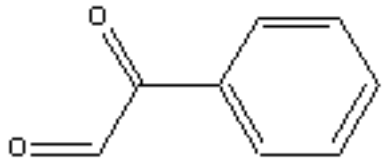
PHENYLGLYOXAL
CAS NO. 1074-12-0

Structure, Molecular Formula and Molecular Weight:



$C_8H_6O_2 \cdot H_2O$ Mol. wt.: 152.15

BASIS OF NOMINATION TO THE CSWG

PG is widely used as a reagent chemical in several industries, in academic institutions and in clinical laboratories. Therefore, the major potential for human exposures would be to industrial and biomedical research workers and students. Direct consumer exposure may result from the introduction of this chemical into food products as an antimicrobial agent or into food-contact products as a disinfectant. Positive short-term test results and the -keto aldehyde structural feature suggest that this chemical has a suspicion of carcinogenicity. PG is the prototypical arylketo aldehyde which could be interesting to study from a mechanistic standpoint.

SELECTION STATUS

ACTION BY CSWG: 12/16/94

Studies Requested: Carcinogenicity

Priority: Moderate

Rationale/Remarks:

- Potential for human exposure
- Widely used reagent in industry, academia and in clinical laboratories
- Proposed use as an antimicrobial agent could increase human exposure
- Positive genotoxicity test data
- Member of ketoaldehydes chemical class which has not been adequately tested for carcinogenicity

INPUT FROM GOVERNMENT AGENCIES/INDUSTRY

PG is undergoing evaluation by the U.S. Department of Agriculture (USDA) for potential commercialization as an antimicrobial additive for processed foods. One of the principal scientists, Dr. Bobby L. Bowles of the USDA's Agricultural Research Service, (Eastern Regional Research Center / Microbial Food Safety Research Unit in Philadelphia), informed the TRI staff that a patent application has been filed on the use of PG and some related chemicals, both singly and in combinations, as direct antibotulinal food additives and also for several indirect food additive uses, such as food-contact surface cleaning products and food wraps. Dr. Bowles and colleagues are currently seeking to pursue cooperative development agreements toward commercialization with corporate business entities.

SUMMARY OF DATA FOR CHEMICAL SELECTION

CHEMICAL IDENTIFICATION

CAS Registry No.:	1074-12-0
Chemical Abstracts Name:	Benzeneacetaldehyde, alpha-oxo- (9CI); glyoxal, phenyl (8CI)
Synonyms:	Benzoylcarboxaldehyde; benzoylformaldehyde; 1-phenyl-1,2-ethanedione; phenylethanedione; phenylglyoxal monohydrate; PG

Chemical and Physical Properties:

Description:	White to light yellow fine crystalline powder (Anon., 1994a; Anon., 1994b)
Boiling Point:	142°C at 125 mm Hg (Lide, 1993; Anon., 1994b)
Melting Point:	76-79°C (Anon., 1994b); 91 C (Lide, 1993)
Solubility:	Soluble in water (1:20 in hot water), acetone, benzene diethyl ether, ethanol (5% in 95% hot ethanol), and methanol (0.1 g. in 10 ml) (Anon., 1994a,c,d; HODOC, 1994; Lide, 1993)
Flash Point:	< 73.4oF (Sax & Lewis, 1989)
Stability:	Non-hydrated compound may undergo hydration, air oxidation or polymerization (Yang & Brush, 1993)
Reactivity:	Dangerous fire hazard when exposed to heat or flame (Anon., 1994c); can react vigorously with oxidizing materials. When heated to decomposition, it emits acrid smoke and irritating fumes (Sax & Lewis, 1989)

Technical Products and Impurities: -Ketoaldehydes are prone to hydration and are, therefore, typically supplied as the more stable aldehyde hydrate which must then be purified and standardized prior to use as an analytical reagent (Yang & Brush, 1993). Phenylglyoxal is commercially available as the monohydrate with purities ranging from 95% to 98% from numerous catalog suppliers, including the following: Sigma Chemical Co.; Aldrich Chemical Co.; TCI America Organic Chemicals; Chem Service Inc., Fluka Chemical Corp., and Pfaltz & Bauer. Fluka reports the presence of phenylglyoxylic acid as an impurity at < 1% (Anon., 1993).

