Ethyldimethylamine

(CAS No: 598-56-31)

Health-based Reassessment of Administrative Occupational Exposure Limits

Committee on Updating of Occupational Exposure Limits,
a committee of the Health Council of the Netherlands

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1 Introduction

The present document contains the assessment of the health hazard of ethyldimethylamine by the Committee on Updating of Occupational Exposure Limits, a committee of the Health Council of the Netherlands. The first draft of this document was prepared by H Stouten, M.Sc. (TNO Nutrition and Food Research, Zeist, the Netherlands).

The evaluation of the toxicity of ethyldimethylamine has been based on the review by Åkesson (Åke91). Where relevant, the original publications were reviewed and evaluated as will be indicated in the text. In addition, in April 1999, literature was searched in the on-line databases Medline, Toxline, Chemical Abstracts, and NIOSHTIC, starting from 1966, 1965, 1967, and 1973, respectively, and using the following key words: dimethylaminoethane, dimethylethylamine, ethyldimethylamine, and 598-56-1.

In February 2001, the President of the Health Council released a draft of the document for public review. Comments were received from the following individuals and organisations: H Lindeman (Bayer AG, FRG) and L Whitford (Health and Safety Executive, London, UK). These comments were taken into account in deciding on the final version of the document.

An additional search in Toxline and Medline in January 2004 did not result in information changing the committee's conclusions.

2 Identity

name	:	ethyldimethylamine (EDMA)
synonyms	:	*N*,*N*-dimethylethylamine; dimethylethylamine; *N*-ethyldimethylamine; ethanamine, *N*,*N*-dimethyl-; ethylamine, *N*,*N*-dimethyl-; methanamine, *N*-ethyl-*N*-dimethyl-
molecular formula	:	$C_4H_{11}N$
structural formula	:	$(CH_3)_2N\text{-}CH_2\text{-}CH_3$
CAS number	:	598-56-31

3 Physical and chemical properties

molecular weight	:	73.1
boiling point	:	37°C
melting point	:	-140°C
flash point	:	-36°C (closed cup)
vapour pressure	:	at 20°C: 55 kPa
solubility in water	:	soluble
log $P_{octanol/water}$	:	0.70 (experimental); 0.53 (estimated)
conversion factors	:	at 20°C, 101.3 kPa: 1 ppm = 3.0 mg/m^3 1 mg/m^3 = 0.33 ppm

Data from Åke91, http://esc.syrres.com.

EDMA is a very flammable, volatile, colourless liquid, with an unpleasant fishy-ammonia-like odour. An odour threshold of 0.023 mg/m^3 (7.6 ppb) has been reported. It is a strong alkaline compound (Åke91).

4 Uses

EDMA is mainly used in foundries as a curing catalyst for resin bounding of sand cores. Furthermore, it is an intermediate in chemical industry and used as a stabiliser for chlorinated hydrocarbons (Åke91, HSE96, NLM99).

It may occur naturally in traces from protein decomposition (HSE96).

5 Biotransformation and kinetics

In humans, EDMA was readily absorbed by the respiratory tract. At exposure to concentrations of 10-50 mg/m^3 (3-17 ppm), for 8 hours, a mean uptake of 87% (range: 81-94%) was calculated from exhaled air and urinary excretion data. No difference in uptake was found between the individual volunteers (n=4) or between the exposure levels (Stå91a).

The skin absorption of EDMA vapour was examined *in vivo* by exposing the right forearm of 3 human volunteers to concentrations of 250, 500, and 1000 mg/m^3 (82.5, 165, 330 ppm), for 4 hours, in a 0.5 m^3 Plexiglas chamber. The median dermal uptake was calculated to be 44 (range: 43-47), 64 (range: 32-76), and 88 (range: 63-116) µg (probably from urine samples collected for 24 hours after the start of exposure). The median permeability coefficient (K_p) was 0.037 cm/h. Tested *in vitro*, the median steady-state flux and the permeability

coefficient were 0.017 mg/cm^2/h and 0.003 cm/h, respectively, for human skin, and 0.009 mg/cm^2/h and 0.001 cm/h, respectively, for guinea pig skin (Lun97).

