Ammonium chloride (fume)

(CAS No: 12125-02-9)

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 ammonium chloride fume 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 MA Maclaine Pont, M.Sc. (Wageningen University and Research Centre, Wageningen, the Netherlands).

In February 1998, literature was searched in the databases Medline, Toxline, and Chemical Abstracts covering the periods 1966 until February 1998, 1981 until October 1997, and 1937 until December 1997, respectively, and using the following key words: ammonium chloride (and isotopic compounds), NH_4Cl, and 12125-02-9.

In December 1998, the President of the Health Council released a draft of the document for public review. Comments were received by the following individuals and organisations: A Aalto (Ministry of Social Affairs and Health, Tampere, Finland) and P Wardenbach, Ph.D. (Bundesanstalt für Arbeitsschutz and Arbeitsmedizin, Dortmund, Germany). These comments were taken into account in deciding on the final version of the document.

An additional literature search in Toxline and Medline in September 2003 did not result in information changing the committee's conclusions.

2 Identity

name	:	ammonium chloride
synonyms	:	Amchlor; ammonium muriate; Darammon; Salammoniac; salmiac
molecular formula	:	NH_4Cl
CAS number	:	12125-02-9

3 Physical and chemical properties

molecular weight	:	53.49
boiling point	:	520°C (triple point)
melting point	:	338°C (sublimation point)
flash point	:	not available
vapour pressure	:	at 160°C: 0.13 Pa
solubility in water	:	soluble (at 15°C: 26 g/100 mL)
log $P_{octanol/water}$	:	-4.37 (estimated)
conversion factors	:	not applicable

Data from Lid96, http://esc.syrres.com.

Ammonium chloride consists of colourless, odourless crystals or of crystallic masses, or it is a white, granular powder. The crystals are somewhat hygroscopic. It has a cooling, saline taste (ACG91).

4 Uses

Ammonium chloride is used in the manufacture of various ammonia compounds and dry batteries, as a mordant in dyeing and printing, as a soldering flux, fertiliser, and pickling agent in zinc coating and tinning, and in electroplating, washing powders, snow treatment, resins and adhesives of urea-formaldehyde, medicine, and the food industry. Large amounts of ammonium chloride fume are frequently evolved during galvanising operations (ACG91).

In galvanising plants, the aerosols consist not only of ammonium chloride, but also of zinc oxide, zinc chloride and complexes of zinc chlorides, and traces of zinc hydroxide (Duf88).

In Europe, ammonium chloride used to be coded as food additive E510, with an unlimited daily intake, but nowadays it is not any longer on the Dutch list of permitted food additives as published in the Food and Drugs Act. In the USA, ammonium chloride received the GRAS (generally recognised as safe) status in 1983 (FDA83), and was still listed among these substances in 2003 (see 21CFR184.1138).

5 Biotransformation and kinetics

Single oral doses of 1-40 mg/kg bw had no effect on the ammonia (NH_3) blood level of normal subjects, but caused hyperammonaemia in 32 patients suffering from liver cirrhosis (Rud73).

6 Effects and mechanism of action

Human data

Until 1977, no cases of intoxication have been reported after occupational exposure to ammonium chloride fume (Hou77).

Two cases of occupational asthma are described due to soft corrosive soldering fluxes containing zinc chloride and ammonium chloride. A challenge with ammonium chloride caused a fall in FEV_1* in one subject but the magnitude

* FEV_1: forced expiratory volume in 1 second.

of the response was less than with the whole flux, suggesting that it may not be the sole active agent. A challenge with zinc chloride had no effect on this subject. In the second case, the authors were unable to challenge with the individual constituents of the flux. It is, therefore, not possible to ascribe the effects to a particular constituent in the flux (Wei89).

The oral intake of 12 g ammonium chloride/day (resulting in 0.13-0.21 g/kg bw/day) for 12 days had no effect on several ventilatory parameters during submaximal or maximal exercise of healthy volunteers. Heart rates and performance time were similarly unaltered (Bul83).

Ingestion of ammonium chloride produces metabolic acidosis and diuresis, and ammonium chloride is administered for these effects (WHO86)

Animal data

Referring to the same source, instillation of 500 mg of ammonium chloride into the eyes of rabbits was reported to be mildly (NIO03) or severely irritating (Lew92); instillation of 100 mg was listed as severely irritating (NIO03).

Oral LD_{50} values of 1650 and 1300 mg/kg bw have been reported for rats and mice, respectively (NIO03). Following intraperitoneal or intravenous injections into mice, LD_{50} values were 485 (NIO03) and 367 mg/kg bw (War58), respectively. Intravenous injections of doses as low as 78 and 220 mg/kg bw were lethal to rabbits and guinea pigs, respectively (NIO03).