EXPOSURE INFORMATION

Commercial Availability

Production and Producers: PG is reportedly synthesized by the selenium dioxide oxidation of acetophenone, a method originally published in 1943 and used to prepare reagent grade product by Yang and Brush (1993).

PG is listed on EPA's TSCA Inventory (STN, 1994). No annual production volumes were found for PG in the available literature. PG is not listed in the U.S. International Trade Commission (USITC) publication Synthetic Organic Chemicals, US Production and Sales (see Search Resource List). The USITC reporting guidelines specify that each company's report of a chemical represents manufacture of a quantity 4,500 kg [10,000 lbs] or sales of $10,000.

Principal producers listed in recent chemical industry directories are:

Hoechst Celanese Corp., Somerville, NJ
Janssen Chimica / Spectrum Chemical Mfg. Corp., New Brunswick, NJ
King's Laboratory, Inc., Blythwood, SC.

In addition, the following catalog producers / suppliers were listed in the DIALOG Fine Chemicals Database: Aldrich Chemical Co., Alfa Products, American Tokyo Kasei, Chem Service, Inc., Crescent Chemical Co., Inc., Fluka Chemical Corp., Lancaster Synthesis Ltd., Pfaltz & Bauer, Inc., and Sigma Chemical Co. ICN Biochemicals also produces this chemical.

According to recent patent literature, several companies manufacture PG for internal processing or R&D use, including Hoechst Celanese Corp., Allied Signal Inc., Rhone-Poulenc Rorer Ltd., Asahi Chemical Industry, Merck & Co., Inc., and Imperial Chemical Industries (Zeneca).

Use Pattern: PG is principally used as a biochemical reagent for the chemical modification of arginine and histidine residues in enzymes and other proteins and as a molecular probe (Janssen Chimica, 1992; Yang & Brush, 1993).

PG is also widely used as a starting material / chemical intermediate in organic syntheses and in pharmaceutical manufacture. Other types of products derived from PG as a starting material / chemical intermediate include agrochemicals (herbicides), electronic industry chemicals (nonlinear optical material), and fluorescent dyes. Some recent examples of reactions in which PG is used as a starting material were found in the chemical literature and include the following:

- PG can be used as an enophile to yield allylcarboxaldehydes when it undergoes tin tetrachloride induced ene reaction with olefins (Achmatowicz et al, 1986).

- PG can be used in a Knoevenagel condensation type reaction to produce epoxy-y-butyrolactams (Kim & Bjeldanes, 1992).

- In a study of experimental air pollution, PG was reported by Nojima and Isogami (1993) to act as an epoxidizing agent similar to nitrogen dioxide capable of reacting photochemically with aldrin to form the corresponding epoxide, dieldrin. A xenon lamp was the source of irradiation which promoted this photoepoxidation reaction.

Kojima et al. (1991) have reported the usefulness of PG as a bioanalytical fluorogenic reagent for the determination of tryptophan. They have developed improved analytical techniques for the quantitative fluorometric determination of tryptophan in human serum as a diagnostic tool in clinical medicine. Serum-samples are first treated with dilute perchloric acid and then with 0.5 ml of 60 mM PG in DMSO. The PG reagent is prepared as a shelf-stable reagent for short term storage at room temperature.

