

Glycidaldehyde; CASRN 765-34-4

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the [IRIS assessment development process](#). Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the [guidance documents located on the IRIS website](#).

STATUS OF DATA FOR Glycidaldehyde

File First On-Line 08/22/1988

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	yes	08/22/1988*
Inhalation RfC (I.B.)	not evaluated	
Carcinogenicity Assessment (II.)	yes	08/01/1991*

*A comprehensive review of toxicological studies was completed (05/27/05) - please see sections I.A.6. and II.D.2. for more information.

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Glycidaldehyde

CASRN — 765-34-4

Last Revised — 08/22/1988

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk

of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. Oral RfD Summary

Critical Effect	Experimental Doses*	UF	MF	RfD
Weight gain retardation, enlarged adrenals, hydroptic renal pelvis and hematopoietic effects	NOAEL: 10 ppm (29 mg/cu.m) converted to 1.09 mg/kg/day	3000	1	4E-4 mg/kg/day
Rat Subchronic Inhalation Study	LOAEL: 20 ppm (59 mg/cu.m) converted to 2.23 mg/kg/day			
Hine et al., 1961				

*Conversion Factors: 4 hours/24 hours, 60 days/84 days, 0.223 cu.m/day/0.35 kg rat (rat breathing rate/rat body weight), 0.5 absorption rate; thus, 29 mg cu.m x 4 hours/24 hours x 60 days/84 days x 0.223 cu.m/day/0.35 kg x 0.5 = 1.09 mg/kg/day

I.A.2. Principal and Supporting Studies (Oral RfD)

Hine, C.H., R.J. Guzman, M.K. Dunlap, R. Lima and G.S. Loquvam. 1961. Studies on the toxicity of glycidylaldehyde. Arch. Environ. Health. 2: 23- 30.

Hine et al. (1961) exposed groups of 10 male Long-Evans rats to glycidylaldehyde vapors at concentrations of 0, 10, 20, 40, and 80 ppm (29, 59, 118 and 236 mg/cu.m), 4 hours/day, 5 days/week for 12 weeks. Endpoints of toxicity assessed were general appearance, body weight and organ-to-body weight ratios, gross and microscopic pathological examination, and hematological parameters. A NOAEL of 10 ppm was determined from this study because the only effect was a significantly lower thymus weight than that of controls; the authors stated that the chance of experimental error was considerable because of the small size of the thymus.

Furthermore, no effect on thymus weight was observed at 20 ppm. At 20 ppm, retardation of weight gain, enlarged adrenals, hydropic renal pelvis and hematopoietic effects were observed. Some deaths were observed at 40 and 80 ppm. The NOAEL of 10 ppm (29 mg/cu.m) corresponds to a converted dose of 1.09 mg/kg/day (see Conversion Factor). When the converted dose is divided by an uncertainty factor of 3000, an RfD of 0.0004 mg/kg/day or 0.03 mg/day for a 70-kg man is obtained.

I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF — 10 for species to species extrapolation; 10 to protect sensitive humans; and 10 to extrapolate from subchronic to chronic exposure. An additional UF of 3 was added for lack of supporting reproductive and other toxicity data.

MF — None

I.A.4. Additional Studies/Comments (Oral RfD)

Glycidylaldehyde has not been tested for teratogenicity.

I.A.5. Confidence in the Oral RfD

Study — Low

Database — Low

RfD — Low

The study is given a low confidence level because it was a subchronic inhalation study and group size was small. The database is given a low confidence level because of the unavailability of supporting toxicity, teratogenic, and reproductive effect data. The RfD is given a low confidence level to reflect the lack of confidence in the study and database.

I.A.6. EPA Documentation and Review of the Oral RfD

Source Document — This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation — None

Agency Work Group Review — 12/15/1987

Verification Date — 12/15/1987

A comprehensive review of toxicological studies published through May 2005 was conducted. No new health effects data were identified that would be directly useful in the revision of the existing RfD for Glycidaldehyde and a change in the RfD is not warranted at this time. For more information, IRIS users may contact the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

I.A.7. EPA Contacts (Oral RfD)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Glycidaldehyde
CASRN — 765-34-4

Not available at this time.

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Glycidaldehyde
CASRN — 765-34-4
Last Revised — 08/01/1991

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. Evidence for Human Carcinogenicity

II.A.1. Weight-of-Evidence Characterization

Classification — B2; probable human carcinogen

Basis — Based on no human data and an increased incidence of malignant tumors in rats and mice following subcutaneous injection of glycidaldehyde and of skin carcinomas following dermal application to mice. Glycidaldehyde shows mutagenic activity in many assay systems and is known to be highly reactive because of the epoxide and the aldehyde groups. A number of structurally related epoxide compounds are also carcinogenic in experimental animals, including the analogues glycidol and propylene oxide.

