被曝はことによると遅延性或は深く潜在する痛みを伴う厳しい皮膚熱傷を引き起こすかもしれない。この化学剤が皮膚に局在すると、その影響はより厳しいかもしれない。1回だけの長時間皮膚被曝は有害量を吸収した素地を結果として生じさせるかもしれない。彼とツァング[26] はアリルスルホン酸ソーダを製造している 2 棟の工場において職業上被曝した被験者に対するアリルクロリドの効果を研究した。1 棟の工場の 26 人の被験者は 2.5 ヶ月から 6 年の間 2.6 ~ 6650 mg/m^3 (0.83 - 2127 ppm)のレベルでアリルクロリドに曝された；別の工場では、27 人の被験者が 1 から 4.5 年の間 0.2 ~ 25.13mg/m^3 (0.06-8.03 ppm)で曝露された。可能な皮膚の被曝についての情報は提供されなかった。最初の工場のほとんどの作業者は、四肢に、衰弱、感覚異常、およびしびれを持っていた。試験された 19 人の被験者の 10 人において神経筋電図記録は神経性の異常を示した；従って、神経障害の有病率は 52.6%であった。2 番目の工場の作業員の同様な症状はずっと穏やかであった；ほとんどの神経学的異常の徴候がなかったけれども穏やかな神経障害を示す神経筋電図記録が 27 人の被験者の中の 13 人において認められた。得られた証拠は長期的アリルクロリド被曝が有毒な多発性神経炎を引き起こすことを示した。

TLV 勧告

1959 年のトルケルソンら[12] の報告に基づいて、肝損傷と潜在的腎臓効果を防止するために 1ppm の TLV-TWA が勧告されている。さらなる肝臓保護と潜在的な眼と呼吸の刺激に対して保護するために 2ppm の TLV-STEL も勧告されている。皮膚記号も勧告されているが、但しそれが根拠とする産業リポートは入手不可能であった。噴門洞の腫瘍性損傷をもたらした 1978 のマウスへの NCI の強制投与研究[14] 及びファン デューレンら[15] によるマウス皮膚腫瘍開始研究の陽性に基づいて、発癌性記号の A3、即ち、動物に対する発癌性は確認されたが人に関しては不明、が保証されている。
ACGIH は皮膚記号の必要性を評価するのに役立つかもしれない追加の定量的データを求めている。SEN 記号を勧告するのに十分なデータは入手できなかった。

TLV の歴史

1957-1962：TLV-TWA、5 ppm
1963-現在：TLV-TWA、 1 ppm
1976-現在：TLV-STEL、2 ppm
1995：提案：A3、動物に対する発癌性は確認されたが、人に対しては不明
1996-現在：TLV-TWA、 1 ppm；TLV-STEL、2 ppm；A3

資料出所　ＡＣＧＩＨ提案理由書（2004）

ALLYL CHLORIDE

CAS number: 107-05-1

Synonyms: Chlorallylene; 1-Chloro-2-propene; α-Chloropropylene; 3-Chloropropylene

Molecular formula: CH_2CHCH_2Cl

Structural formula: $H_2C{=}CH{-}CH_2Cl$

Skin

TLV–TWA, 1 ppm (3 mg/m^3)

TLV–STEL, 2 ppm (6 mg/m^3)

A3 — Confirmed Animal Carcinogen with Unknown Relevance to Humans

Summary

A TLV–TWA of 1 ppm (3 mg/m^3) and a TLV–STEL of 2 ppm (6 mg/m^3) are recommended for occupational exposure to allyl chloride. These limits are intended to minimize the potential for hepatic and possible renal effects identified in animal inhalation studies at exposure concentrations of 3 to 8 ppm. The TLV–STEL is also intended to minimize the potential for respiratory and eye irritation. A Skin notation is recommended, based on data from industry reports of workers and dermal exposure. Oncogenic studies indicate that allyl chloride is carcinogenic in mice, based on neoplastic lesions of the forestomach following 78 weeks of administration by gavage, and it also acts as a skin tumor initiator in mice. Accordingly, an A3, Confirmed Animal Carcinogen with Unknown Relevance to Humans, notation is assigned. Sufficient data were not available to recommend a SEN notation.

