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An overview of a glyoxal: Production, Applications and Adverse effects

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Abstract

A 1% solution of glyoxal is the standard delivery form. The brigades of its organic emulsion react with polyfunctional composites, such as group or amino brigades, making it useful in cross-linking and condensation reactions. These reactions are analogous to those involving organic composites and their derivatives, such as bounce, cellulose, cotton, casein, or animal products. This is due to the high reactivity of its chemical component groups. Furthermore, outside its physiological activity, it also acts as an inhibitory agent in the process of weathering and aids in the solidification of rubber as well as the pigmentation of animal skin. It contributes to the synthesis of pigments, medicines, polymers, and textile reinforcements, among several other applications. Possible sources of commercially feasible glyoxal include the gas-phase reactivity of ethanediol with a metal or tableware catalyst, or the liquid-phase reactivity of ethanal with acid. Glyoxal can be produced from ethylene diol by gas phase processes. Glyoxal can be made from ethanediol, which is a powerful option that can be investigated anytime, especially in countries where ethanediol is produced in large quantities.

Keywords— glyoxal, pharmaceuticals, ethanediol, plastics, textile.

I. INTRODUCTION

The Fraunhofer Center for Toxicity and Experimental Medicine created the glyoxal Comprehensive International Chemical Assessment Documents (CICAD). The Expert Council on Known Substances of Environmental Importance papers serve as its foundation. To find any pertinent references released after those used in these reports, an extensive literature review of pertinent sources up to February 2003 was carried out. The original document's production and peer review process are discussed. This CICAD peer evaluation information is provided. At a conference of the Final Evaluation Board conducted from September 8–11, 2003, this CICAD was taken into consideration and authorised as a worldwide review. Presenting the attendees of the Final Evaluation Board gathering. The document also contains a replica of the Glyoxal International Chemical Safety Certificate (ICSC 1162), produced by the International Project on Chemical Safety (IPCS, 2002).

The freezing temperature of anhydrous glyoxal (CAS No. 107-22-2) is approximately 15 °C. However, it is usually offered as a liquid solution that contains hydrated oligomers and commonly contains 30 to 50 percent glyoxal. A variety of various polymers are produced using glyoxal as a cross-linking agent, a pesticide, a disinfectant, and a chemical intermediary in the manufacture of medicines and pigments. Emissions to outdoor air and water make up the majority of environmental releases.

Glyoxal fixatives credit their success to three characteristics in comparison to formalin: a superior safety profile, a quicker response rate, and a low propensity to crosslink under certain circumstances. Formalin's success can be attributed to its ability to crosslink. Because of this latter characteristic, glyoxal is preferable to other substances

when it comes to maintaining immunoreactivity and reducing the necessity of antigen retrieval. Herbicides and medicines are two other businesses that heavily utilize glyphosal because of the many benefits it provides in terms of transforming molecules without generating crosslinking. Glyoxal exposure at work occurs primarily through skin absorption and mist intake when it is used as a cleaner. The general public is primarily subjected through the consumption of foods containing glyoxal, but they may also be exposed through metropolitan air pollution and minute amounts of glyoxal in potable water.

Glyoxal is generated endogenously by multitudinous enzyme-dependent processes during typical cellular respiration. A vascular system has been established to hold a concentration of 0.1 - 1 micromole per liter of glyoxal, with those who have diabetes or renal insufficiency receiving lower levels of attention. Less than 10% of the glyoxal found in live organisms exists in its free and hydrated forms in a dry state. Because of its unique interaction with amino groups found in proteins, DNA, and lipids, glycoxal is thought to be an essential step in the production of advanced glycation endproducts. Cellular metabolism is disrupted, proteolysis is reduced, cell growth and protein production are inhibited, and AGE revision occurs. In doing so, it changes proteins and renders enzymes inactive. In order to reduce the negative consequences of the extremely reactive glyoxal, the glyoxalase pathway, which depends on glutathione (GSH), transforms it into the less reactive glycolate.

II. Materials and Methods

Acetaldehyde Conversion to Glyoxal by Nitric Acid: In a chromium steel autoclave kept at autogenous pressure, the oxidation of aldehyde or aldehyde by binary compound acid to glyoxal was the primary focus of the investigation. For the operation to take place, acid has to be present. Glyoxylic acid and ethanoic acid were the main ingredients. Various factors such as acid content, acid and aldehyde concentration, temperature, and reactivity quantity were investigated in order to determine ideal circumstances for glyoxal production.

