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TOXICOLOGICAL EVALUATIONS

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TOXICOLOGICAL EVALUATION

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Propargyl alcohol

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Propargyl alcohol

This Toxicological Evaluation replaces a previously published version in volume 2 of the book series “Toxicological Evaluations” published by Springer. In addition to propargyl alcohol, Toxicological Evaluations on the structurally related compounds butynediol (volume 10, revised version will also be published soon via internet) and 2-methyl-3-butyn-2-ol (volume 6, revised version will also be published soon via internet) are available and can be consulted for comparison.

1 Summary and assessment

Propargyl alcohol is metabolised to propargyl aldehyde in vitro.

Based on acute toxicity studies in experimental animals, propargyl alcohol should be evaluated as toxic on single oral, dermal and inhalative administration (LD_{50} rat oral 35 to 110 mg/kg body weight; LD_{50} mouse oral 50 mg/kg body weight; LD_{50} rabbit dermal 16 to 32 and 88 mg/kg body weight, depending on the source of information; LC_{50} rat, 2 hours: 2000 mg/m³, and 1 hour: 2748 (male rats) and 2384 mg/m³ (female rats)). An atmosphere enriched with propargyl alcohol at 20 °C or 24 °C is lethal for the exposed rats within a few minutes.

In two 28-day studies in rats, oral doses of propargyl alcohol ranging from 5 to 60 mg/kg body weight dose-dependently led to increased relative liver and kidney weights, signs of hypochromic anaemia, elevated liver enzyme activities and histopathological changes in the liver (megalocytosis) at doses of more than 15 mg/kg body weight. The *no observed adverse effect level* is reported as being 5 mg/kg body weight. In contrast, an exploratory study in rats receiving oral treatment with propargyl alcohol at doses of 0.1 to 10 mg/kg body weight for 14 days gave no findings up to the highest test dose of 10 mg/kg body weight inclusive. A 14-day exposure of rats to propargyl alcohol at concentration levels of 0 (controls), 10, 50 and 200 ppm (equivalent to analytical concentrations of 0, approx. 22, 115 and 456 mg/m³) led to retardation of body weight development at exposure levels above 50 ppm and, in particular, caused irritation of the mucous membranes at the top concentration. At 200 ppm liver enzyme activities and relative liver weights

were elevated. Histopathological examination revealed changes in the nasal mucosa at levels of 50 ppm and above and liver changes at 200 ppm.

In the rabbit, propargyl alcohol has an irritant to corrosive effect on the skin, depending on the concentration. The undiluted substance is corrosive to the rabbit eye.

In rats, subchronic oral administration of propargyl alcohol for 90 days at dose levels of 0 (controls), 5, 15 and 50 mg/kg body weight was observed to cause altered haematological parameters in the form of anaemia and increased relative liver weights at doses of 15 mg/kg body weight and above; at 50 mg/kg body weight increased relative kidney weights (male rats) and changes in the clinical chemistry parameters were also seen, and beginning at 15 mg/kg body weight propargyl alcohol caused histopathological changes of the liver (megalocytosis) and the kidney (karyomegaly in the tubular epithelial cells). In rabbits, dermal application of propargyl alcohol to the intact or scarified skin for 91 days showed no treatment-related effects up to the highest test dose of 20 mg/kg body weight. In a subchronic 90-day inhalation study in rats exposed to concentrations of 0 (controls), 1, 5 and 25 ppm (equivalent to analytical concentrations of 0, approx. 2.5, 12 and 56 mg/m³) the highest concentration (25 ppm) led to increased relative liver and kidney weights without histopathological correlates. In a further 90-day inhalation study in rats exposed to 80 ppm (equivalent to approx. 183 mg/m³), increases in leukocyte counts, elevated alanine aminotransferase activity, increased relative liver weights and histopathological changes of the liver were found. Findings which were similar in tendency to the oral studies were obtained in a 5-month inhalation study in rats, although due to the insufficient documentation the results are of limited usefulness for assessment purposes.

In the Salmonella/microsome test, propargyl alcohol has not shown any evidence of mutagenicity in *Salmonella typhimurium* strains TA 97, TA 98, TA 100, TA 102, TA 1535, TA 1537 and TA 1538. A Salmonella/microsome test using strain D3052 showed a weakly positive result in the absence of metabolic activation. In the chromosome aberration test in CHO cells, propargyl alcohol gave a positive result with and without metabolic activation. In contrast, two mouse micronucleus tests conducted independently of one another did not reveal any clastogenic effect following oral administration.

No reproductive toxicity studies have been conducted with propargyl alcohol itself, but studies with structurally related compounds (butynediol and 2-methylbutyn-3-ol-2, cf. volumes 10 and 6, respectively) have failed to show a foetotoxic effect in prenatal rats. None of the studies involving repeated administration of propargyl alcohol have revealed macroscopic or histopathological changes in the testes or ovaries.

In the Federal Republic of Germany, the Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area ("MAK-Kommission") of the Deutsche Forschungsgemeinschaft has established a MAK value (maximum workplace concentration) for propargyl alcohol of 2 ppm (4,7 mg/m³). The limitation of peak concentrations was set according to category I, excursion factor 2. Because of the risk of percutaneous absorption, the chemical has been designated with "H" in addition.

In the context of the US National Toxicology Program, carcinogenicity studies, including subchronic dose-finding studies, are currently being conducted in rats and mice.

2 Name of substance

2.1	Usual name	Propargyl alcohol
2.2	IUPAC name	2-Propyn-1-ol
2.3	CAS No.	107-19-7
2.4	EINECS No.	203-471-2

3 Synonyms, common and trade names

Acetylene carbinol
Agrisynth PA
Ethynylcarbinol
Golpanol PA
1-Hydroxy-2-propyne
3-Hydroxy-1-propyne
Methanol, ethynyl
Propargylalkohol

Prop-2-in-1-ol
2-Propyn-1-ol
3-Propynol
1-Propyne-3-ol
2-Propynyl alcohol

4 Structural and molecular formulae

4.1	Structural formula	$HC{\equiv}C\text{-}CH_2OH$
4.2	Molecular formula	C_3H_4O

5 Physical and chemical properties

5.1	Molecular mass, g/mol	56.06	
5.2	Melting point, °C	–48	(GAF, 1988)
		–51.8	(Lide and Frederikse, 1996)
		–53	(BASF, 1996)
5.3	Boiling point, °C	113,6	(Lide and Frederikse, 1996)
		114–115	(BASF, 1996)
		115	(GAF, 1988)
5.4	Vapour pressure, hPa	0.947 (at 20 °C)	(BASF, 1996)
		0.9478 (at 20 °C)	(Lide and Frederikse, 1996)
		0.9484 (at 20 °C)	(GAF, 1988)
5.5	Density, g/cm³	11.6 (at 20 °C)	(GAF, 1988)
		18 (at 20 °C)	(BASF, 1996)
5.6	Solubility in water	Miscible in all proportions	(BASF, 1996; Rowe and McCollister, 1982)
5.7	Solubility in organic solvents	Miscible with benzene, chloroform, 1,2-dichloroethane, ethanol, ether, acetone, dioxan, tetrahydrofuran, pyridine; does not mix with aliphatic hydrocarbons	(Rowe and McCollister, 1982)
		Miscible with ethanol and ethyl ether, soluble in chloroform	(Lide and Frederikse, 1996)