A 2-hour exposure of rabbits to a mist of a 30% solution of ammonium chloride caused a slight degree of catarrhic pneumonia and infiltration of small round cells in the lungs, a slight degree of vacuolar degeneration in the acinous and infiltration of small round cells in Glisson's capsule in the liver, exfoliation of epithelium of the uriniferous tubules and infiltration of small round cells in the kidney, and a slight degree of atrophy of lymphatic follicles in the spleen (Yas59).

Ex vivo, ammonium chloride increased mucus transport velocity and ciliary beat frequency in the tracheobronchial tree isolated from Wistar rats. The effective concentrations were in the range of 10^{-7}-10^{-4} g/mL. The effects were more severe in the trachea isolated from rats with bronchitis than from normal rats (Mel80).

The additional amount of ammonia (i.e., above the amount normally produced in the body) that can be safely ingested and assimilated is difficult to assess. In short-term (28-90 days) studies carried out in rats and pigs, no adverse effects were reported at higher levels of ammonia intake (75-545 mg NH_3/kg bw/

day) in the form of sulphamate, phosphate, citrate, or chloride (calculated for ammonium chloride this would be 236-1712 mg/kg bw). The effects attributed directly to elevated ammonium ion levels are acute pulmonary oedema and central nervous system (CNS) toxicity, depression of appetite due to a direct effect of the ammonium ion on the brain, and promotion of growth via the use of ammonium salts as a source of nitrogen under certain circumstances. Some effects (such as renal growth and demineralisation of bone) arising from the administration of ammonium chloride seem to be secondary effects of acidosis (WHO86).

The committee found a number of studies concerning the effects of changes in urinary parameters on proliferative responses of the bladder epithelium of male rats induced by certain compounds. In these experiments, the effect of urine acidification by concomitant administration of ammonium chloride was investigated, and 'control' groups receiving a single dose of ammonium chloride only were included.

De Groot et al. investigated the difference in response to monosodium glutamate concerning bladder epithelial hyperplasia in rats with the feeding of different basal diets. Groups of male rats (Cpb:WU; Wistar random; n=10/group) were fed a cereal-based stock diet or a 'purified' diet containing acid casein supplemented with methionine as the only protein source, with and without monosodium glutamate and with and without acidifying and alkalising supplements. One of the groups received 'purified' diet containing - the acidifying salt - ammonium chloride at concentrations that were gradually increased from 1% at week 1 to 5 % at week 6 that was subsequently maintained for 7 weeks. [Based on data on mean body weight and food intake presented by De Groot et al., the committee estimated that the ammonium chloride levels could range from roughly 1500 mg/kg bw/day during week 1 to roughly 3,000 mg/kg bw/day during week 8-13]. One animal died at week 10, showing haemorrhagic fluid in the abdominal cavity, swollen ureters, a blood clot in the enlarged urinary bladder, inflammation of the seminal vesicles and prostate, and haemothorax. No abnormalities in condition or behaviour were observed in the other rats. Body weights of ammonium chloride-treated rats were statistically significantly lower at weeks 4, 8, and 12, when compared to controls. Some decrease in food intake was noticed in the first 8 weeks. Urinary pH of the animals receiving the ammonium chloride-supplemented and the unsupplemented 'purified' diet ranged from ca. 5.5-5.9 and from ca. 5.6-6.2, respectively, during the 13-week treatment period, compared to ca. 6.7-7.7 in animals receiving the unsupplemented stock diet. At sacrifice of the surviving treated animals, there was a marked, statistically significant increase in relative

kidney weights while those of the liver were comparable to controls. Microscopic examination included the urinary bladder, ureters, kidneys, liver, testes, thyroid with parathyroids, adrenals, and bone. In the ammonium chloride-treated rats, only changes in the bladder epithelium were found. Moderate diffuse epithelial hyperplasia and severe diffuse hyperplasia with small papillary protrusions were seen in 2/10 and 2/10 rats, respectively, while there were focal hyperplasia and very small intra-epithelial cysts in 1/10 and 1/10 rats, respectively. Although these changes were not seen in any of the control rats, the differences in incidence between the 2 groups were not statistically significant. The induction of bladder epithelial hyperplasia by dietary ammonium chloride was confirmed in a supplementary study of similar design, lasting 15 instead of 13 weeks, in which 5/9 rats showed moderate diffuse hyperplasia vs. none in 10 controls ($p<0.05$; Fisher's pairwise test). In addition, severe diffuse hyperplasia with small papillary protrusions was seen in one ammonium chloride-treated rat (Gro88).