Production from Benzene by ozonolysis: Glyoxal is produced by the ozonation of benzene in the presence of zinc and water (Zn/H2O).

The manufacture of glyoxal from glycol: Glycerine and an enzyme produced by the fungus Aspergillus versicolor make up the catalyst. Fungus mycelia produced glycerine enzyme when cultured in an environment with glycol as the sole carbon source. Degradation of byproduct oxide, which inhibits production of peroxide-dependent methanolate and glycerine enzyme deactivation, relies on enzyme activity as a co-catalyst. Aspergillus versicolor's original strain is known for its high glycerine enzyme and enzyme activity levels.

Alcohol Oxidase from Methanol Yeast: The enzyme glyoxal synthesizes ethylene glycol in this process. Glyoxal can be produced from ethylene glycol with the help of alcohol oxidases by first producing glycolaldehyde. Formaldehyde, acetaldehyde, propionaldehyde, and butyraldehyde are the end products of the alcohol oxidases' catabolism of n-propanol, butanol, methanol, and ethanol, respectively.

By using a silver catalyst and oxidative dehydrogenation: It helps to break down ethylene glycol, glyoxal is produced. Following a number of tests, it was discovered that using a silver spiral catalyst and a residual pressure of 400–600 millimetres produced the optimum reaction conditions. In the aforementioned container containing 7 milliliters of catalyst, a mixture of 40% liquid ethylene glycol gas (0.78 mole/h) and a 1:1 mixture of air and nitrogen was introduced. A total of 180 liters of oxygen were cycled every hour. The quantity of oxygen consumed was therefore 150% of what was recommended. Glyoxal (II) concentration in the vapour was 0.25 g/ml, or an output of 63.2%.

III. Result and Discussion

Metabolic and Comparative Kinetics in Experimental Animals and Humans

Endogenous glyoxal

The metabolism of glycolaldehyde, ethylene glycol, and β-hydroxy-substituted N-nitrosamines, along with microsomal oxidation, result in the production of glycoxal. These compounds may have more harmful effects, including toxicity, genotoxicity, and tumorigenicity, if it is present (Loeppky & Goelzer, 2002; Loeppky et al.,

2002). A majority of the reactive carbonyl groups form reversible bonds with cysteinyl, lysyl, and arginyl regions of proteins. Therefore, fewer than 10% of glyoxal in organic materials exists in a free state and undergoes hydration (Thornalley, 1995).Glyoxal, along with other α-oxoaldehydes, is well controlled in human tissues and bodily fluids by the efficient glyoxalase system (Thornalley, 1995) and its rapid reactivity with proteins (Sady et al., 2000).

Glyoxal concentrations increase in chronic conditions such as diabetes and uraemia. The blood samples from typical individuals (n = 19) had an average of 0.21 ± 0.14 μmol/kg of glyoxal, as reported by Thornalley et al. in 1996. The blood plasma value for normal healthy individuals is about 0.1 μmol/litre, whereas diabetics have a value that is twice as high (Thornalley, 1998; Thornalley et al., 2000).A separate research team consisting of 15 to 20 participants discovered that the plasma glyoxal levels in healthy individuals were measured at 67 μg/litre, whereas non-insulin-dependent diabetics had levels of 78 μg/litre, which is comparable to 1 μmol/litre. Glyoxal accumulation was seen in cases with severe renal failure, resulting in a mean plasma level of 221 μg/litre (about 4 μmol/litre). This is likely attributed to heightened glucose autoxidation in patients with uremia, as reported by Odani et al. in 1999. In individuals with diabetes and high blood sugar levels, the production of glyoxal, which is not natural to the body, may lead to the accumulation of this substance in certain areas (Akhand et al., 2001).

In pig heart tissue deprived of blood flow, the concentration of "free glyoxal" in the lipid fraction rose by a factor of 4 after 4 hours and by a factor of 24 after 6 hours (0.2 μg/g lipid), as compared to heart tissue with normal blood flow (Dudda et al., 1996).A total of 31.2 picomoles of glyoxal per 106 live P388D1 cells (a kind of murine macrophage cell) were grown. The concentration of glyoxal in the extracellular media increased from a level below the limit of detection to 61 nmol/litre after a 3-hour culture period ($P < 0.01$). The reference is from Abordo et al. in 1999.