Following single oral doses of 5 and 25 mg, EDMA was readily absorbed from the gastrointestinal tract. Within the first 24 hours, means of 91 (range: 86-97%) and 81% (range: 74-85%), respectively, of the administered doses were excreted in the urine (Lun95, Stå91a). When a single oral dose of 25 mg/kg bw EDMA was given simultaneously with a single dose of trimethylamine of 300 or 600 mg/kg bw, 80% of the EDMA was excreted in urine within 24 hours (Lun95)

EDMA is rapidly distributed. The plasma half-lives of EDMA and ethyldimethylamine *N*-oxide (EDMAO), its main metabolite, were 1.3 and 3.0 h, respectively. The mean volume of distribution was on average 310 L (i.e., about 5 times the body water content) (Stå91a).

Generally, tertiary aliphatic amines are mainly metabolised by oxygenation of the nucleophilic nitrogen by a flavin-containing monooxygenase (FMO; 'Ziegler's enzyme') to an amine *N*-oxide while *C*-oxidative dealkylation to a secondary amine forms a minor pathway (Åke91). As to EDMA, oxygenation to EDMAO is by far the most important pathway. Urinary excretion data from experimentally and occupationally exposed persons did not indicate that dealkylation plays a role in the metabolism of EDMA (Lun91, Stå91a). When single oral doses of EDMA and trimethylamine were simultaneously administered (see above), large interindividual differences were observed, but the urinary EDMA excretion data indicated reduced *N*-oxygenation and some dealkylation of EDMA (Lun95).

The main route of eliminating EDMA from the body is by urinary excretion. Following inhalation, 80% or more is excreted in the urine within 24 hours. Both in experimental and occupational settings, EDMAO accounted for ca. 90% of the sum of urinary excreted parent compound and EDMAO. In 2 workers (sisters), a considerably lower EDMAO fraction (18, 63%) was found probably due to an inherited decreased capacity for *N*-oxygenation of tertiary amines. Following oral administration, all is excreted as EDMAO (Lun91, Stå91). When trimethylamine was given simultaneously, the median EDMAO fraction decreased to 90-93%, but the sum of EDMA and EDMAO did not change (Lun95). In the experimental inhalation study, a two-phase urinary excretion of EDMA and EDMAO was observed with half-lives of 1.5 and 7 and 2.5 and 9 h, respectively. The mean total (plasma), renal, and metabolic (non-renal) clearances were calculated to be 170, 16, and 160 L/h, respectively (Åke91, Stå91a).

Elimination by exhalation was minimal. In the volunteer experiment, the mean concentrations in exhaled air (as percentage of the exposure level) sampled 1 hour or 2 hours post-exposure were 0.5 (range: 0.2-1.2%) and 0.2% (range: 0.1-0.4%), respectively, and independent of the exposure concentration. Absorption in the mucous membranes of the respiratory tract (and subsequent release into exhaled air) rather than elimination through the lungs may have taken place (Åke91, Stå91a).

In the experimental and occupational studies, good correlations were found between the concentration of EDMA in air and the post-shift sums of the concentrations of EDMA and EDMAO in plasma and urine. A concentration of 10 mg/m^3 (3 ppm) EDMA in occupational air was calculated to correspond with summed concentrations of EDMA and EDMAO of 5.7 µmol/L plasma or 135 mmol/mol creatinine (Åke91, Stå91a). Provided that the sum of the EDMA and EDMAO concentrations is taken, biological monitoring of EDMA is not confounded by dietary intake of trimethylamine (Lun95).

No experimental animal data on the kinetics of EDMA have been found.

6 Effects and mechanism of action

Human data

Two episodes of increased incidences of complaints of skin irritation by employees of a Belgian core-making facility were reported. There was exposure to a wide variety of compounds such as phenol, formaldehyde, ammonia, aliphatic amines, isocyanates, dimethylformamide, cresols, benzenes. No specific relation between complaints and effects could be made, but Hermans suggested that indoor climate conditions (low temperature, low humidity, high extent of static electricity) might have played a significant role (Her89).