5.8	Solubility in fat	Partition coefficient n-octanol/water, log P_{ow}: –0.35	(BASF, 1996)
5.9	pH value	7 (at 330 g/l, 20 °C)	(BASF, 1996)
5.10	Conversion factor	1 ml/m³ (ppm) $\triangleq$ 2.29 mg/m³ 1 mg/m³ $\triangleq$ 0.437 ml/m³ (ppm) (at 1013 hPa and 25 °C)	

6 Uses

Used as a corrosion inhibitor, solvent for cellulose acetate, glazing agent in electroplating, stabiliser for chlorinated hydrocarbons, as a herbicide and as an intermediate in organic syntheses (Falbe et al., 1985).

7 Experimental results

7.1 Toxicokinetics and metabolism

Propargyl alcohol is presumably metabolised to propargyl aldehyde. The corresponding conversion was demonstrated by experimental evidence from in-vitro studies using bovine liver catalase. The investigators suspected that the catalytic conversion of propargyl alcohol to propargyl aldehyde takes place preferentially via catalase and to a much lesser extent via alcohol dehydrogenase. Furthermore, they pointed out the possibility that this type of conversion could also take place via reactions involving hydroxyl radicals or cytochrome P-450 catalysis (DeMaster and Nagasawa, 1978; DeMaster et al., 1986, 1994).

There are indications from in-vitro studies with phenobarbital-induced rat liver microsomes that propargyl alcohol binds weakly to cytochrome P-450. In these studies, propargyl alcohol reduced the haem and cytochrome P-450 levels in the rat liver microsomes (Ivanetich et al., 1978).

7.2 Acute and subacute toxicity

Acute toxicity

In animal studies, propargyl alcohol has proved toxic to various species following oral administration, dermal application and inhalation exposure. The values for LD_{50} and LC_{50} reported in the literature are compiled in Table 1 below.

Beginning of Table 1

Table 1. Acute toxicity of propargyl alcohol					
Species, strain, sex*	Route	Dose (mg/kg b.w. or mg/m³)	Effects	Observation period	Reference
Rat, Sprague-Dawley, male	oral	93	LD_{50}	14 days	Vernot et al., 1977
Rat, Sprague-Dawley, female	oral	54	LD_{50}	14 days	Vernot et al., 1977
Rat, Sprague-Dawley, male	oral	110	LD_{50}	2 days	Archer, 1985
Rat, Sprague-Dawley, female	oral	55	LD_{50}	2 days	Archer, 1985
Rat	oral	35	approximate LD_{50}; mortality at 50 mg/kg b.w. was 3/3 within 24 hours, at 20 mg/kg b.w. it was 0/3, no noticeable clinical signs at this dose level	no information	Dow, 1952, 1964
Rat, male, female	oral	54.9	LD_{50}; agitation, accelerated breathing, lying on the abdomen; necropsy: hepatomegaly, blood in the enteral contents, pulmonary haemorrhages	7 days	BASF, 1963
Rat	oral	70	LD_{50}	no information	GAF, year not given
Mouse	oral	50	lethal dose	no information	Eastman Kodak, 1964
Mouse	oral	50	LD_{50}; at 30 mg/kg b.w. no clinical signs; at ≥ 100 mg/kg b.w. apathy, reduced motility, lying on the abdomen; necropsy of the deceased animals: congestion of the internal organs and the brain, haemorrhages in the gastric mucosa, yellowish discoloration of the liver	14 days	Stasenkova and Kochetkova, 1966

Table 1. Acute toxicity of propargyl alcohol					
Species, strain, sex*	Route	Dose (mg/kg b.w. or mg/m³)	Effects	Observation period	Reference
Rabbit	oral	> 19 < 38	approximate LD_{50}; accelerated breathing, diarrhoea, atonia, lying on the side, tonic convulsions; proteinuria, erythrocytes and renal epithelia in the urinary sediment; necropsy: accumulation of fluid in the pleural space and the pericardial sac, congestion of the internal organs, haemorrhages in the stomach, intestines and muscles	no information	BASF, 1987
Guinea pig	oral	> 38 < 95	approximate LD_{50}; apathy, atonia; liver and kidney damage	no information	BASF, 1987
Guinea pig	oral	60	LD_{50}	no information	GAF, year not given
Cat	oral	> 10 < 19	approximate LD_{50}; necropsy: haemorrhages in the thymus gland and the abdominal cavity, gastric mucosal lesions, liver and kidney damage	no information	BASF, 1987
Rat	dermal	1894	exposures of > 3 minutes were lethal to all 5 animals used; a 1-minute exposure was survived by all 5 animals; apathy and irregular breathing; animals exposed for 10 minutes showed local tissue necrosis and skin bleeding as well as haemorrhages in the thymus and the jejunum	no information	BASF, 1987
Mouse	dermal	no information	immersion of the tails up to 2/3 of their length into the test substance for 30 minutes or one hour resulted in death in all of 20 animals; necropsy: congestion of the internal organs, swellings and haematomas in the brain	no information	Stasenkova and Kochetkova, 1966
Rabbit, New Zealand, female	dermal	88	LD_{50}	14 days	Vernot et al., 1977

Table 1. Acute toxicity of propargyl alcohol					
Species, strain, sex*	Route	Dose (mg/kg b.w. or mg/m³)	Effects	Observation period	Reference
Rabbit	dermal	16–32	LD_{50}; mortality at 63 mg/kg b.w. was 2/2 after 2 days, at 32 and 16 mg/kg b.w. it was 1/2 after 11 and 7 days, respectively, at 8 mg/kg b.w. it was 0/2; slight diarrhoea, hyperaemia and moderate oedema	no information	Dow, 1957, 1964
Rabbit	dermal	47–190	mortality at 95 and 190 mg/kg b.w. was 3/3, mortality at 47 mg/kg b.w. was 2/3; apathy and diarrhoea, local tissue necrosis; necropsy of the deceased animals: at 180 mg/kg b.w. gastric haemorrhages, severely injected intestinal vessels	21 days	BASF, 1963, 1987
Rat, Sprague-Dawley, male	inhalation (1 hour)	2748 (1200 ppm)	LC_{50}	no information	Vernot et al., 1977
Rat, Sprague-Dawley, female	inhalation (1 hour)	2382 (1040 ppm)	LC_{50}	no information	Vernot et al., 1977
Rat, Sprague-Dawley, male, female	inhalation (1 hour)	3412 (1490 ppm)	lethal concentration; hunched posture, rough haircoat, lacrimation and salivation, low body temperature, apathy, prostration	–	Hazleton, 1989
Rat	inhalation (1 hour)	3000	mortality 1/10 after 3 days; mild mucous membrane irritation; necropsy of the deceased animal: intestinal irritation; necropsy of the surviving animals: no findings	7 days	BASF, 1965
Rat	inhalation (2 hours)	2000	LC_{50}; mortality at 1500, 2000 and 8000 mg/m³ was 1/10, 5/10 and 10/10, respectively; mucous membrane irritation, motor excitation, apathy, dyspnoea, lying on the side	3 days	Stasenkova and Kochetkova, 1966