Two investigational uses of PG as a biochemical reagent have been reported in R&D citations. PG is one of several -dicarbonyls which have been recommended as an effective antimicrobial for food use. It was reported to be relatively comparable in activity to diacetyl; however, it was more effective against Gram-positive bacteria and less effective against fungi and Gram-negative bacteria than diacetyl (Jay et al., 1983). Vander Jagt (1975) found that PG effectively inhibited the growth of Escherichia coli K12 and yeast. Bowles and Miller (1993) of the Department of Agriculture have investigated the use of PG as a canned food protectant and inhibitor of spoilage. In a screening study of several aldehydes for effectiveness against Clostridium botulinum spores and cells, 0.06 mM PG delayed germination in botulinal assay medium (BAM) while 125 mM PG was active against vegetative cells. In canned chicken and beef broths, PG (5 mM) delayed C. botulinum neurotoxin production for 48 hr. at 32°C. According to Dr. Bowles, PG is potentially useful singly and in various combinations in a number of medical type research areas, including surface cleaning formulations and food wraps to enhance shelf life. In a patent issued in 1978 it was claimed that PG acted as an effective charge-controlling stabilizing agent for decreasing thrombogenicity on the surface of collagen vascular graft prostheses. By rendering the surface charge more negative as a result of reaction with arginine sites on the collagen helix, PG was reported to improve the collagen material in terms of controlling thrombogenicity in the treated vascular or valve prostheses (Sawyer, 1978).

Human Exposure: Phenylglyoxal is not listed in the National Occupational Exposure Survey (NOES) database which was conducted by NIOSH between 1981 and 1983 (NIOSH, 1990). There is potential for occupational exposure to this chemical in laboratory settings mainly by the oral or dermal route. Direct low-level consumer exposures could result from commercialization of PG as a food- or medical device-treating reagent.

Environmental Occurrence: PG has been identified as a naturally occurring growth stimulator which acts as a flower-inducing substance found in extracts of immature seeds of Pharbitis purpurea (Suzuki et al., 1988).

Concerned about the potential effects of the sun on cosmetics applied to the skin, Shibamoto and Umano (1985) studied irradiation of the common skin-care product ingredient, benzyl benzoate, by exposing the compound in ethanol solution to sunlight for 11 days. PG was identified as one of the products of the resulting photochemical reaction when 500 mg of benzyl benzoate with 10 mg of acetophenone as photosensitizer in 50 ml of ethanol was exposed to atmospheric oxygen and irradiated. [N.B. The authors attribute the presence of the irradiation products to a mechanism involving formation of the benzoyl radical; however, the TRI staff reporter thinks it possible that acetophenone may have been the source of PG by an oxidative process.]

Regulatory Status: No standards or guidelines for occupational exposures to or environmental levels of PG were found in a search of available literature.

EVIDENCE FOR POSSIBLE CARCINOGENIC ACTIVITY

Human Data: No epidemiological studies or case reports investigating the association of exposures to PG and a cancer risk in humans were identified in the available literature. PG is reported to be an irritant (Anon. 1994a).

Animal Data: No 2-year carcinogenicity studies of PG in animals were identified in the published literature. The Registry of Toxic Effects of Chemical Substances (RTECS) record for PG did not contain any general or acute toxicity data (NLM, 1994). However, Sugai et al. (1990) reported that PG ranks as a severe to corrosive eye irritant in rabbits.

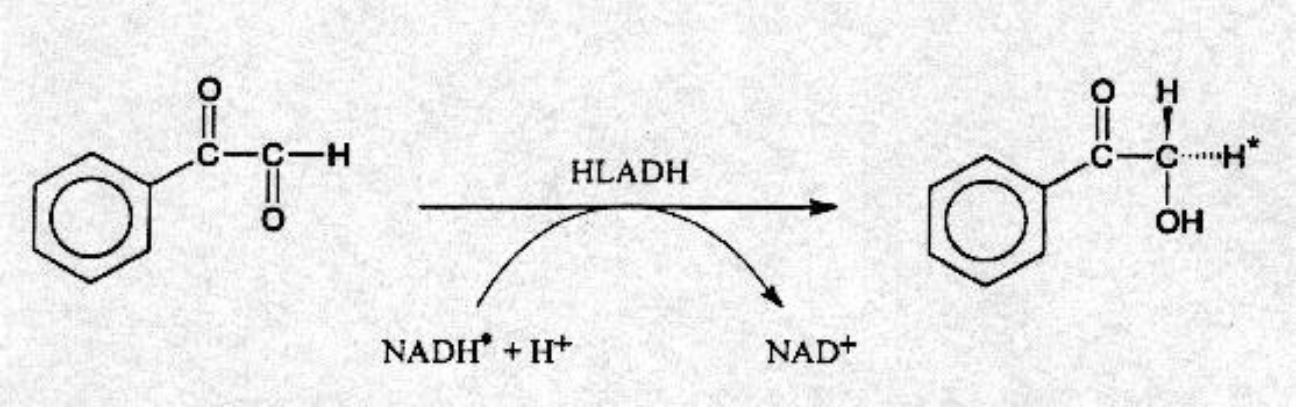
Short-Term Test: Several studies have documented test results associating PG with chemically induced genetic effects.