II.A.2. Human Carcinogenicity Data

None.

II.A.3. Animal Carcinogenicity Data

Sufficient. Evidence of carcinogenicity from two different routes of administration and in two different species have been reported. Female ICR/Ha Swiss mice (50/group) received weekly subcutaneous injections of either 0.05 mL of tricapyrylin (vehicle control), or 0.1 mg or 3.3 mg glycidaldehyde in 0.05 mL tricapyrylin (30 mice/group) for life. These groups were not run concurrently. Two additional groups of 50 and 39 mice received no treatment; these groups were run concurrently with the low- and high-dose groups, respectively. As a positive control three groups of mice received weekly injections of 0.1, 0.01 or 0.001 mg 7,12-dimethylbenz(a)anthracene (Van Duuren et al., 1966). The mean survival times of treated groups were not shortened when compared with both vehicle and untreated control groups. In the low-dose glycidaldehyde group the incidence of malignant tumors was 3/50 (2 fibrosarcomas and 1 squamous cell carcinoma); the first tumor appeared during month 16 of the study. No benign tumors were reported. In the high-dose group there was 1 benign tumor; the incidence of malignant tumors was 7/30 (3 fibrosarcomas, 1 squamous cell carcinoma, 2 adenocarcinomas and 1 undifferentiated sarcoma). In the original study a statistical analysis was not performed. The apparent increased tumor incidence in the low-dose groups, when compared with the respective untreated concurrent control group, was not significant; the increase in the high-dose group, when compared with the respective untreated concurrent control group, was statistically significant. When malignant injection-site tumor incidence data from the two untreated control groups were combined (0/80), a dose-related trend test was statistically significant (Richards, 1991). A single incidence of adenocarcinoma of the breast was reported in an animal that received glycidaldehyde injections (dose not reported). Mice bearing tumors at distant sites were

observed in both the control and vehicle control groups. No injection site tumors were seen in either the vehicle control groups or the untreated control groups. No benign tumors were reported in the positive control groups; the incidences of malignant tumors were 9/30, 21/30 and 32/50 in the 0.001-, 0.01- and 0.1-mg 7,12-dimethylbenz(a)anthracene groups, respectively.

In a skin-painting assay, Van Duuren et al. (1965) treated the backs of 30 shaved female Swiss mice (10/cage) with a solution of 3% glycidaldehyde (purity not reported) in benzene (approximately 100 mg/application) 3 times weekly for life. A positive control group consisting of 30 female mice received a 0.2% solution of dibenz(a,h)anthracene in benzene; 2 negative control groups (60 female mice/group) either were untreated or received benzene alone. The median survival times were 496 days in the glycidaldehyde group, 498 days in the benzene control group, 441 days in the untreated control group and 313 days in the positive control group. Glycidaldehyde induced hair loss and crusting which persisted for at least 3 months; this condition recurred at least two times during the experiment.

There were statistically significant increases in the incidence of skin papillomas in the glycidaldehyde group and in the incidences of skin carcinomas in the glycidaldehyde and positive control groups (Rice and Wymer, 1991). The incidence of skin papillomas in the glycidaldehyde group was 8/30; the tumor incidences for the positive-, benzene- and untreated-control group were 1/30, 0/60 and 0/60, respectively. The incidences of skin carcinomas in the glycidaldehyde and the positive control groups were 8/30 and 23/30, respectively. In the glycidaldehyde group the first papilloma appeared on day 212 and the first carcinoma on day 338. In the positive control group papillomas and carcinomas both first appeared on day 112. No statistical analysis was reported.

Using the same protocol described above, Van Duuren et al. (1967a) applied glycidaldehyde (purity not reported) as a 10% solution in acetone (approximately 100 mg/application) 3 times/week to the skins of 41 female ICR/Ha Swiss mice for 598 days. Solvent- and untreated-control groups of 50 and 30 mice, respectively, were employed. A positive control group (40 mice) was treated 3 times/week with 0.1% dibenz[a,h]anthracene in acetone. No papillomas or malignant skin tumors developed in either the solvent- or the untreated-control groups. In the glycidaldehyde-treated group, papillomas developed in 6/41 mice and malignant skin tumors developed in 3/6 mice with papillomas (none in the other mice). The first papilloma appeared on day 154 and the first malignant skin tumor on day 308 of the study. In the positive control group the papilloma incidence was 39/40 and the incidence of malignant skin tumors was 32/40; the first papilloma appeared on day 132 and the first malignant tumor on day 177. The increased papilloma incidences in both the glycidaldehyde and the positive control groups were statistically significantly greater than the incidence in the solvent control group.