Chemical and Physical Properties

Allyl chloride exists as a colorless, yellow, or purple chlorinated liquid, which is highly reactive and flammable. Its odor is irritating, unpleasant, and pungent, similar to that of garlic.[1] Chemical and physical properties include:[1–4]

Molecular weight: 76.5
Specific gravity: 0.94 at 20°C (water = 1.0)
Freezing point: –134.5°C
Boiling point: 44.6°C (760 torr)
Vapor pressure: 295 torr at 20°C
Vapor density: 2.6 (air = 1)
Flash point: –31.7°C (Tag closed cup)
Explosive limits: lower, 3.3%; upper, 11.1% by volume
Solubility in water: 0.36 g/100 g water at 20°C
Decomposition products: hydrogen chloride, phosgene, and carbon monoxide
Odor threshold: 0.47 ppm in air
Conversion factors at 25°C and 760 torr:
1 ppm = 3.15 mg/m^3; 1 mg/m^3 = 0.318 ppm

Major Uses

Allyl chloride has been utilized primarily as an intermediate in the manufacture of epichlorohydrin and glycerol. It also has been used in the synthesis of allyl compounds.[1,5]

Animal Studies

Acute

Grant[6] reported allyl chloride was damaging to the skin, but application of a drop to a rabbit's eye caused only mild transient injury due to the low boiling point and rapid evaporation of the chemical. The vapor itself caused irritation of the eyes and respiratory passages. A concentration of 20 to 50 mg/L of air of allyl chloride (6397 to 15,993 ppm) caused irritation of the eyes and nose in guinea pigs and albino rats, and slight irritation occurred at 10 mg/L (3198 ppm).[6]

Quast et al.[7] conducted acute (6-hour) inhalation exposure studies with CDF Fischer 344 rats and B6C3F1 mice. Single 6-hour exposure concentrations were 200, 300, 500, 800, 1000, 1200, or 2000 ppm for rats and 500, 800, 1000, 1200, or 2000 ppm for mice. Groups of ten animals/sex/species were compared to a control group of equal size. Parameters monitored included in-life observations, mortality, body weight, and clinical chemistry. A gross necropsy was performed and selected tissues were examined by light microscopy to further define the effects of allyl chloride on target organs. The kidneys were determined to be the most sensitive. No exposure-related effects were detected in the 200-ppm group of rats. Minimal kidney effects, detected only

histologically, were noted in the 300-ppm group of rats and in the 500-ppm group of mice. Microscopic changes and other parameters indicative of kidney damage became progressively more severe in both species with increasing levels of exposure. Mortality was not observed in either species at exposure concentrations below 1000 ppm but was reported above 1000 ppm, with mice more susceptible than rats. Sex differences in mortality and kidney toxicity were noted with female rats and male mice being more affected than male rats or female mice.

Boqin et al.[8] determined the toxicity of allyl chloride in several species of animals. The oral LD_{50} levels in the mouse and rat were 425 mg/kg and 460 mg/kg, respectively. The 2-hour LC_{50} levels in the mouse, rat, guinea pig, rabbit, and cat were 11.5, 11.0, 5.8, 22.5, and 10.5 mg/L, respectively. In the inhalation studies, the target organs were the liver, kidneys, nervous system, and the lungs.

Subchronic

Boqin et al.[8] performed inhalation studies with rabbits and cats 6 hours/day, 6 days/week for 3 months at an allyl chloride concentration of 66 ppm (206 mg/m^3) and with rabbits and rats 6 hours/day, 6 days/week for 5 months at 6 ppm (18 mg/m^3). At the high exposure concentration, muscle weakness of the extremities, lurching motion, and unsteady gait were only seen in one rabbit. In the second month of exposure, the other rabbits also exhibited these toxic signs. At a later stage, the extremities of the first rabbit developed flaccid paralysis, followed by muscle atrophy and emaciation. Two other rabbits also developed paralysis. The cats, however, only showed muscle weakness and unsteady gait at the end of exposure. At the low exposure level using rabbits and rats, there was no evidence of ill effect.

Quast et al.[9] conducted 4-day probe and 90-day subchronic allyl chloride inhalation studies in male and female Fischer 344 rats and B6C3F1 mice. In the 4-day probe study, 10 animals/sex/species per control and a 250-ppm exposure group were used to derive data for establishing exposure levels used in the subsequent 90-day subchronic study. In the 90-day subchronic study, group sizes of 25 animals/sex/species/group were exposed for 6 hours/day, 5 days/week at 0, 50, 100, or 250 ppm. An interim necropsy was performed on 10 animals/sex/species from each group after approximately 30 days on test, while the remaining animals in each group were necropsied at the end of the study. Parameters evaluated included in-life observations, body weight, hematology, clinical chemistry, urinalysis, organ weights, gross necropsy, and light microscopic examination of tissues.

