Equivalent Concentration ($NOAEL_{[HEC]}$) is derived using the default Regional Gas Dose Ratio ($RGDR_{[ET]}$) model for extrathoracic (ET) respiratory effects of a reactive gas (per EPA (1990), section 4.1.1.2). The default equation (4-9) is used here.

$$NOAEL_{[HEC]}\ (mg/m^3) = NOAEL_{[ADJ]}\ (mg/m^3) \times RGDR_{[ET]}$$

where RGDR is the ratio of regional gas doses (RGD_{animal}/RGD_{human}) in the test animal species to that in humans for the region of

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has promised review of the issues involved and guidance once the review is complete. When these issues have been resolved satisfactorily, the RfC and ITSL calculated here may be re-evaluated in light of that guidance.

interest for the toxic effect. Per EPA (Guth, 1995), the RGD for each species is determined:

$$RGD = \frac{V_E}{S_{ET}}$$

where V_E is the ventilation rate (m^3/day) and S_{ET} = regional surface area (cm^2) of the toxic effect observed. In this case, since the toxic effects observed in the Hinz study were all in the nasal cavity and turbinates, the value used for S_{ET} reflects the surface area of the extrathoracic (ET) airways. The values of V_E are specific to sex, species, strain and duration of the experiment. These values were obtained from conversations with Dr. Dan Guth (Guth, 1995), of the EPA Offices of Research and Development and Health Risk Assessment. Since there was no apparent differential gender sensitivity for 2-DEAE nasal lesions in the Hinz *et al.* (1992) study, the mean of the $V_{E(animal)}$ values for male and female F344 rats in a subchronic exposure scenario is used to calculate the RGDR. The current EPA default $V_{E(human)}$ of 20 m^3/day is also used. As for the values of S_{ET} the default surface area values (Guth, 1995) for respiratory effects of EPA are used.

So,

$$RGD_{animal} = \frac{0.165\ m^3/day}{15\ cm^2} = 0.011$$

$$RGD_{human} = \frac{20\ m^3/day}{200\ cm^2} = 0.10$$

The $RGDR_{[ET]}$ is thus determined

$$RGDR_{[ET]} = \frac{RGD_{animal}}{RGD_{human}} = \frac{0.011}{0.10} = 0.11$$

Consequently,

$$NOAEL_{[HEC]}\ (mg/m^3) = NOAEL_{[ADJ]}\ (mg/m^3) \times RGDR_{[ET]}$$

$$= 9.57\ mg/m^3 \times 0.11$$

$$= 1.05\ mg/m^3$$

Inhalation Reference Concentration (RfC) calculation:

Per EPA (1990), section 4.1.1, pp. 4-4 to 4-5:

$$RfC = NOAEL_{[HEC]} / (UF \times MF)$$

$$= \frac{1.05\ mg/m^3}{([3 \times 10 \times 10] \times 1)} = 0.0035\ mg/m^3$$

where the total UF of 300 is composed of 2 10-fold uncertainty factors to account for extrapolation from healthy humans to sensitive humans and

extrapolation from the subchronic NOAEL of Hinz *et al.* (1992) to a chronic NOAEL, and a 3-fold factor for interspecies extrapolation from rats to humans. The use of a 3-fold interspecies factor instead of the more traditional 10-fold factor is considered appropriate here, since the dosimetric adjustment of the $NOAEL_{(HEC)}$ by the RGDR should already correct the $NOAEL_{(HEC)}$ for uncertainty due to interspecies pharmacokinetic differences, prior to the application of uncertainty factors. The MF assumes the default value of 1.

Derivation of the ITSL:

Applying Rule 232(1)(a) of Article II, Chapter 1, Part 55 of Act 451, the ITSL for 2-DEAE equals the inhalation RfC. Therefore:

$$\textbf{ITSL} = \text{RfC} = 0.0035\ \text{mg/m}^3 \times \frac{1000\ \mu\text{g}}{1\ \text{mg}} = 3.5\ \mu\text{g/m}^3 \cong \mathbf{4\ \mu g/m^3}$$

and per rule 232(2)(b), a **24 hour averaging time** applies.

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cc: W. Presson