In an initiation-promotion experiment using conditions similar to the previous experiment, Van Duuren et al. (1965) treated the skins of 20 female Swiss-Millerton mice/group once with either 1 mg glycidaldehyde or 150 ug 7,12-dimethylbenz(a)anthracene (as a positive control). These initial treatments were followed 2 weeks later with applications of croton resin (25 ug/application, 3 times/week) for life. Two additional groups received only the initial treatment of either glycidaldehyde or 7,12-dimethylbenz(a)anthracene and a final group received only the croton resin treatments 3 times/week. The median survival times of the lifetime glycidaldehyde/croton resin-treated group and the 7,12-dimethylbenz(a)anthracene/croton resin group were 386 and 276 days, respectively. The median survival times of the groups treated only once with glycidaldehyde or 7,12-dimethylbenz(a)anthracene were 348 and 370 days, respectively. The median survival time of the group receiving only the croton resin treatments was 439 days. In the glycidaldehyde/croton resin treated group the papilloma incidence was 2/20; the first papilloma appeared on day 264. No other tumor types were reported. In the 7,12-dimethylbenz(a)anthracene/croton resin group, the papilloma incidence was 9/20; the first papilloma appeared on day 51. The carcinoma incidence was 8/20 and the first carcinoma appeared on day 110. Except for a single papilloma incidence appearing on day 426 in the group receiving only the croton resin treatments, no other tumors were reported.

In a tumor promotion assay Shamberger et al. (1974) applied 2.5 mg of glycidaldehyde in 0.25 mL of acetone once to the shaved backs of groups of 30 female Swiss mice. Starting 3 weeks later 0.1% croton oil in acetone was applied 5 days/week for 27 weeks. By week 30, glycidaldehyde and croton oil- treated mice had an incidence of 42% of skin tumors; however, the tumor incidences for acetone-treated and croton oil only-treated animals were not provided. No survival data were provided.

Groups of female Eastern Sprague-Dawley rats received either injections of 1 mg glycidaldehyde in 0.1 mL tricapyrylin (50 rats), injections of the solvent alone (40 rats) or no injections (30 rats). In the glycidaldehyde group an injection site tumor was reported in 1/50 rats. No injection site tumors were observed in the vehicle- and untreated-control groups. The median survival times were similar for the three groups (555 days) (Van Duuren et al., 1966). No statistical analysis was reported in the original study. Further statistical analysis showed that the apparent increase in tumor incidence was not statistically significant (Richards, 1991). In a follow-up study (Van Duuren et al., 1967b) groups of 20 female Eastern Sprague-Dawley rats received weekly injections of either 33 mg glycidaldehyde in tricapyrylin or tricapyrylin alone. Another group was not treated. Median survival times were similar. No injection site tumors occurred in the vehicle- and untreated-control groups; the injection site tumor incidence was 5/20 in the high-dose group. No statistical analysis was reported. Further analysis showed that the increased incidence in the 33-mg glycidaldehyde-treated group was statistically significant when compared with the vehicle control group. When data from the control group and 1-mg glycidaldehyde groups (Van Duuren

et al., 1966) were combined with this study there was a statistically significant positive dose-related trend (Richards, 1991).

Five female Sprague-Dawley rats, weighing 120-125 g, were given 33 mg glycidaldehyde (purity not reported) in 0.5 mL tricapyrin by gastric intubation once weekly for 70 weeks (Van Duuren et al., 1966). The rats were examined for palpable tumors on a regular basis and a complete autopsy was performed after sacrifice or natural death. Concurrent solvent controls received tricapyrin only. Glycidaldehyde did not induce gastric tumors or tumors at other sites. In a group of six female rats that received a single intragastric treatment of 50 mg of 7,12-dimethylbenz[a]anthracene as a concurrent positive control, two fibroadenomas, two mammary adenocarcinomas and one lymphoma were observed. No tumors were seen in rats receiving tricapyrin alone. This study is limited by the small group size.

II.A.4. Supporting Data for Carcinogenicity

Glycidaldehyde is mutagenic in many assay systems. Studies of reverse mutation with glycidaldehyde in *Salmonella typhimurium* in the presence or absence of activating systems gave positive results in strains TA1535 and TA100 (McCann et al., 1975; Rosenkranz and Leifer, 1980; Simmon, 1979a; Simmon et al., 1979; Wade et al., 1979). Glycidaldehyde was mutagenic in *Klebsiella pneumoniae* using the reverse mutation test (Knaap et al., 1982; Voogd et al., 1981); this compound was positive in *Escherichia coli* as judged by the DNA repair assay (Fluck et al., 1976; Rosenkranz and Leifer, 1980).