A number of the various parameters measured during the study showed statistically significant changes in the 50, 100, or 250 ppm exposed groups. These were generally considered to represent normal biological variability or physiological adaptation as a result of allyl chloride exposure. However, an observation was made in the kidneys of the 250-ppm exposed rats, which was considered toxicologically significant. There were light microscopic changes in the kidneys of this group, which were characterized by increased cytoplasmic granularity, and eosinophilic staining of the cortical epithelial cells. In addition, an increase in the incidence and degree of focal tubular collapse and atrophy was noted when compared to their controls. These changes were minimal in degree but were consistent with kidney effects noted in rats following single 6-hour exposures at substantially higher levels. No adverse exposure-related effects were noted in mice at any exposure concentration.

Quast et al.[10] also conducted a subchronic allyl chloride toxicity study with CDF-Fischer 344 rats and B6C3F1 mice at exposure concentrations of 0, 1, 3, 10, or 20 ppm conducted 6 hours/day, 5 days/week except for holidays for up to 3 months, with an interim kill after 1 month. Clinical observation, body weights, hematology, urinalysis, clinical chemistry, organ weights, gross pathology, and histopathologic examination of the tissues were performed. No changes attributable to exposure to allyl chloride were documented at any concentration.

O'Donoghue[11] reviewed the experimental neurotoxicity of rabbits given allyl chloride by inhalation or subcutaneous injection and of mice given allyl chloride orally. Peripheral neuropathy and axonal damage of the "dying back" type, with proliferation of neurofilaments, were exhibited. The most severe degeneration occurred in the distal regions of the peripheral nerves. Degeneration was also present in the spinal cord following the pattern of a central–peripheral distal axonopathy.

Torkelson et al.[12] reported on the results of animal inhalation studies to evaluate the hazard associated with repeated, daily low-level exposures to allyl chloride.

A pilot experiment was conducted in which small groups of animals were exposed repeatedly at an average concentration of 8 ppm. A total of 28 seven-hour exposures were made during a 35-day period. Matched groups consisted of 5 rats of each sex, 4 male guinea pigs, and 1 female rabbit. Male and female rats, male guinea pigs, and the female rabbit showed no evidence of ill effects when judged by growth, behavior, mortality, gross appearance, and average final body weight. Female rats showed only a variation from controls in the average spleen weight. There were no supporting histopathological findings in the spleen. Histopathological examination did show, however, significant evidence of ill effects of a severe degree in the liver and kidneys of essentially all animals. These were characterized by dilation of the sinusoids, cloudy swelling, and focal necrosis in the liver and by changes in the glomeruli, necrosis of the epithelium of the convoluted tubules, and proliferation of the interstitial tissues in the

kidneys.[12]

Based on the pilot experiment, a larger scale experiment was performed using three groups of animals: allyl chloride-exposed; room air, inhalation chamber controls; and animal quarters controls. Each group consisted of 24 rats, 3 rabbits, 9 guinea pigs, and 1 dog of each sex. The first group was exposed 7 hours/day, 5 days/week at 3 ppm allyl chloride for a total of 127 to 134 exposures in 180 to 194 days.[12]

Animals showed no evidence of ill effects when judged, based on growth, behavior, mortality, gross appearance, and final body weight. The only deviations of significance in organ weights or hematological values were for the male dog, but these were not considered to be exposure related. The only finding of importance seen during the histopathological examination was a slight change in the livers of the female rats that consisted of slight lobular degeneration of a type often seen in control animals, but since it was not seen in the male rats nor in any other species and was not present in any of the rats allowed to recover for two months, it was considered to be related to exposure. Torkelson et al.[12] concluded that, based on these experiments, an exposure concentration of 3 ppm offered no margin of safety, and exposures should be limited to less than 3 ppm if the probability of injury was to be minimized. Upon this basis, an occupational hygiene exposure limit of 2 ppm was suggested for allyl chloride as a concentration below which practically all fluctuations should fall during a working day where repeated, prolonged exposure was likely. Because 2 ppm appears to offer a very small margin of safety, a TWA of daily exposures not to exceed 1 ppm was proposed. Under such conditions, the probability of injury would seem remote.