Shibata et al. examined changes of urinary electrolyte levels and pH, and DNA synthesis and the morphology of bladder epithelium in groups of F344 rats feeding diets containing various carbonates or ammonium chloride with and without L-ascorbic acid, for 4 or 8 weeks. One of the groups comprised 11 rats receiving 1% ammonium chloride (780 mg/kg bw/day, based on an average body weight over this 8 weeks of 200 mg and a food consumption of 15.6 g/rat/day); data from Shi89) for 8 weeks. Body weights of ammonium chloride-treated rats were statistically significantly decreased at week 4 and 8 when compared to untreated control animals. Apart from a decrease in pH (5.9 vs. 6.9 in controls) and in potassium levels, urinary parameters (volume; osmolality; ascorbic acid, sodium, chloride, calcium, phosphate, and magnesium levels; crystals) were not affected. The degree of DNA synthesis, estimated by determining the number of cells incorporating bromodeoxyuridine (BrdU) into the DNA per 1000 cells (BrdU-labelling index), was comparable among ammonium chloride-treated and untreated rats. No morphological changes were observed in bladder epithelium at light microscopy and scanning electron microscopy examinations (Shi89).

In a study to investigate the effects of tributyl phosphate on the urine and bladder epithelium in male Sprague-Dawley rats and the effect of urine acidification by concomitant administration of ammonium chloride, a 'control' group of 10 rats received daily dietary doses of ammonium chloride of 1.23% (roughly 980 mg/kg bw/day based on a mean food intake of 80 g/kg bw/day; adapted from Arn97), for 10 weeks. Treatment did not affect body weight throughout the study, and no abnormalities were observed during weekly clinical evaluations. Urinalyses at the end of the experiment (week 11) showed decreased

pH (6.04 vs. 7.56 in controls; $p<0.05$), increased calcium, which is frequently seen with acidic urine (11.0 vs. 5.9 mg/dL; $p<0.05$), and decreased creatinine levels (52 vs. 80 mg/dL; $p<0.05$); there was no crystaluria. Relative and absolute bladder and kidney weights of ammonium chloride-treated animals did not differ from those of untreated controls. There was no difference in BrdU-labelling index between ammonium chloride-treated and untreated control rats. Examination by light microscopy did not reveal urothelial changes in the bladders of animals of the ammonium chloride group, but focal, small areas of superficial cell necrosis, proliferation, and prominent vascularity were seen in 2/10 animals using scanning electron microscopy. Microscopic examination of the kidneys and the stomach did not reveal treatment-related changes. No other data were presented (Arn97).

Carcinogenicity

Early literature already indicated that ammonium chloride could inhibit tumour growth in mice inoculated with the Twort carcinoma, when given via the drinking water (Tho43).

Mutagenicity and genotoxicity

In vitro, ammonium chloride was negative in a bacterial mutation assay using *S. typhimurium* strains TA92, TA94, TA98, TA100, TA1535, and TA1537 when tested with and without rat liver metabolic activation at concentrations up to 10 mg/plate (Ish84, Ish88).

Tested in mammalian cell systems, i.e., for polyploidy and chromosomal aberrations in Chinese hamster fibroblasts (tested without metabolic activation only), ammonium chloride caused an increase in the incidence of cells with structural chromosomal aberrations (including gaps), but not of polyploid cells (Ish84, Ish88).

In vivo, ammonium chloride did not induce an increase in the incidence of micronucleated polychromatic erythrocytes obtained from the bone marrow of male ddY mice, 24 hours after one intraperitoneal injection of doses of 62.5-500 mg/kg bw or 4 intraperitoneal injections of doses of 31.3-250 mg/kg bw (Hay88).

Reproduction toxicity

Khera investigated the possible relationships between maternal acid-base-electrolyte imbalance, histological changes in the maternal/extra-embryonic tissues, and fetal anomalies induced by maternally toxic doses of ethylene glycol, sodium salicylate, and cadmium chloride. As a part of this study, the influence of concomitant administration of ammonium chloride on the effects of sodium salicylate was examined using a control group of 13 pregnant Sprague-Dawley rats given amounts of ammonium chloride of ca. 750 mg/kg bw/day via the drinking water on gestational days 7-10*. There was a (not statistically significant) increase in the incidence of wavy and supernumerary ribs of 6 and 2%, respectively, vs. 1 and 1%, respectively, in control animals receiving pure drinking water. It is unlikely that these effects can be ascribed to the altered acid-base homeostasis in the mother. This was shown in a separate study, where the dams were given 421 mg ammonium chloride/kg bw via the drinking water from gestational day 7 onwards. After 7 hours, 4 of the 17 parameters measured in blood and urine were decreased: P_{CO2}, HCO_3^- concentration, phosphate concentration, and a parameter 'BE ec F' (in mEq/l; not explained in the text or legenda). After 48 hours, the urinary pH was decreased ($p<0.01$), but all the other parameters were within the normal range (Khe91).