Occupational exposure to tertiary amines in general and EDMA in particular has been associated with, especially, transient effects such as eye irritation (watery, itchy, stinging sensation, reddening) and visual disturbances (hazy/ blurry vision, halo effect). Mydriasis (widely dilated pupils with no response to light) and cyclopeglia (loss of accommodation) have been reported as well. In addition, nose and throat irritation, dizziness, nausea, headache, stomach pain, and discomfort of the chest have been complained of as well. In some studies, higher incidences or risks of bronchial construction, high blood pressure, and subjective facial pains (trigeminus-neuralgia) were found in core workers than in other foundry workers. As far as EDMA levels are reported, they are generally up to approximately 50 mg/m^3 (17 ppm) as full-shift average levels, but peak

exposures to 100 to 300 mg/m^3 (30-100 ppm) may have occurred. In addition, workers may have been exposed to other compounds such as triethylamine, chloro-substituted amines, and isocyanates as well (see Alb88, Åke91).

In assessing the relationship between ocular effects and exposure levels, 2 papers are of particular interest. In the first study, effects due to experimental and occupational exposure were examined. In the experimental setting, irritation of the mucous membranes of the eyes (in 3/4 and 3/4 volunteers, respectively), subjective visual disturbances (haze) (in 1/4 and 3/4, respectively), and slight corneal epithelial oedema (in 1/4 and 3/4, respectively) were seen in the volunteers (male; n=4) exposed to 40 or 50 mg/m^3 (13, 17 ppm), for 8 hours. No such effects were seen at exposures to 10 and 20 mg/m^3 (3, 7 ppm; in the latter group, only 2 volunteers were exposed). Further, there was an increase in corneal thickness in 2/4, 2/2, 4/4, and 4/4 volunteers exposed to 10, 20, 40, and 50 mg/m^3, respectively. Exposure to 80 and 100 mg/m^3 (26, 33 ppm), for 15 minutes, caused eye irritation (in 3/4 of both groups), but no visual disturbances or corneal oedema. In the experimental setting, 2/12 workers reported visual disturbances, starting at the last hour of the 8-hour shift and lasting one hour after the end of the shift. In one eye of one of these workers, slight corneal epithelial oedema was found at examination 40 minutes after the end of the shift. The onset of ocular effects was associated with a temporary 15-minute-lasting malfunction of the exhaust ventilation during the last hour of the shift. These 2 workers were exposed to 8-hour TWA levels of 28 and 23 mg/m^3 (approximately 8-9 ppm; estimated from 8 1-hour air samples) while during the last hour, concentrations were 107 and 125 mg/m^3 (approximately 35-40 ppm). At another occasion with proper ventilation, no visual disturbances were recorded at 8-hour TWA levels of 14 and 5 mg/m^3 (approximately 2-5 ppm) (Stå91b).

The second study was conducted to define visual effects experienced by amine-cured cold box foundry workers and to determine ambient concentrations at which these effects were occurring. Data were from 54 employees from 26 foundries. Short-term and 8-hour geometric mean exposure levels (GM) of EDMA were 10.7 (95% confidence limits: 10.2-11.2) and 6.3 (95% CL: 5.7-6.9) mg/m^3 (3.5, 2.0 ppm), respectively. Workers were divided into exposure categories ($\leq$5 ppm; >5-10 ppm; >10 ppm; both for 8-hour and short-term exposure), and exposure and effect correlations were established. As to 8-hour exposure categories, 23/26 workers of the low-exposure group (GM: 3.0 ppm or 9.0 mg/m^3) were free of effects on the eyes while 3/26 (GM: 4.4 ppm or 13 mg/m^3) were affected. All the workers of the mid- and high-exposure group (n=10; GM 8-hour: 7.7 ppm and n=18; GM: 15.5 ppm, respectively) had reported visual disturbances. In the short-term categories, none of the workers of

the low-exposure group (n=16; GM: 3.2 ppm) were affected. In the mid-exposure group, 6 workers (GM: 8.0 ppm) had experienced effects while the other 6 (GM: 8.0 ppm) had not; in the high exposure group, these figures were 25 (GM: 20.7 ppm) and 1 (GM: 16.8 ppm), respectively. According to Warren and Selchan, an exposure level of 9 mg/m^3 (3 ppm) can considered to be a no-observed-effect level. At levels between 9 and 15 mg/m^3 (3-5 ppm), there were occasional reports of slightly hazy vision after more than 6 hours of exposure, but not of halos or other discomfort. At higher levels, incidence and severity of complaints increased. At levels exceeding 45 mg/m^3 (15 ppm), pronounced visual disturbances, frequent bluish halo vision, definite eye, nose, and throat irritation, and occasional headaches were reported (War88).