Table 1. Acute toxicity of propargyl alcohol					
Species, strain, sex*	Route	Dose (mg/kg b.w. or mg/m³)	Effects	Observation period	Reference
Rat	inhalation	vapour-enriched atmosphere at 20 °C	inhalation hazard test; mortality after a 3-minute exposure was 6/12, after a 10-minute exposure it was 6/6, after 1 and 3-hour exposures 6/6 each; flight reactions; mucous membrane irritation, pallor of the ears and paws, dyspnoea; necropsy findings: occasional haemorrhages in the gastrointestinal tract	no information	BASF, 1963
Rat	inhalation	vapour-enriched atmosphere at 24 °C	inhalation hazard test; mortality after a 6-minute exposure was 2/3, after 12- and 30-minute exposures it was 3/3, after a 2-hour exposure 3/3	14 days	Dow, 1952
Mouse	inhalation (1 hour)	3000	mortality was 3/10 after one day; mild mucous membrane irritation; necropsy of the deceased animals: irritated colon; necropsy of the surviving animals: no findings	7 days	BASF, 1965
Mouse	inhalation (2 hours)	2000	LC_{50}; mortality at 500, 1500, 2000 and 3500 mg/m³ was 0/20, 1/20, 10/20 and 20/20, respectively; mucous membrane irritation, motor excitation, apathy, dyspnoea, lying on the side	3 days	Stasenkova and Kochetkova, 1966
Guinea pig	inhalation (1 hour)	3000	mortality was 0/6; mild mucous membrane irritation	no information	BASF, 1965
Rabbit	inhalation (1 hour)	3000	mortality was 0/2; mild mucous membrane irritation; impaired hepatic and renal function (positive urine tests for urobilinogen and protein, slightly elevated blood levels of aminotransferases and/or urea)	14 days	BASF, 1965

Table 1. Acute toxicity of propargyl alcohol					
Species, strain, sex*	Route	Dose (mg/kg b.w. or mg/m³)	Effects	Observation period	Reference
Cat	inhalation (1 hour)	3000	mortality was 1/2; mild mucous membrane irritation; apathy, vomiting, lying on the side and lack of appetite in the deceased animal; impaired hepatic and renal function (positive urine tests for urobilinogen and protein, slightly elevated blood levels of aminotransferases and/or urea)	14 days	BASF, 1965
No information	inhalation (6 hours)	380–4020	at 4020 mg/m³ all animals died after 5¾ hours, at 1600 mg/m³ all animals died within 1½ hours after the end of exposure, at 790 mg/m³ deaths occurred within 48 hours after the end of exposure, while all animals survived 380 mg/m³	no information	Eastman Kodak, 1964
Mouse	intraperitoneal	50	lethal dose	no information	Eastman Kodak, 1964
Mouse	intraperitoneal	44	LD_{50}; agitation, accelerated breathing, lying on the abdomen; necropsy: blood in the enteral contents, pulmonary haemorrhages	7 days	BASF, 1963
Rabbit	subcutaneous	47–474	at 474 and 237 mg/kg b.w. death of the treated animal occurred 2½ hours after administration in both cases, diarrhoea, paresis, dyspnoea; 47 mg/kg b.w. was survived by the treated animal, administration of 94 mg/kg b.w. the next day resulted in death 6½ hours after dosing	no information	Tietze, 1926
* if specified b.w. body weight					

End of Table 1

Following oral administration, LD_{50} values determined in rats after observation periods of 2 to 14 days ranged from 35 to 110 mg/kg body weight (Vernot et al., 1977; Archer, 1985; Dow, 1952, 1964; BASF, 1963; GAF, year not given), the LD_{50} value ascertained for mice being 50 mg/kg body weight (Eastman Kodak, 1964; Stasenkova and Kochetkova, 1966). For rabbits, guinea pigs and cats, oral LD_{50} values were reported as > 19 < 38, > 38 < 95 and > 10 < 19 mg/kg body weight, respectively (BASF, 1987; GAF, year not given). Following dermal application, LD_{50} values found for the rabbit were between 16 and 88 mg/kg body weight (Dow, 1957, 1964; Vernot et al., 1977). The LC_{50} values established in rats after 2- and 1-hour exposures to propargyl alcohol were 2000 and 2382 (females), respectively, and 2748 mg/m³ (males; Stasenkova and Kochetkova, 1966; Vernot et al., 1977). In inhalation hazard tests conducted in rats, even a 3-minute exposure to an atmosphere enriched with propargyl alcohol was lethal to half of the animals (6/12; BASF, 1963). In mice, the LD_{50} value determined for single intraperitoneal injection was reported as 44 mg/m³. Clinical signs seen in rats, mice and rabbits upon oral administration and dermal application included diarrhoea, motor excitation, atonia, accelerated breathing, lying on the abdomen and tonic convulsions (BASF, 1963, 1987; Stasenkova and Kochetkova, 1966). Following dermal application, local tissue necrosis and bleeding of the skin were observed in rats and rabbits (BASF, 1987). Clinical signs seen in rats and mice following acute inhalation exposure to propargyl alcohol included mucous membrane irritation, motor excitation, apathy, hunched posture, lying on the side and dyspnoea (BASF, 1963; Hazleton, 1989; Stasenkova and Kochetkova, 1966). Necropsy conducted upon acute oral administration to rats revealed hepatomegaly, blood in the enteral contents and pulmonary haemorrhages (BASF, 1963). In rabbits, proteinuria as well as erythrocytes and renal epithelia in the urinary sediment were observed following oral administration (BASF, 1987). In this species, necropsy revealed congestion of the internal organs as well as haemorrhages in the stomach, intestines and muscles (BASF, 1987). In the rat, haemorrhages in the thymus gland and the jejunum were also observed after dermal application, while occasional gastrointestinal haemorrhages were seen following inhalation exposure (BASF, 1963, 1987). In rabbits and cats, there were findings indicating impaired hepatic and renal function (positive urine tests for urobilinogen and protein, slightly elevated blood levels of aminotransferases and/or urea) after single inhalation exposure (BASF, 1965).