7 Existing guidelines

The current administrative occupational exposure limit (MAC) for ammonium chloride (fume) in the Netherlands is 10 mg/m^3, 8-hour TWA.

Existing occupational exposure limits for ammonium chloride (fume) in some European countries and in the USA are summarised in the annex.

* The quantity of ammonium chloride taken up in those 4 days was calculated as follows: 4 (number of days) x 41.7 mL (mean water consumption/day during these days) x 4.73 mg/mL (concentration of ammonium chloride solution administered) = 789 mg. The body weight of the animals on gestational day 11 was estimated to be 265 g (body weights at the start of the experiment ranged from 225 - 275 g; the weight gain from gestational days 8-11 was 15 g - comparable with the control group). Thus, the total intake of ammonium chloride was approximately 3000 mg/kg bw or 750 mg/kg bw/day.

8 Assessment of health hazard

Although the critical effect of occupational exposure to ammonium chloride is likely to be upper respiratory tract irritation, the committee did not find human data from which a concentration-effect relation for inhalation exposure can be estimated.

The committee did not find data from inhalation studies in experimental animals.

Ammonium chloride was mildly or severely irritating to the eyes of rabbits. The committee did not find data from studies on skin irritation or sensitisation.

Oral LD_{50} values in rats and mice were 1650 and 1300 mg/kg bw, respectively.

Apart from a number of studies concerning the effects of changes in urinary parameters on proliferative responses of the bladder epithelium of male rats induced by certain compounds, the committee did not find data on the toxicity of ammonium chloride following repeated administration. In these experiments, the effect of urine acidification by concomitant administration of ammonium chloride was investigated, and 'control' groups receiving a single high dose of ammonium chloride only, for 10-15 weeks, were included. At an estimated amount of 780 mg/kg bw for 8 weeks, ammonium chloride caused decreased body weights but no morphological changes in bladder epithelium. An estimated dose of 980 mg/kg bw, for 10 weeks, did cause hyperplasia in bladder epithelium in 2/10 rats, but did not affect body weights or relative and absolute bladder and kidney weights. At doses estimated to range from 1500 mg/kg bw at week 1 to 3000 mg/kg bw from week 6 to week 13 or 15, decreased body weights and increased relative kidney weights were observed as well as bladder epithelial hyperplasia in approximately half of the animals.

In a reproduction toxicity study, in which the influence of administration of ammonium chloride on the effects of sodium salicylate was investigated in Sprague-Dawley rats, administration in the drinking water of doses of ammonium chloride of ca. 750 mg/kg bw/day on gestational days 7-10 did not cause significant, treatment-related changes in the number of live fetuses per litter, the number of resorptions, fetal body weight, or the incidence of fetal malformations.

Ammonium chloride was negative in an *in vitro* bacterial mutation assay and in an *in vivo* mouse bone marrow micronucleus test (route: intraperitoneal). In an *in vitro* chromosomal aberration assay, it caused an increase in the number of human fibroblast cells with structural aberrations (including gaps).

The committee considers the toxicological database on ammonium chloride (fume) too poor to justify recommendation of a health-based occupational exposure limit.

At exposure to 10 mg/m^3 ammonium chloride (fume), the current MAC-value, systemic effects are unlikely, but local effects on eyes and respiratory tract may occur. Since data on irritation are lacking, the committee concludes that there is insufficient information to comment on the level of the present MAC-value.

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Coh95 Cohen SM, Garland EM, Cano M, et al. Effects of sodium ascorbate, sodium saccharin and ammonium chloride on the male rat urinary bladder. Carcinogenesis 1995; 16: 2743-50.

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Swe00 Swedish National Board of Occupational Safety and Health. Occupational exposure limit values and measures against air contaminants. Solna, Sweden: National Board of Occupational Safety and Health, 2000; Ordinance AFS 2000:3.

SZW03 Ministerie van Sociale Zaken en Werkgelegenheid (SZW). Nationale MAC-lijst 2003. The Hague, the Netherlands: Sdu, Servicecentrum Uitgevers, 2003: 16.

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Annex

Occupational exposure limits for ammonium chloride (fume) 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	-	10	8 h	administrative		SZW03
Germany						
- AGS	-	-				TRG00
- DFG MAK-Kommission	-	-				DFG03
Great-Britain						
- HSE	-	10	8 h	OES		HSE02
	-	20	15 min	STEL		
Sweden	-	-				Swe00
Denmark	-	10	8 h			Arb02
USA						
- ACGIH	-	10	8 h	TLV		ACG03b
	-	20	15 min	STEL		
- OSHA	-	-				ACG03a
- NIOSH	-	10	10 h	REL		ACG03a
		20	15 min	STEL		
European Union						
- SCOEL	-	-				EC03

[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.