He et al.[13] investigated the pathology of allyl chloride neurotoxicity in mice. The study described the neuropathy which developed in mice given oral doses of 300 or 500 mg/kg three times weekly for periods of 2 to 17 weeks. Functional disability was observed in some animals. Apart from evidence of local kidney damage in 70% of the dosed mice, pathological changes were restricted to the nervous system. Nerve fiber degeneration was found in many peripheral nerves and roots, tending to be more marked distally and to reflect more motor than sensory nerves. Degenerated fibers were also found in dorsal, ventral, and lateral columns of the spinal cord. Males were more severely affected than females. Increased numbers of filaments were an early axonal change, occurring multifocally and apparently preceding axonal degeneration. No neuronal death was observed, but occasional anterior horn and dorsal root ganglion cells showed some morphological changes. Vacuolated lesions, mainly due to swelling of astrocytes and their processes, were found in the ventral horn in the cervical and lumbar regions of the spinal cord. Animals appeared to become tolerant to allyl chloride after continuous dosing. The authors indicated that this neuropathy appeared to be a central–peripheral distal type of axonopathy.

Chronic/Carcinogenicity

A bioassay for possible carcinogenicity of technical-grade allyl chloride was conducted under the Carcinogenesis Testing Program of the U.S. National Cancer Institute (NCI) using Osborne–Mendel rats and B6C3F1 mice.[14] Allyl chloride in corn oil was administered by gavage to two groups of each species for 5 days/week for 78 weeks. The TWA dosages were, respectively, 77 and 57 mg/kg/day for high- and low-dose male rats; 73 and 55 mg/kg/day for high- and low-dose female rats; 199 and 172 mg/kg/day for high- and low-dose male mice; and 258 and 129 mg/kg/day for high- and low-dose female mice. For each species, 20 animals of each sex were placed on test as vehicle controls. Twenty animals of each sex were placed on test as untreated controls for each species. Survival of high-dose male mice and high-dose rats of both sexes was extremely poor. Of the high-dose male mice, 50% were dead by week 27. Among the high-dose rats, 50% of the males had died by week 14 and 50% of the females had died by week 38. Because of early mortality in these groups, the number of animals surviving long enough to be at risk from late-developing tumors was not adequate for meaningful statistical analysis. Under the conditions of this bioassay, no convincing evidence was presented for the carcinogenicity of allyl chloride in Osborne–Mendel rats of either sex. The results do indicate that allyl chloride is carcinogenic in male and female B6C3F1 mice because, when administered by gavage, the compound is associated with neoplastic and non-neoplastic lesions of the forestomach. Van Duuren et al.[15] tested allyl chloride for carcinogenicity by chronic administration in Ha:ICR Swiss mice. Allyl chloride was active as a skin tumor initiator in the two-stage carcinogenesis assays; phorbol myristate acetate was used as a promoter.

The International Agency for Research on Cancer[16] evaluated the carcinogenic risk posed by allyl chloride. No case report or epidemiological study relating to the carcinogenicity of allyl chloride to humans was available to the Working Group. The Working Group concluded that there was inadequate evidence for the carcinogenicity of allyl chloride in experimental animals.

Reproductive/Developmental

John et al.[17] conducted a teratological evaluation in which pregnant Sprague–Dawley rats and New Zealand white rabbits were exposed to vapors of allyl chloride at concentrations of 0, 30, or 300 ppm. Exposures were for 7 hours/day on days 6 through 15 (rats) or 6 through 18 (rabbits) of gestation. Maternal toxicity occurred in both rats and

rabbits treated at 300 ppm. These consisted of depressed weight gain during gestation and increases in liver weight (both species) and kidney weights (rats only). Fetuses from rats exposed at 30 ppm had a slight delay in skeletal development, but there were no other signs of embryo toxicity. Allyl chloride was not considered teratogenic or embryo-lethal in rats or rabbits following inhalation exposure at concentrations that induced effects in the maternal animals.