Subacute toxicity

In an exploratory subacute toxicity study, groups of 10 male and 10 female Sprague-Dawley rats (initial weight 200 to 300 g) received propargyl alcohol (≥ 97% pure) at doses of 0, 0.1, 1 and 10 mg/kg body weight in corn oil by oral gavage for 14 consecutive days. In previously conducted studies, 100 mg/kg body weight had proved toxic (no further details, but presumably lethal). All doses were tolerated without externally visible signs of toxicity and without any effect on body weight development. Haematology, clinical chemistry and activity determinations of the xenobiotic-metabolising enzymes, aminopyrine demethylase and ethoxyresorufin deethylase, as well as macroscopic and microscopic examinations (of 29 organs) revealed no substance-related changes. The *no observed effect level* was thus 10 mg/kg body weight (Komsta et al., 1989).

In a study complying with OECD guideline No. 407, groups of 10 male and 10 female Wistar rats (Bor:WISW(SPF-Cpb)) received propargyl alcohol (99% pure) in aqueous solution at daily doses of 0 (controls), 5, 15 and 45 mg/kg body weight by oral gavage for 4 weeks. At and above a dose level of 5 mg/kg body weight, there were significant increases in relative liver and kidney weights without histopathological correlates. From 15 mg/kg body weight, there were signs of hypochromic anaemia (dose-dependently reduced haemoglobin levels and haematocrit values). The 45 mg/kg body weight dose resulted in retardation of body weight gain, elevated activities of alanine aminotransferase, alkaline phosphatase and glutamate dehydrogenase in the male rats, inhibition of cholinesterase activity in the female rats and histopathologically confirmed hepatocellular damage (markedly enlarged nuclei of the peripherolobular hepatocytes, formation of perinuclear spaces in the cytoplasm of the altered hepatocytes, occasional nuclear vacuoles and chromophilic single-cell necrosis, slightly high parenchymal mitotic rate, minor focal bile duct proliferation in one animal). In a subsequent 4-week study with varying doses of 50 and 60 mg/kg body weight (60 mg/kg from days 1 to 3, 50 mg/kg from days 4 to 21, 60 mg/kg from days 22 to 28; 15 animals/group) marked signs of intoxication occurred (apathy, atonia, salivation with bloody saliva), and 1 out of 15 treated male rats died on the third day of the study. At post-mortem, this animal was observed to have extensive haemorrhages of the gastric mucosa. Moreover, at these dose levels, the γ-glutamyl transferase activity was markedly increased. In addition, the N-demethylase activity and the cytochrome P-450

levels were significantly reduced in both sexes. Dose levels of and above 15 mg/kg body weight were considered by the investigators to be toxic, and due to slight increases found in the relative liver and kidney weights, 5 mg/kg body weight was regarded as the threshold dose (Bayer, 1984).

Groups of 10 male and 10 female Crl:CD(SD)BR rats received propargyl alcohol (> 99% pure) doses of 0 (controls), 5, 15 and 50 mg/kg body weight per day by oral gavage over a period of 28 days (interim sacrifice of a subchronic toxicity study; see Section 7.5; TRL, 1988). The investigations included the recording of body weight development and food consumption, blood and clinical pathology evaluation as well as histopathological examination of the liver. The 50 mg/kg body weight dose was lethal to one male. Clinical signs of toxicity included an increased incidence of salivation in the high dose group. In this dose group, the body weights of the males were significantly lower than the controls, while in the females there was only a tendency towards lower body weights relative to the controls. The females of the 50 mg/kg dose group exhibited significantly reduced values for haemoglobin, mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration, whereas in the males the mean corpuscular haemoglobin concentration was reduced. Furthermore, the male rats had higher leukocyte and neutrophil counts than did the controls. In several animals of the high dose group, elevated serum enzyme activity levels (alanine and aspartate aminotransferase, lactate dehydrogenase) were observed. Glucose and sodium were found to be low in the males receiving the 50 mg/kg dose, whereas in the females of this dose group, inorganic phosphate levels were elevated. Histopathological examination revealed megalocytosis of the liver in 9 out of 10 male and 3 out of 10 female rats of the 15 mg/kg group as well as in all rats of the 50 mg/kg group (TRL, 1988).

Rabbits survived 20 oral administrations (5 per week) of propargyl alcohol at a dose level of 9.5 mg/kg body weight. In contrast, all animals receiving oral doses of 18.9 mg/kg body weight died after 9 to 23 days of treatment. The deceased animals exhibited bleeding in the gastrointestinal tract and the cardiac muscle, and peripheral vasodilation and signs of lung congestion and exudation were seen in addition. Histopathological examination revealed minor liver and kidney damage (no further details; BASF, 1987).

Two cats given daily oral administrations (5 per week) of 9.5 mg propargyl alcohol/kg body weight died on days 16 and 17 of treatment. Toxicity was

characterised by vomiting, lack of appetite and body weight loss, one of the two animals also exhibiting considerable impairment of liver function. Histopathological examination revealed liver and kidney damage (no further details; BASF, 1987).

Inhalation of propargyl alcohol by 10 mice, 10 rats, 4 guinea pigs, 2 rabbits and one cat at concentration levels of approx. 1300 ppm (equivalent to approx. 2977 mg/m³; static study, nominal concentration) for 1 hour per day on 5 consecutive days led to the findings compiled in Table 2. The post-exposure observation period for the surviving animals was 3 weeks. Development of body weight was normal in the survivors. Clinical examination of the blood (including clotting), urine, liver and kidney revealed no abnormal findings (no further details; BASF, 1965).

Table 2. Subacute inhalation toxicity of propargyl alcohol (approx. 1300 ppm one hour/day for 5 days, static study, nominal concentration; BASF, 1965)

Species	Dead/exposed animals	Findings
Rat	4/10 (after 3–4 exposures)	irritation of the mucous membranes; deceased animals: liver damage (jaundice); surviving animals: no findings
Mouse	7/10 (after 2–3 exposures)	irritation of the mucous membranes; deceased animals: suspicion of liver damage; surviving animals: no findings
Guinea pig	0/4	irritation of the mucous membranes
Rabbit	1/2 (6 days after the 5th exposure)	irritation of the mucous membranes; deceased animal: hydrothorax, fatty liver; surviving animal: no findings
Cat	1/1 (after 2 exposures)	irritation of the mucous membranes, lying prone, tonic clonic spasms, opisthotonus; autopsy: petechial bleeding in various organs, surface of the liver spotted, bloody mucus in the stomach, liquid blood in the abdominal cavity; histopathologically toxic dystrophy of the liver

In a preliminary study to a 90-day inhalation study (see Section 7.5; BASF, 1992), groups of 5 male and 5 female Wistar rats were exposed for 6 hours per day to propargyl alcohol concentrations of 0 (controls), 10, 50 and 200 ppm (analytical concentrations of 9.8, 50.4 and 199 ppm; equivalent to approx. 22, 115 and 456 mg/m³; purity of the test substance 99.4%) over a period of 2 weeks (10 exposures). The study was conducted in accordance with OECD guideline No. 412. The rats of the control group as well as those of the 10 and 50 ppm groups showed no clinical signs of toxicity. At 200

ppm, apathy, closed eyes, reddish nasal discharge and irregular breathing occurred during the exposures. In between exposures, particularly towards the end of the exposure period, the signs did not subside. In addition, there was a deterioration in general condition. One female of this group died. Body weight gain was significantly retarded in the males of the 50 ppm group and in the males and females of the 200 ppm group. At study termination, the rats of the 200 ppm group showed elevated activity levels of alanine aminotransferase and alkaline phosphatase. Thromboplastin time was prolonged and the serum bilirubin concentration was elevated. The reduction in total protein content in the serum of the 50 and 200 ppm females as well as the decreases in triglyceride and cholesterol levels noted in the top concentration group were also related to treatment. At autopsy, the male rats of the top concentration group had significantly higher relative liver weights, while the males and females of the mid and top concentrations groups had significantly increased relative kidney weights. Histopathological examination revealed lesions in the nasal mucosa (metaplasia was seen from 50 ppm) and the liver (hepatocellular hypertrophy and cytoplasmic granulation, parenchymal single-cell necrosis at 200 ppm; BASF, 1990).