- NCI has reported that PG was mutagenic in the Ames/Salmonella assay in strains TA98 and TA100 with and without metabolic activation when tested in the Division of Cancer Etiology Short-Term Test Program (NCI, 1994).
- Dorado et al. (1992) tested PG as one of 9 analogous -dicarbonyl compounds in an Ames/Salmonella assay in TA100 without activation. PG was one of six which gave positive results with a relative mutagenic activity less than glyoxal and methylglyoxal but much greater than 1,2-cyclohexanedione, diacetyl and 3,4-hexanedione. This research group also evaluated reactivity parameters on these compounds and proposed a mechanism of mutagenicity based on adduct formation with puric bases (guanine and guanosine) (Rodriguez Mellado & Ruiz Montoya, 1994).
- According to Wagenheim and Bolcsfoldi (1988), PG was positive without S9 activation in a mouse lymphoma L5178Y thymidine kinase locus forward mutation assay. PG demonstrated increasing mutation frequencies of 143, 367 and 659 mutants/106 surviving cells relative to a control frequency of ~100 for concentrations of 1.580, 2.100, and 3.150 x 10-5 mol/L respectively.
- Rinkus et al. (1988) reported that PG produced a dose-dependent increase in DNA single strand breakage (SSB) in Chinese hamster ovary (AUXB1) cells. Co-treatment with ascorbate and hydrogen peroxide both enhanced the DNA damage caused by PG.
- Hellmer and Bolcsfoldi (1992) reported that PG was genotoxic in an E. coli K-12 uvrB/recA DNA repair host-mediated assay.
- Cornago et al. (1989) concluded that PG inhibited semiconservative DNA synthesis and potentiated unscheduled DNA synthesis (UDS) in TC-SV40 hamster cells when tested in a repair deficient strain without S9 activation at a concentration of 1.27 mmole/L.

Metabolism: PG as a substrate for the enzyme, horse liver alcohol dehydrogenase (HLADH), is reduced to -hydroxyacetophenone as shown in the scheme in Figure 1, according to Yang and Brush (1993). They reported that HLADH is protected from

deactivation by -ketoaldehydes by pretreatment with NADH, and that quantitation of PG and other -ketoaldehydes can be accomplished by monitoring the disappearance of NADH at 340 nm. Linear response was observed over a concentration range of 50 to 350 µM PG. -Ketoaldehydes have also been reported to be detoxified by glycolase I catalyzed conversion to -hydroxycarboxylic acids (Vander Jagt, 1975).

[original text displays: Figure 1. Proposed reaction stereochemistry for the reduction of phenylglyoxal by HLADH (Yang & Brush, 1993)--see original text filed in NTP Central Files]



Other Biological Effects: PG is an arginine-modifying agent which has been reported to exert an inhibitory effect on some enzymes. Numerous studies have been conducted on enzyme deactivations by PG in various systems. Shyamala and Daveluy (1982) studied the effect of PG on the mouse mammary cytoplasmic glucocorticoid receptor. PG was reported to both inhibit binding of 3(H)dexamethasone to the steroid-free receptor and displace bound 3(H)dexamethasone from the steroid-receptor complex. The authors suggested that inhibition by PG may be due to its ability to interact with the steroid binding site of the receptor.