Hardin et al.[18] tested allyl chloride for teratogenic potential. The study used intraperitoneal treatments (80 mg/kg of allyl chloride suspended in corn oil) of rats on days 1 to 15 of gestation. Maternal heart, liver, spleen, and kidney weights were significantly increased ($p < 0.05$), but there were no treatment-related histopathological changes. Fetal toxicity was reflected in a significant increase ($p < 0.05$) in resorptions in treated animals. No visceral or skeletal malformations were seen, but there was a significant incidence of fetuses from treated litters with edema ($p < 0.01$) and short snout with protruding tongue ($p < 0.05$). These defects were not seen in any of the control litters. Based on these studies, allyl chloride is not considered to constitute a significant developmental hazard at levels below that which would result in maternal toxicity.

In a second study by Hardin et al.,[19] allyl chloride was administered to female CD-1 mice by gavage (5 ml/kg) in corn oil at a dose stated to be the LD_{10}. This predicted LD_{10} (value not stated by the authors) was determined in nonpregnant mice, employing five doses and ten mice per day, for 8 consecutive days. Dosing in the reproductive phase occurred once daily on gestation days 6 to 13 (vaginal plug = day 0) with a dosing volume determined, based on body weight on the first treatment day (day 6). Mortality was higher than the predicted 10% among the pregnant mice, 21/28 pregnant mice died versus 4/22 nonpregnant mice, suggesting an increased sensitivity in pregnant animals. The administered dose did not significantly affect body weight gain in maternal survivors (7 pregnant, 18 nonpregnant) nor the number of live born per litter; birth weight and weight gain of neonates from five of seven viable litters were also not affected. Survival of neonates, however, was 80% from treated mice, compared with 98% in controls.

Genotoxicity Studies

Dean et al.[20] tested allyl chloride for genotoxic activity in bacterial mutation assays, in *Saccharomyces cerevisiae* JD1 for mitotic gene conversion and in a cultured rat-liver cell line for structural chromosome damage. Because of the volatility of allyl chloride, conventional plate-incorporated assays failed to demonstrate mutagenic activity. Even after a 20-minute pre-incubation of bacteria with allyl chloride in sealed containers, no mutagenic activity was observed. However, a series of spot tests with undiluted allyl chloride was successful in demonstrating the mutagenicity of the compound to both strains of *Escherichia coli*. No quantitative data were produced in bacteria. Positive results were obtained in tests for gene conversion in yeast, and the incorporation of S9 microsomal fraction did not significantly influence the conversion frequency. Exposure of sealed monolayer cultures of RL1-cells to concentrations of allyl chloride up to 25 µg/ml did not result in significant compound-related chromosome damage.

Neudecker and Henschler[21] reported on the mutagenicity of allyl chloride in the *Salmonella*/-mammalian microsome test. In the presence of S9 mix, allyl chloride exerted considerable indirect mutagenic activity. An increase in the concentration of rat liver homogenate fraction (S9) did not enhance mutagenicity. Longer standard incubation times (120 minutes instead of 20 minutes) at 37°C also led to an increase in mutagenic activity.

Eder et al.[22] investigated the mutagenic potential of allyl chloride using *Salmonella-typhimurium* strain TA 100 in a modified Ames mutagenicity assay system. Allyl chloride clearly proved to be mutagenic, even without the addition of S9 mix to the mutagenicity test system. Allyl chloride was also clearly positive in the nitrobenzyl–pyridine (NBP) test. Therefore, in the judgment of the authors, it can be regarded as a directly acting mutagen.

Bignami et al.[23] tested allyl chloride for its ability to induce gene mutations in prokaryotic and eukaryotic microorganisms. The *Salmonella* reversion test with strains TA1535 and TA100 (with and without metabolic activation), a forward and back mutation system in *Streptomyces coelicolor* and two forward mutation systems in *Aspergillus nidulans* were used. The spot and plate incorporation assay techniques were employed. Allyl chloride was active in *S. typhimurium* and *S. coelicolor* and negative in *A. nidulans*.

Schiffmann and co-workers[24] tested allyl chloride for its ability to induce unscheduled DNA synthesis (UDS) in HeLa cells and mutations in the Ames test. The effective dose range of UDS induction was rated according to the lowest dose at which UDS occurred. Allyl chloride required a relatively high dose of 0.001 mol/L to induce UDS. The authors indicated that the test results represented an important confirmation of previously demonstrated correlations between bacterial mutagenicity in the Ames test and alkylating activity in the NBP test.