Twelve rats and 20 mice (no indication of strain or sex) inhaled propargyl alcohol at concentrations ranging between 200 and 300 mg/m³ air for 2 hours per day over a period of 3 weeks. The animals exhibited reductions in body weight gain, neuromuscular stimulus threshold and haemoglobin levels. On study day 10, 2 of the mice died. Part of the mice in this study were subsequently exposed to a concentration of 600 mg/m³ (no further details). All animals receiving this treatment died within 1 or 2 days, while the untreated controls survived this concentration (no further details; Stasenkova and Kochetkova, 1966). Insufficient documentation of the study protocol and results (e.g. no details of analytical checks are given) render it unsuitable for the assessment of the systemic toxicity of propargyl alcohol following repeated inhalation administration.

Groups of 10 Swiss mice (20 to 25 g) inhaled propargyl alcohol (99% pure) at 88 ppm (RD_{50}, concentration which lowers the respiratory rate by 50%; equivalent to approx. 201 mg/m³) and 25.3 ppm (1/3 RD_{50}; equivalent to approx. 58 mg/m³) 4, 9 or 14 times for 6 hours per day. Subsequently, the respiratory and olfactory epithelium of the nasal cavity as well as the trachea and lungs were histopathologically examined. Changes were found in both the respiratory and the olfactory epithelium, but not in the trachea and

lungs (no further details). The histopathological findings indicated that the severity of the lesions in the nasal region varied with the duration of exposure but not with the concentration of test substance (Zissu, 1995).

7.3 Skin and mucous membrane effects

A 1-minute application of undiluted propargyl alcohol (no details of dose) to the backs of rabbits caused very slight oedema formation within 24 hours. Slight necrosis was seen 8 days after application. Applications lasting 5 or 15 minutes resulted in very slight oedema formation and bleeding of the skin. Eight days after application, severe necrosis and anaemia were evident. Observation of the irritant effect of a 20-hour application of the undiluted product to the dorsal skin and the ear was not possible as the two exposed rabbits died after 4 hours (BASF, 1963). The substance thus exhibited a corrosive potential in this study.

In a further study, a 1-hour application of undiluted propargyl alcohol to the intact abdominal skin of rabbits caused mild hyperaemia and moderate oedema as well as slight encrustation after a few days. One animal receiving a single application of undiluted substance died within 2 hours without skin damage (no further details). A 10-percent solution in water caused very mild hyperaemia and oedema. The animal died within 24 hours. Ten applications of a 1-percent aqueous solution was tolerated without local or systemic effects (no further details; Dow, 1952, 1964).

One hour upon instillation of 50 µl undiluted propargyl alcohol into the conjunctival sacs of rabbits (number of animals not given), slight reddening with pronounced oedema formation as well as slight clouding of the cornea were seen. These effects remained almost unchanged during the first days of the observation period. Eight days after application, a scar developed on the upper lid. Moreover, the reddening of the mucous membranes with the accompanying oedema and the clouding of the cornea had not cleared up by then (BASF, 1963).

In a further eye irritation study in rabbits, the instillation of undiluted propargyl alcohol into the conjunctival sac produced clear signs of pain, irritation and permanent damage to the cornea. When the eye was rinsed out, the same findings were recorded, but the corneal clouding was only very slight. Instillation of a 10-percent solution in water with and without rinsing the eye

caused a very slight pain reaction and irritation which lasted several days. A 1-percent solution in water was without any effect (Dow, 1952, 1964).

7.4 Sensitisation

Propargyl alcohol is described as nonsensitising in one safety data sheet (no further details; GAF, 1988).

7.5 Subchronic and chronic toxicity

To determine the subchronic oral toxicity of propargyl alcohol (> 99% pure), groups of 20 male and 20 female Crl:CD(SD)BR rats received daily doses of 0 (controls), 5, 15 and 50 mg/kg body weight by oral gavage as aqueous solutions for a period of 90 days. The investigations included the recording of body weight development and food consumption, blood and clinical pathology evaluation, ophthalmological examination as well as histopathological examination of numerous organs. By the end of the study, 4 male rats from the 50 mg/kg group had died, the deaths being considered substance-related. The animals of this dose group displayed marked salivation. Throughout the study, body weight development was significantly retarded in the males of the 50 mg/kg group, although food consumption tended to be increased. In the 5 and 15 mg/kg groups, body weights were comparable to those of the controls. The ophthalmological examination was without findings. Haematological determinations carried out at study termination showed the males of the 50 mg/kg dose group to have reduced values for haemoglobin, mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration, while the females only had reduced values for mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration. In the male rats of the 50 and 15 mg/kg groups, neutrophil counts were significantly increased. The activities of aspartate and alanine aminotransferase and alkaline phosphatase were significantly elevated in both sexes at the top dose level. The males of the top dose group additionally had high levels of glucose and sodium as well as bilirubin. Although the differences from the controls were not statistically significant, these alterations were considered by the authors to be treatment-related. In the high dose group both sexes were observed to have low cholesterol, globulin and creatinine levels, the males additionally showing low albumin levels.

The rats of the 5 mg/kg group exhibited no changes in the haematological and clinical pathology parameters. In the 50 and 15 mg/kg groups, the absolute and relative liver weights were significantly increased in both sexes. In the 5 mg/kg group, increases in absolute and relative liver weights were also observed, but they did not differ from the controls in a statistically significant manner. Increased absolute kidney weights were found in the female rats from the 50 and 15 mg/kg groups. Relative kidney weights were higher only in the males of the 50 mg/kg dose group. These findings were consistent with lesions seen histopathologically. The hepatic lesions found in all animals of the 50 and 15 mg/kg groups were predominantly megalocytosis with cytoplasmic vacuolisation as well as minor bile duct proliferation. The kidneys of the male rats from the 50 and 15 mg/kg groups showed dose-dependent karyomegaly in the tubular epithelial cells, whereas the female rats exhibited the same type of karyomegaly only in the 50 mg/kg group (TRL, 1988).