Effects on membrane transport activities related to protein binding and enzyme modification have also been reported. For example, Mancini et al. (1992) studied the effect of PG on transport activity of the rat liver lysosomal membrane. They reported that 1 mM PG produced significant inactivation of glucuronic acid.

The potential use of PG as an antimicrobial food additive is based on its enzyme deactivating effect on food-spoiling microbes. PG is generally active in inhibiting anaerobic bacteria; strict anaerobes were more affected than facultative anaerobes (Bowles & Jay, 1993).

PG and several analogs were tested by Cornago et al. (1990) for radiosensitizing effect on survival in a TC-SV40/INO tumor cell line. PG demonstrated a moderate radiosensitizing effect under anaerobic conditions on hypoxic cells, but did not have a comparable effect under aerobic conditions. PG was considered the most promising of the glyoxylic compounds tested and may be potentially useful as a radiosensitizer in clinical radiotherapy.

When Trypanosoma cruzi Mg2+-stimulated ATPase was reacted with phenylglyoxal there was a rapid loss of enzymatic activity (Cataldi de Flombaum & Stoppani, 1982).

Structure/Activity Relationships: The literature was searched for mutagenic and carcinogenic data on the dicarbonyl compounds (dialdehydes, ketoaldehydes, vicinal diketones and aldehyde carboxylic acids) shown in Appendix A to ascertain the possibility of a structurally related basis for predicting bioactivity and potential for chronic health effects.

Dorado et al. (1992), determined the relative mutagenicity of several -dicarbonyl compounds in S. typhimurium strain TA100 without preincubation or the addition of S9. PG induced 278 rev/µmole in this system compared to 51 for glyoxal [Analog A] and 921 for methylglyoxal [B]; all -diketones studied induced lower levels of mutations. When corrected for the concentration of the active, unhydrated species in solution, the mutagenic activity of the -ketoaldehydes studied was much greater than that of the -diketones. The activity of glyoxal, methylglyoxal and PG decreased in the order of increasing molecular weight of the -keto substituent (3.7, 1.0, 0.11 ◊ 106 rev/µmole, respectively). In all cases, the mutagenic activity of the -ketoaldehydes was several orders of magnitude greater than that of the -diketones, dimethylglyoxal [C], 3,4-hexanedione [D], and 1,2-cyclohexanedione [E].

Rodriguez Mellado and Ruiz Montoya (1994) extended the work of Dorado et al. (1992) by relating mutagenic activity to the formation of adducts between the -dicarbonyl compounds and guanine and guanosine. The authors observed good correlation between mutagenic activity and both the equilibrium constant of adduct formation and the apparent enthalpy of the adduct formation reactions. The authors concluded that mutagenic activity is related to the extent or stability of adduct formation.

Glyoxal [A], methylglyoxal [B] and dimethylglyoxal [C] were reported by Yamaguchi and Nakagawa (1983) to be mutagenic in S. typhimurium strain TA100. The mutagenicity of all three compounds was reduced in the presence of S9, and to a lesser extent by catalase; the degree of inhibition of glyoxal and methylglyoxal mutagenicity was dependent on the concentration of S9. In addition, various superoxide, singlet oxygen, and hydroxy radical scavengers reduced methylglyoxal mutagenicity, although to a lesser extent than S9. Fifty percent degradation of calf thymus DNA was observed following incubation for 20 hours with 10 mM methyl glyoxal; 40% degradation was observed following incubation with 10 mM glyoxal.