The U.S. National Toxicology Program Annual Plan for fiscal Year 1983 reported the results of studies on rat dominant lethal; mouse sperm head morphology; *Drosophila* sex-linked recessive lethal; *in vitro* UDS in human fibroblasts; and rat bone

marrow cytology studies, all of which were negative.[25]

Pharmacokinetics/Metabolism Studies

Waechter et al.[26] investigated the pharmacokinetics and metabolism of allyl chloride when administered by the oral, inhalation and intravenous routes to Fischer 344 rats. The pharmacokinetic fate of allyl chloride appeared to be dose dependent and route dependent following oral or inhalation exposure. Allyl chloride caused marked depletion of nonprotein sulfhydryls (NPSH) in the liver, kidney, and lung in rats exposed at 1000 or 2000 ppm for a single 6-hour exposure. A single 6-hour exposure of rats at 100 ppm caused a slight but statistically significant decrease in liver, kidney, and lung NPSH concentrations. NPSH was not diminished in the kidney, liver, lung, or blood in rats exposed at 10 ppm allyl chloride vapor for 6 hours. Rats exposed at 1000 to 2000 ppm allyl chloride vapor for 6 hours showed treatment-related cytotoxicity in the kidneys when sacrificed 48 hours postexposure.

Human Studies

Human sensory tests were conducted[12] using 13 human volunteers who evaluated the presence or absence of the characteristic odor of allyl chloride at a concentration of 3 ppm. Volunteers remained in the concentration for 1 to 3 minutes and each, independently, gave a verbal report to the tabulator. Ten of 13 volunteers reported a definite odor, but no irritation, when exposed at 3 ppm for 1 to 3 minutes. However, the authors emphasized the warning properties are very inadequate insofar as providing any degree of protection when repeated exposure is likely.

Dow Chemical Company reported[27] that eye contact with liquid allyl chloride may cause pain and severe irritation with corneal injury that may result in permanent impairment of vision, even blindness. Vapors may irritate the eyes and the effects may be delayed. Prolonged or repeated skin exposure may cause severe skin burns, possibly of a delayed nature, or deep-seated pain. The effects may be more severe if the chemical is confined to the skin. A single, prolonged skin exposure may result in the material being absorbed in harmful amounts. He and Zhang[28] studied the effects of allyl chloride on occupationally exposed subjects in two factories manufacturing sodium allyl sulfonate. Twenty-six subjects in one factory were exposed to allyl chloride at levels of 2.6 to 6650 mg/m^3 (0.83–2127 ppm) for 2.5 months to 6 years; in another factory, 27 subjects were exposed at 0.2 to 25.13 mg/m^3 (0.06–8.03 ppm) for periods of 1 to 4.5 years. Information regarding possible dermal exposures was not provided. Most workers in the first factory had weakness, paresthesia, and numbness in the extremities. Electroneuromyography showed neurogenic abnormalities in 10 of the 19 subjects examined; the prevalence of neuropathy, therefore, was 52.6%. Similar symptoms of workers in the second factory were much milder; there were few abnormal neurological signs, yet electroneuromyographic findings indicating mild neuropathy were found in 13 of the 27 subjects. Evidence obtained indicated chronic allyl chloride exposure caused toxic polyneuropathy.

TLV Recommendation

Based on the 1959 report of Torkelson et. al.,[12] a TLV–TWA of 1 ppm is recommended to prevent liver injury and potential renal effects. A TLV–STEL of 2 ppm is also recommended, to provide additional protection for the liver and to protect against potential eye and respiratory irritation. A Skin notation is also recommended, even though the industry reports on which it is based were not available for review. Based on data from the 1978 NCI gavage study with mice[14] that produced neoplastic lesions of the forestomach and the positive mouse skin tumor initiation study by Van Duuren et. al.,[15] the carcinogenicity notation of A3, Confirmed Animal Carcinogen with Unknown Relevance to Humans, is warranted. ACGIH solicits additional quantitative data, which may help further evaluate the need for a Skin notation. Sufficient data were not available to recommend a SEN notation.

Historical TLVs

1957–1962: TLV–TWA, 5 ppm
1963–present: TLV–TWA, 1 ppm
1976–present: TLV–STEL, 2 ppm
1995: *Proposed:* A3, Confirmed Animal Carcinogen with Unknown Relevance to Humans
1996–present: TLV–TWA, 1 ppm; TLV–STEL, 2 ppm; A3

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