Groups of 2 or 3 male and female albino rabbits (2 to 3 kg) received dermal applications of propargyl alcohol at dose levels of 0 (controls), 1, 3 and 10 mg/kg body weight to the depilated intact or scarified skin. Applications of 0.1, 0.3 and 1-percent solutions in distilled water were carried out 5 days per week for 8 hours per day over a period of 91 days. Beginning on day 63, the top dose was increased to 20 mg/kg body weight (applied as a 2-percent solution). As regards body weight gain, haematology determinations (haemoglobin, erythrocytes, leukocytes, differential blood count), alkaline phosphatase and alanine aminotransferase activity levels as well as blood urea nitrogen, no treatment-related findings were observed in comparison with the controls at 45 days and study termination (haematology), and at 14 and 80 days (blood chemistry). Similarly, gross pathology and histopathological examination performed at autopsy revealed no significant differences compared with the controls. From these findings it was concluded that propargyl alcohol has no systemic effects following repeated dermal exposure. No local effects were seen (IBL, 1965).

The subchronic inhalation toxicity of propargyl alcohol (99.4% pure) was tested in male and female Wistar rats (average weight at study initiation was 251 and 169 g) in accordance with OECD guideline No. 413. Groups of 10 males and 10 females inhaled 0 (controls), 1, 5 and 25 ppm (analytical concentrations of 0, 1.1 ± 0.17, 5.1 ± 0.53 and 24.6 ± 2.15 ppm; equivalent to 0, approx. 2.5, 11.8 and 56 mg/m³, respectively) per day (5 times

per week) for 6 hours over a period of 90 days (65 exposures). Particularly during the first two weeks of the study, 25 ppm caused body weight retardation in the male rats. At this concentration, both sexes had increased relative liver and kidney weights at the end of the study. Serum cholinesterase activity was significantly reduced in the females of the top concentration group. Macroscopic and histopathological examination revealed no treatment-related findings. The 5 and 1 ppm exposure levels showed no effect (BASF, 1992).

In further subchronic inhalation toxicity studies (59 treatment days, total study duration 89 days) 12 male and 12 female rats were exposed (for 7 hours/day, 5 days/week) to an analytically determined propargyl alcohol concentration of 80 ppm (equivalent to approx. 183 mg/m³). Twelve male and 12 female rats served as controls. During the first day of the study, irritation of the mucous membranes and lethargic behaviour occurred. These symptoms did not reappear during further exposure. Body weights did not differ significantly from controls. Haematology results obtained at study termination showed significantly elevated white blood cell counts in both sexes. There were no alterations in the differential blood counts and red blood cell counts. In both sexes alanine aminotransferase activity was elevated, while the alkaline phosphatase activity and serum urea nitrogen values were within the ranges of the controls. At autopsy, the relative liver weights were increased, and the females also exhibited higher relative kidney weights. Histopathologically, there were degenerative liver changes (varying from focal necrosis to hydropic degeneration associated with cellular infiltration). In the female rats, the changes were more marked than in the males (no further details; Dow, 1964).

A group of 40 white rats inhaled propargyl alcohol at a mean concentration of 7 mg/m³ air for 4 hours per day over a period of 5 months (6 months according to other parts of the text and the figures) in a dynamic study. After 5 months, body weight was reduced by 5 to 10% and as of the third month, the central nervous stimulus threshold was significantly lower. From these observations it was concluded that the excitability of the central nervous system was increased. At the end of study month 5, a 65% decrease in haemoglobin and significantly elevated white blood cell counts were observed. At study termination, arterial blood pressure was significantly reduced. Further findings included reduced hippuric acid excretion in urine and an increase in plasma sulfhydryl compounds by 25%. Upon exposure, half of

the animals in the study were histopathologically examined while the other half was placed under observation for 4 weeks. By the end of the observation period, all findings in these rats had normalised. At autopsy, the animals sacrificed following exposure were found to have moderate pulmonary emphysema, nutmeg-like markings of the liver and some granulation tissue in the spleen. Histopathological alterations were observed in the lung, spleen and liver but mostly they were not very marked (Stasenkova and Kochetkova, 1966). However, insufficient documentation of the findings renders the study suitable only to a limited extent for the assessment of the systemic effect of propargyl alcohol following repeated inhalation exposure.

7.6 Genotoxicity

7.6.1 In vitro

In the Salmonella/microsome test, propargyl alcohol (no indication of purity) was found to produce a weak mutagenic effect (15 revertants/µmol) as compared with propargyl aldehyde (1370 revertants/µmol) in the his D 3052 strain of *Salmonella typhimurium* without metabolic activation, whereas the results obtained with strains TA 98 and TA 100 were negative. Metabolic activation (S9 mix from Aroclor 1254-induced rat liver) did not enhance the effect (no information on negative or positive control values; Basu and Marnett, 1984).

In a further Salmonella/microsome test conducted in *Salmonella typhimurium* strains TA 98, TA 100, TA 1535, TA 1537 and TA 1538, propargyl alcohol (approx. 99% pure) concentrations ranging from 4 to 2500 µg/plate caused no increase in revertant counts in the presence or absence of metabolic activation (S9 mix from Aroclor 1254-induced rat liver) and thus caused no gene mutations. Concentrations of and above 500 µg/plate were toxic to the bacteria (BASF, 1979).

Propargyl alcohol (97% pure) also proved devoid of mutagenicity in another Salmonella/microsome test. In this test, *Salmonella typhimurium* strains TA 97, TA 98, TA 100 and TA 102 were exposed to concentrations of 0 (control, DMSO), 0.0075, 0.025, 0.075, 0.25 and 0.75 µl/plate (equivalent to 0, 7.1, 23.7, 71, 237 and 711 µg/plate). The test was carried out in the presence and absence of metabolic activation (S9 mix from Aroclor

1254-induced rat liver). Compared with the controls, there was no increase in revertant counts. The 0.75 µl/plate concentration was found to be toxic to the bacteria in the presence of metabolic activation (Blakey et al., 1994).

Propargyl alcohol (97% pure) was studied in vitro with and without metabolic activation (S9 mix from Aroclor 1254-induced rat liver) for clastogenic effects in CHO cells of the Chinese hamster. The test concentrations employed in the absence of metabolic activation were 0 (control, DMSO), 3, 6 and 10 mM (equivalent to 168, 336 and 561 µg/ml), while in the presence of metabolic activation 0 (control, DMSO), 0.4, 0.6, 0.8 and 1.0 mM (equivalent to 22, 34, 45 and 56 µg/ml) were used. The cells were treated with the test substance for one hour and subsequently incubated for an additional period of 10 or 16 hours. No details were given as to the cytotoxicity of propargyl alcohol. Without metabolic activation, no chromosome aberrations were observed following preparation at 10 hours, but 10 mM produced a significant clastogenic effect of 0.11 aberrations/cell when preparation was carried out at 16 hours (control value was 0.04 aberrations/cell). In the presence of metabolic activation, there was no clastogenic effect either when preparation was carried out at 10 hours. Preparation following 16-hour incubation, however, showed a significant concentration-dependent increase in aberration rate of up to 0.24 aberrations/cell (controls had 0.005 aberrations/cell) starting at levels as low as 0.4 mM. In this context, particularly chromatid and chromosome breaks as well as exchanges were observed. Thus, propargyl alcohol proved to be clastogenic in this test system with and without metabolic activation (Blakey et al., 1994).