Simmon (1979b) found glycidaldehyde to be positive in *Saccharomyces cerevisiae* in the mitotic recombination assay and the plate incorporation technique; however, the same assay was negative when the intraperitoneal host assay method was used (Simmon et al., 1979). Glycidaldehyde was not mutagenic in mouse lymphoma cells at the HGPRT locus (Knaap et al., 1982), but was mutagenic at the thymidine kinase locus (Amacher and Turner, 1982). Glycidaldehyde was positive in Syrian hamster embryo cell systems in cell transformation assays (Dunkel et al., 1981; Pienta, 1980) and was also positive in a human fibroblast unscheduled DNA synthesis assay at concentrations of 0.1-100 µg/mL (Mitchell, 1976; Mitchell et al., 1983). In addition, glycidaldehyde was found to be mutagenic in *Drosophila melanogaster* as shown by the recessive lethal test (Knaap et al., 1982).

Glycidaldehyde is expected to be a highly reactive compound because of its bifunctionality (due to the presence of the epoxy and aldehyde groups on the molecule) (Van Duuren et al., 1963). When ingested, glycidaldehyde is likely to be hydrolyzed rapidly in the low-pH gastric environment (Van Duuren et al., 1966); however, quantitative data have not been located. In vivo, epoxides can be metabolized to dihydrodiols by epoxide hydratases or transformed into more excretable products by conjugation via glutathione-S-transferase (Manson, 1980). Manson

(1980), however, reported that small, highly reactive molecules such as glycidaldehyde are probably removed from the body through nonenzymatic means since they are not good substrates for epoxy hydratase. Glycidaldehyde has been shown to react with guanosine, one of the purine bases present in DNA, to form adducts that may alter the genetic integrity of the DNA molecule (Goldschmidt et al., 1968; Van Duuren and Loewengart, 1977; Nair and Turner, 1984; Golding et al., 1986; Bleasdale et al., 1986).

Epoxides related to glycidaldehyde have been shown to be carcinogenic in animals. Glycidol (the alcohol derivative of glycidaldehyde) has been shown to induce tumors in two different animal species. Propylene oxide (another related three-carbon epoxide) has also been shown to induce tumors in two different animal species and has also been classified as B2, probable human carcinogen, by the CRAVE Work Group.

II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

None.

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

None.

II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Source Document — U.S. EPA, 1989

The 1989 Health and Environmental Effects Document for Glycidaldehyde has received Agency Review.

II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review — 03/07/1991

Verification Date — 03/07/1991

A comprehensive review of toxicological studies published through May 2005 was conducted. No new health effects data were identified that would be directly useful in the revision of the existing carcinogenicity assessment for Glycidaldehyde and a change in the assessment is not warranted at this time. For more information, IRIS users may contact the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

III. [reserved]

IV. [reserved]

V. [reserved]

VI. Bibliography

Substance Name — Glycidaldehyde
CASRN — 765-34-4

VI.A. Oral RfD References

Hine, C.H., R.J. Guzman, M.K. Dunlap, R. Lima and G.S. Loquvam. 1961. Studies on the toxicity of glycidylaldehyde. Arch. Environ. Health. 2: 23-30.

VI.B. Inhalation RfC References

None

VI.C. Carcinogenicity Assessment References

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VII. Revision History

Substance Name — Glycidaldehyde
CASRN — 765-34-4

Date	Section	Description
08/22/1988	I.A.	Oral RfD summary on-line
08/01/1991	II.	Carcinogenicity assessment on-line

Date	Section	Description
12/03/2002	I.A.6., II.D.2.	Screening-Level Literature Review Findings message has been added.
06/22/2005	I.A.6., II.D.2.	Screening-Level Literature Review Findings message has been removed and replaced by comprehensive literature review conclusions.

VIII. Synonyms

Substance Name — Glycidaldehyde

CASRN — 765-34-4

Last Revised — 08/22/1988

- 765-34-4
- epihydrinaldehyde
- epihydrine aldehyde
- 2,3-epoxypropanal
- 2,3-epoxy-1-propanal
- 2,3-epoxypropionaldehyde
- formyloxiran
- glycidal
- glycidaldehyde
- Glycidyaldehyde
- glycidylaldehyde
- oxirane-carboxaldehyde
- propionaldehyde, 2,3-epoxy-
- RCRA waste number U126
- UN 2622