In an investigation on the potential chronic health effects associated with moulding in foundry industry, an increased mortality from cancer (SMR: 152; 95% confidential interval: 100-221) was found among a cohort of 632 Danish moulders (age range at the day of the census: 15-74 years) followed through 10 years, when compared with a control cohort consisting of approximately 52,000 unexposed skilled workers (e.g., carpenters, electricians, instrument makers). This was caused mainly by an excess number of deaths from bladder cancer (no of deaths: 6 vs. 0.67 expected; SMR: 896; 95% confidential interval: 329-1,949) (Han91). However, since these workers were exposed to a number of substances such as polycyclic aromatic hydrocarbons, aromatic amines, phenols, cresols, and aldehydes, the committee could not draw conclusions concerning the potential carcinogenic effects due to occupational exposure to EDMA from this study.

Animal data

Irritation and sensitisation

The committee did not find experimental animal data on the potential eye and skin irritating and sensitising effects of EDMA.

The upper respiratory tract irritation was evaluated in mice (male Swiss OF_1) during a 15-minute oronasal exposure to increasing concentrations of EDMA. The airborne concentration resulting in a 50% decrease in the respiratory rate (RD_{50}) was 483 mg/m^3 (161 ppm). EDMA was also tested for pulmonary toxicity in mice and for the effects of a 120-minute exposure on the respiratory rates of non-anaesthetised, tracheally cannulated mice ($RD_{50}TC$). The $RD_{50}TC$ value for EDMA was found to be 2073 mg/m^3 (691 ppm). From these results, it was

concluded that EDMA is essentially an upper respiratory tract-irritating compound (Gag89).

Acute toxicity

When male rats (n=4 per group) were exposed to 240, 300, 360, or 420 mg/m^3 EDMA (80, 100, 120, 140 ppm), for 1 hour, no mortality occurred in the lowest exposure group. In the 300-mg/m^3 group, one animal died during the 10-day observation period, while exposure to 360 mg/m^3 (120 ppm) caused mortality in 3/4 animals, all dying within the 1-hour exposure period, indicating that, in this study, a 1-hour LC_{50} would be between 300 and 360 mg/m^3 (100, 120 ppm). Exposure to 420 mg/m^3 (140 ppm) was lethal to all rats within 20 minutes. Clinical signs observed during exposure included dyspnoea, tremors, ocular erythema and discharge, nasal discharge, salivation, and collapse (You70). A 1-hour LC_{50} (rat) of > 2300 < 15,400 mg/m^3 (760, 5080 ppm) was listed (no details or reference presented) (Gre98).

An oral LD_{50} (rat) of 606 mg/kg bw was presented (no details or reference presented) (Gre98).

Mutagenicity and genotoxicity

EDMA was stated to be negative in *in vitro* mutagenicity tests in *S. typhimurium* (no details or reference presented) (Gre98).

The committee did not find other data on the mutagenic or genotoxic potential of EDMA.

The committee did not find data on the effects of repeated exposure, including carcinogenicity and reproduction toxicity, of EDMA.

7 Existing guidelines

The current administrative occupational exposure limit (MAC) for EDMA in the Netherlands is 6 mg/m^3 (2 ppm), 8-hour TWA.

Existing occupational exposure limits in some European countries and the USA are summarised in the annex.

8 Assessment of health hazard

In humans, EDMA was readily and almost completely absorbed following inhalation and oral exposure. Dermal absorption of vapours was insignificant. EDMA was rapidly distributed. Following inhalation, EDMA was almost completely *N*-oxygenated to EDMAO. Plasma half-lives were 1.3 and 3.0 h for EDMA and EDMAO, respectively. The main excretion route is via the urine. Urinary excretion followed a biphasic pattern with half-lives of 1.5 and 7 h for EDMA and of 2.5 and 9.0 h for EDMAO. Occupational exposure can be monitored by measuring the sum of the post-shift concentrations of EDMA and EDMAO in plasma or urine.