7.6.2 In vivo

In a micronucleus test, groups of 15 male and 15 female NMRI mice (mean initial weight 29.1 and 23.3 g, respectively) received single propargyl alcohol (99.4% pure) doses of 70 mg/kg body weight as an aqueous solution by oral gavage. This had been established in preliminary studies as the maximum tolerated dose. After 24, 48 and 72 hours, subgroups of 5 males and 5 females were killed and their femoral bone marrow was examined for micronuclei (1000 polychromatic erythrocytes/animal). Two subgroups, each comprising 5 males and 5 females, served as the negative and positive controls (50 mg endoxane/kg body weight). At 24 and 72 hours, the female mice showed a very small yet statistically significant increase in the

number of micronucleated polychromatic erythrocytes, which was within the historical control range, however, and therefore no toxicological significance was attributed to this finding. The number of normochromatic erythrocytes containing micronuclei was not increased. In addition, the ratio of polychromatic to normochromatic erythrocytes was unchanged in both sexes. Based on these results, propargyl alcohol proved not to be clastogenic in vivo (Hoechst, 1990).

In a further micronucleus test, propargyl alcohol was also devoid of clastogenicity. Groups of 5 male and 5 female C57BL mice aged 17 weeks were treated twice with propargyl alcohol (97% pure) doses of 0 (controls, olive oil), 24, 48 and 72 mg/kg body weight (equivalent to 0, 25, 50 and 75% of the LD_{50}), administered by oral gavage at an interval of 24 hours. Thirty-six hours after the second dose, the femoral bone marrow was collected, and 500 polychromatic erythrocytes/animal were examined for micronuclei. The top dose was lethal to all male mice, which rendered evaluation of these animals impossible. In the other dose groups, the ratio of polychromatic to normochromatic erythrocytes remained unchanged and there was no increase in micronucleated polychromatic erythrocytes. Propargyl alcohol produced no clastogenic effect in this study, either (Blakey et al., 1994).

7.7 Carcinogenicity

No information available.

7.8 Reproductive toxicity

No information available.

However, two structurally related compounds, butynediol (cf. Toxicological Evaluations, volume 10) and 2-methyl-3-butyn-2-ol (cf. volume 6), have been investigated in appropriate studies which were conducted in accordance with OECD guideline No. 414 to assess the embryotoxic and teratogenic effects.

Wistar rats received butynediol doses of 0 (controls), 10, 40 and 80 mg/kg body weight by oral gavage from days 6 to 15 of gestation. At 80 mg/kg body weight, there was maternal toxicity (reduced food intake, body weight

loss on days 6 to 8). One of 20 rats showed apathy, poor general condition and uterine bleeding and died on day 8. In 3.9% of foetuses/litter, skeletal variations were noted (accessory 14th ribs). No teratogenic effects were observed. The 40 and 10 mg/kg body weight doses were devoid of maternal toxicity, embryotoxicity and teratogenicity (BASF, 1995).

2-Methyl-3-butin-2-ol was administered orally at doses of 0 (controls), 45, 130 and 400 mg/kg body weight. The 400 mg/kg body weight dose caused maternal toxicity (initially transient reduction of food intake, weight loss, apathy, gait disturbance). In this dose group with maternal toxicity, mean foetal body weight was about 6% below that of the controls, and higher incidence rates of skeletal variations and retardations were noted. Treatment with 130 and 45 mg/kg body weight caused no substance-related effects. None of the doses showed teratogenic effects (BASF, 1997).

None of the studies involving repeated administration of propargyl alcohol revealed histopathological changes in the testes or ovaries (cf. Section 7.5).

7.9 Effects on the immune system

No information available.

7.10 Neurotoxicity

No information available.

7.11 Other effects

In vitro, propargyl alcohol inhibited the growth of *Saccharomyces cerevisiae* by 50% at a concentration level of 56 mg/l (IC_{50}; Koch, 1992; Koch et al., 1993).

8 Experience in humans

No information available.

9 Classifications and threshold limit values

In the Federal Republic of Germany, the Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area ("MAK-Kommission") of the Deutsche Forschungsgemeinschaft has established a MAK value (maximum workplace concentration) for propargyl alcohol of 2 ppm (4,7 mg/m³). The limitation of peak concentrations was set according to category I, excursion factor 2. Because of the risk of percutaneous absorption, the chemical has been designated with "H" in addition (DFG, 1999; TRGS 900, 1999).

In the USA, the TLV-TWA value is 1 ppm (equivalent to 2.3 mg/m³) with the notation "skin" (ACGIH, 1999).

For the former Soviet Union, a value of 1 mg/m³ is given (INRS, 1990; RTECS, 1996).

References

ACGIH (American Conference of Governmental Industrial Hygienists, Cincinnati, OH, USA)
TLVs and BEIs – Threshold limit values for chemical substances and physical agents – biological exposure indices (1999)

Archer, T.E.
Acute oral toxicity as LD_{50} (mg/kg) of propargyl alcohol to male and female rats
J. Environ. Sci. Health, B20, 593–596 (1985)

BASF AG, Gewerbehygienisch-Pharmakologisches Institut
Gewerbetoxikologische Vorprüfung – Propargylalkohol
Unpublished report, Study No. XIII 62 (1963)

BASF AG, Gewerbehygienisch-Pharmakologisches Institut
Bericht über die Prüfung der Inhalationstoxizität von Propargylalkohol im Vergleich zu Allylalkohol
Unpublished report No. XIII/62-63 (1965)

BASF AG, Gewerbehygiene und Toxikologie
Bericht über die Prüfung von Propin-1-ol-3 im Ames-Test
Unpublished report (1979)

BASF AG, Gewerbehygiene und Toxikologie
Written communication to BG Chemie of 18.02.1987

BASF AG, Abteilung Toxikologie
Range-finding Studie zur Inhalationstoxizität von Propargylalkohol als Dampf an Ratten – 14-Tage Versuch
Unpublished report, Project No. 36I0969/88060 (1990)
On behalf of BG Chemie

BASF AG, Abteilung Toxikologie
Study on the inhalation toxicity of Propargylalkohol as a vapor in rats – 90-day test
Unpublished report, Project No. 50I0969/88100 (1992)
On behalf of BG Chemie

BASF AG, Abteilung Toxikologie
Study of the prenatal toxicity of 2-butyne-1,4-diol (2-Butin-1,4-diol) in rats after oral administration (gavage)
Unpublished report, Project No. 30R338/91101 (1995)
On behalf of BG Chemie

BASF AG
Safety data sheet Propargylalkohol hochkonz. (1996)