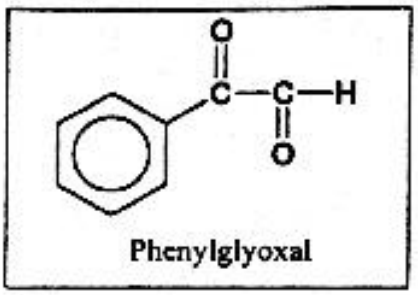
Wangenheim and Bolcsfoldi (1988) included glyoxal [A] and PG as part of a study assessing the mutagenicity of 50 compounds in the mouse lymphoma L5178Y assay without S9 activation. Five of the six aldehydes and ketoaldehydes, including PG, were mutagenic, several inducing a high number of mutants at low concentrations. Among them, formaldehyde and acetaldehyde are ranked as weak carcinogens, according to the authors. PG at a concentration of 31.5 µM (the highest concentration tested) induced 659 mutations / 106 surviving cells, compared to 300 mutations / 106 surviving cells for glyoxal at 479 µM. Greater inhibition of cellular growth was observed with PG than with glyoxal at these concentrations. The authors reported that, at the highest concentration tested, PG caused a 6.5-fold increase in mutants (expressed as mutation index which was defined as mutation frequency of treated culture / average mutation frequency of control cultures). A greater than 4-fold increase over controls was considered to be predictive of carcinogenicity.

The effects of five glyoxylic compounds (glyoxal [A], glyoxylic acid [F], PG, phenylglyoxylic acid [G], and difuranylglyoxal [H]) on semiconservative and unscheduled DNA synthesis (UDS) following 25 G X-irradiation was studied by Cornago et al. (1989). Under these conditions, all five compounds inhibited semiconservative synthesis and increased UDS in TC-SV40 hamster cells. The compounds inhibited semiconservative synthesis by about 30% without modifying the overall kinetics of DNA synthesis. UDS was stimulated to the greatest degree by phenylglyoxylic acid and glyoxal and to a lesser degree by glyoxylic acid, PG, and difuranylglyoxal.

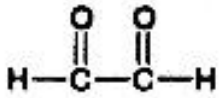
Cornago et al. (1990) studied the same five glyoxylic compounds as potential sensitizers in radiotherapy. PG was the most cytotoxic of the five compounds, reducing the survival of TC-SV40 cell by 50% at a concentration of 0.015 mM. None

of the compounds altered survival of irradiated cells under aerobic conditions while all moderately decreased survival in hypoxic cells.

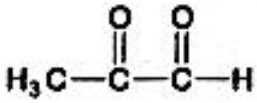
[original text displays Appendix A. Phenylglyoxal and structurally related compounds -- see original document in NTP Central Files]



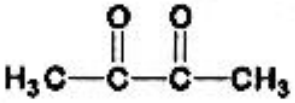
a) Glyoxal



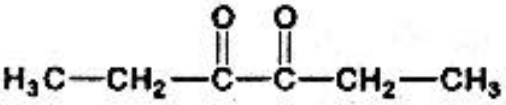
b) Methylglyoxal



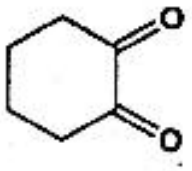
c) Dimethylglyoxal



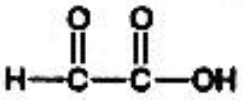
d) 3,4-Hexanedione



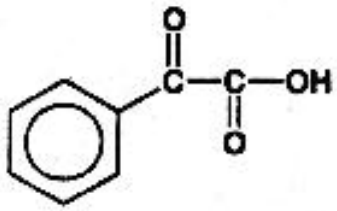
e) 1,2-Cyclohexanedione



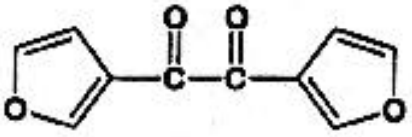
f) Glyoxylic acid



g) Phenylglyoxylic acid



h) Difuranylglyoxal



1074-12-0
Phenylglyoxal

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Summary Sheet Checklist for Phenylglyoxal (1074-12-0)

NLM	STN INTERNATIONAL	DIALOG
CCRIS	CA / CAOLD	Enviroline (40)
EMICBACK	CA Previews	Pollution Abstracts (41)
HSDB	CHEMLIST	CABA (50)
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