Following experimental and occupational exposure, ocular effects, especially corneal effects and visual disturbances such as hazy or blurred or halo vision, irritation of nose and throat, dizziness, nausea, headache, stomach pain, chest discomfort, bronchial construction, high blood pressure, and facial pains (trigeminus-neuralgia) were reported. The study of Warren and Selchan (War88) among 54 foundry workers showed that there were no ocular effects in 16 out of 16 workers exposed to short-time geometric mean levels of approximately 10 mg/m^3 (3.2 ppm) while these effects did occur in 6/12 workers exposed to short-term levels of 24 mg/m^3 (8.0 ppm). When stratified according to 8-hour geometric mean exposure levels, 23/26 workers of the low-exposure (i.e., 5 ppm) group (GM: 3.0 ppm or 9.0 mg/m^3) were free of effects on the eyes while 3/26 (GM: 4.4 ppm or 13 mg/m^3) were affected. In the study of Ståhlbom et al. (Stå91), an increase in corneal thickness without oedema or visual disturbances was seen in 2/4 and 0/2 volunteers after 8-hour exposures to 10 and 20 mg/m^3 (3, 7 ppm), respectively. There were no data on systemic effects due to repeated occupational exposure to EDMA.

The committee did not find experimental animal data on the potential eye and skin irritating and sensitising effects of EDMA.

Based on 1-hour inhalation experiments in rats that suggest a 1-hour LC_{50} between 300 and 360 mg/m^3 (100, 120 ppm) and an oral LD_{50} (rat) of 606 mg/kg bw, EDMA should be regarded as very toxic by inhalation and harmful if swallowed.

The committee did not find data on the repeated-dose toxicity (including reproduction toxicity and carcinogenicity) of EDMA.

Since there were no data on the systemic effects following repeated exposure to EDMA, the committee concludes that the toxicological database on EDMA is

too poor to justify recommendation of a health-based occupational exposure limit.

Concerning protection against local irritating effects, the committee concludes that the present MAC-value of 6 mg/m^3 (2 ppm), as an 8-hour time-weighted average (TWA), is about right.

Skin notation

Despite the presence of skin absorption data, the lack of information on the systemic toxicity of EDMA hampers the evaluation of the need for assigning a skin notation.

References

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Lun97 Lundh T, Boman A, Åkesson B. Skin absorption of the industrial catalyst dimethylethylamine in vitro in guinea pig and human skin, and of gaseous dimethylethylamine in human volunteers. Int Arch Occup Environ Health 1997; 70: 309-13.

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TRG03 TRGS 900. Grenzwerte in der Luft am Arbeitsplatz; Technische Regeln für Gefahrstoffe. BArBl 2003; (9).

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Annex

Occupational exposure limits for ethyldimethylamine in various countries.

country - organisation	occupational exposure limit		time-weighted average	type of exposure limit	note[a]	reference[b]
	ppm	mg/m^3				
the Netherlands						
- Ministry of Social Affairs and Employment	2	6	8 h	administrative		SZW04
Germany						
- AGS	-	20	8 h			TRG03
	-	20	15 min			
- DFG MAK-Kommission	2	6.1	8 h		[d], [e]	DFG03
	4	12.2	15 min[c]			
Great Britain						
- HSE	10	30	8 h	OES		HSE02
	15	46	15 min			
Sweden	-	-				Swe00
Denmark	25	75				Arb02
USA						
- ACGIH	-	-				ACG04
- OSHA	-	-				ACG03
- NIOSH	-	-				ACG03
European Union						
- SCOEL	-	-				EC04

[a] S = skin notation; which means that skin absorption may contribute considerably to body burden; sens = substance can cause sensitisation.

[b] Reference to the most recent official publication of occupational exposure limits.

[c] Maximum number per shift: 4, with a minimum interval between peaks of 1 hour.

[d] Listed among substances with MAK values but no pregnancy risk group classification.

[e] It was noted that reaction with nitrosating agents can result in the formation of carcinogenic *N*-nitrosodimethylamine and *N*-nitrosomethylethylamine.