BASF AG, Department of Toxicology
3-Methyl-1-butyn-3-ol – Prenatal toxicity in Wistar rats after oral administration (gavage)
Unpublished report, Project No. 30R0231/93059 (1997)

Basu, A.K., Marnett, L.J.
Molecular requirements for the mutagenicity of malondialdehyde and related acroleins
Cancer Res., 44, 2848–2854 (1984)

Bayer AG, Institut für Toxikologie
Propargylalkohol – Subakute orale Toxizitätsversuche an Ratten
Unpublished report No. 12653 (1984)

Blakey, D.H., Maus, K.L., Bell, R., Bayley, J., Douglas, G.R., Nestmann, E.R.
Mutagenic activity of 3 industrial chemicals in a battery of in vitro and in vivo tests
Mutat. Res., 320, 273–283 (1994)

DeMaster, E.G., Nagasawa, H.T.
Inhibition of aldehyde dehydrogenase by propiolaldehyde, a possible metabolite of pargyline
Res. Commun. Chem. Pathol. Pharmacol., 21, 497–505 (1978)

DeMaster, E.G., Shirota, F.N., Nagasawa, T.
Role of propiolaldehyde and other metabolites in the pargyline inhibition of rat liver aldehyde dehydrogenase
Biochem. Pharmacol., 35, 1481–1489 (1986)

DeMaster, E.G., Dahlseid, T., Redfern, B.
Comparative oxidation of 2-propyn-1-ol with other low molecular weight unsaturated and saturated primary alcohols by bovine liver catalase in vitro
Chem. Res. Toxicol., 7, 414–419 (1994)

DFG (Deutsche Forschungsgemeinschaft, Senatskommission zur Prüfung gesundheitsschädlicher Arbeitsstoffe)
MAK- und BAT-Werte-Liste 1999, Mitteilung 35
Wiley-VCH Verlag GmbH, Weinheim (1999)

Dow (The Dow Chemical Company), Biochemical Research Department
Results of range finding toxicological tests on propargyl alcohol
Report (1952)
NTIS/OTS 0510184

Dow (The Dow Chemical Company), Biochemical Research Laboratory
Range finding skin absorption tests on propargyl alcohol
Report (1957)
NTIS/OTS 0510183

Dow (The Dow Chemical Company), Biochemical Research Laboratory
Results of repeated exposure of male and female rats to 80 ppm of propargyl alcohol in air
Report (1964)
NTIS/OTS 0510182

Eastman Kodak Company, Laboratory of Industrial Medicine, Rochester, USA
Toxicity and health hazard summary propargyl alcohol
Report (1964)
NTIS/OTS 0510348

Falbe, J., Bahrmann, H., Lipps, W., Mayer, D.
Alcohols, aliphatic
In: Ullmann's encyclopedia of industrial chemistry
5th ed., vol. A1, p. 279–303
VCH Verlagsgesellschaft mbH, Weinheim (1985)

GAF (Deutschland) GmbH
DIN safety data sheet Propargyl Alkohol (1988)

GAF (General Anilin and Film Corporation), year not given
Cited in: Rowe and McCollister (1982)

Hazleton Laboratories America, Inc.
Acute inhalation toxicity study with propargyl alcohol in the rat
Report, HLA Study No. 257-115 (1989)
On behalf of GAF Corporation
NTIS/OTS 0516716

Hoechst AG, Pharma Research Toxicology and Pathology
Propargylalkohol – Micronucleus test in male and female NMRI mice after oral administration
Unpublished report No. 90.0017 (1990)
On behalf of BG Chemie

IBL (Industrial Biology Laboratories, Inc., USA)
90-day chronic skin absorption study with propargyl alcohol 11-72063 - B. #155
Report No. 2695 (1965)
On behalf of GAF
NTIS/OTS 0510181

INRS
Valeurs limites d'exposition professionnelle aux substances dangereuses de l'ACGIH, de la RFA et de l'URSS
Cahiers de Notes Documentaires No. 138, 1er trimestre, page 135–139 (1990)

Ivanetich, K.M., Lucas, S., Marsh, J.A., Ziman, M.R., Katz, I.D., Bradshaw, J.J.
Organic compounds. Their interaction with and degradation of hepatic microsomal drug-metabolizing enzymes in vitro
Drug Metab. Dispos., 6, 218–225 (1978)

Koch, H.P.
Der Hefe-Test: Eine Alternativmethode zur Bestimmung der akuten Toxizität von Arzneistoffen und Umweltchemikalien
Pharmazie, 47, 531–537 (1992)

Koch, H.P., Hofeneder, M., Bohne, B.
The yeast test: an alternative method for the testing of acute toxicity of drug substances and environmental chemicals
Methods Find. Exp. Clin. Pharmacol., 15, 141–152 (1993)

Komsta, E., Secours, V.E., Chu, I., Valli, V.E., Morris, R., Harrison, J., Baranowski, E., Villeneuve, D.C.
Short-term toxicity of nine industrial chemicals
Bull. Environ. Contam. Toxicol., 43, 87–94 (1989)

Lide, D.R., Frederikse, H.P.R. (eds.)
CRC handbook of chemistry and physics
77th ed., p. 3-294
CRC Press, Boca Raton, New York, London, Tokyo (1996)

Rowe, V.K., McCollister, S.B.
Alcohols
In: Clayton, G.D., Clayton, F.E. (eds.)
Patty's industrial hygiene and toxicology
3rd rev. ed., vol. IIC, p. 4671–4673
John Wiley & Sons, New York (1982)

RTECS (Registry of Toxic Effects of Chemical Substances)
2-Propyn-1-ol, RTECS Number UK 5075000
Produced by NIOSH (National Institute for Occupational Safety and Health) (1996)

Stasenkova, K.P., Kochetkova, T.A.
Toxikologische Eigenschaften von Propargylalkohol (German translation of the Russian)
Toksikol. Novykh. Prom. Khim. Veshchestv., 8, 97–111 (1966)
See also: Chem. Abstr., 67, 89293b (1967)

Tietze, K.
Wirkung der Acetylencarbonsäure (Propargylsäure)
Med. Klin., 48, 1843–1845 (1926)

TRGS (Technische Regeln für Gefahrstoffe) 900
Grenzwerte in der Luft am Arbeitsplatz
October 1996 issue, version of September 1999
Carl Heymanns Verlag KG, Cologne (1999)

TRL (Toxicity Research Laboratories, Ltd., USA)
Rat oral subchronic toxicity study
Unpublished report, TRL Study No. 042-004 (1988)
On behalf of Dynamac Corporation

Vernot, E.H., MacEwen, J.D., Haun, C.C., Kinkead, E.R.
Acute toxicity and skin corrosion data for some organic and inorganic compounds and aqueous solutions
Toxicol. Appl. Pharmacol., 42, 417–423 (1977)

Zissu, D.
Histopathological changes in the respiratory tract of mice exposed to ten families of airborne chemicals
J. Appl. Toxicol., 15 (3), 207–213 (1995)