Absorption, distribution, and excretion

The amount of systemic absorption was unclear, but acute and chronic breath inhalation had local effects on the eyes and pulmonary organs. When the medicine is taken orally, it is absorbed into the bloodstream and delivered to various organs such as the adrenal glands, erythrocytes, liver, lungs, kidneys, and pancreas, whether the dosage is acute or chronic. Section 8 provides more detail on this, and BUA (1997) backs it up. For instance, comparable results were also reported by Ueno et al. (1991a). Transdermal absorption of glioxal occurs. Blood glucose levels rise when the material is applied to the skin because the pancreas, liver, and kidneys break down granules and vesicles (Ito, 1963). The qualitative findings about skin irritation, as described in sections 8.7 and 9, provide evidence for the absorption of glyoxal via the skin.

HPLC research found 132 μmol/litre glyoxal in normal adult pee (Espinosa Mansilla et al., 1998). This could come from meal intake or endogenously.

Biotransformation

The main glyoxal detoxifying route is the cytosolic GSH-dependent glyoxalase system (see Figure 1). Glyoxal and GSH non-enzymatically form a hemithioacetal, which glyoxalase I converts to S-glycolylglutathione. Glyoxalase II hydrolyzes S-glycolylglutathione to glycolate, regenerating GSH. The cytosolic GSH content determines glyoxalase I activity in situ. Nevertheless, in conditions of oxidative stress, glyoxal may be metabolized by 2-oxoaldehyde dehydrogenase and aldose reductase, provided that the level of reduced glutathione (GSH) is very high. Intracellular imbalances in redox might hinder the processes of detoxification, resulting in elevated levels of glyoxal. Glyoxalase III functions in the detoxification process without relying on GSH. Escherichia coli is the highest producer of glyoxalase III, as shown by studies conducted by MacLean et al. in 1998 and Okada-Matsumoto & Fridovich in 2000.

Human tissues and blood cells had 0.2 μg/g glyoxalase I. Pancreas, lung, kidney, and brain had the maximum specific activity, while fatty tissue and liver had the lowest. Fetal tissues had three times more specific functions than adult tissues. A diallelic gene caused three variants in human glyoxalase I. GLO1 allele frequency in diverse groups averages 0.046 to 0.853. (Thornalley, 1993).

Effects On Laboratory Mammals And In Vitro Test Systems

Single exposure

Based on the concentration of glyoxal in the sample, the acute toxicity of the substance to the animals used for experiments ranges from negligible to moderate. It is not always obvious if the LC50 and LD50 values given in research articles relate to the chemical being tested at the given dose or have been adjusted to a concentration of 100% glyoxal. An enormous database on serious poisoning is included in the original article (BUA, 1997). Rats subjected to 40% glyoxal particles for 4 hours were found to have an LC50 value of 2440 mg/m3. As stated by Hoechst AG (1984b), the LC50 value was marginally lower for females (2410 mg/m3) and marginally higher for males (2470 mg/m3). When the maximal concentration of 1300 mg/m3 was utilized in the experiment, all ten mice exposed to an atmosphere comprising dust with 80% glyoxal survived (Hoechst AG, 1984c). After being exposed to 30% (Mellon Institute, 1958, 1965) or 40% (Mellon Institute, 1965) glyoxal for 7 or 8 hours, the rats did not die (Hoechst AG, 1984d,e). Localized ocular and respiratory irritation, increased blood flow, and foamy discharge in the airways were all symptoms of drug inhalation. There were no discernible changes to the rats' organs over the fourteen days of observation (Hoechst AG, 1984d,e).Researchers have found that female rats are more susceptible to the effects of goods containing 40% glyoxal. The LD50 values for male and female rats, respectively, range from 2960 mg/kg body weight (the lowest) to 8979 mg/kg body weight (the highest). When exposed to a 40% concentration of glyoxal, male and female rats had a median fatal dosage (LD50) of 4064 mg/kg. Food LD50 values for rats were determined to be 2000 mg/kg body weight and for guinea pigs to be 900 mg/kg body weight for a formulation that included 80% glyoxal. Itching and swelling in the GI tract, along with accumulation in the kidneys, adrenal glands, and lungs, are macroscopic symptoms that have been observed following oral administration (BUA, 1997).When 40% glyoxal was applied topically to rats, rabbits, and guinea pigs, the LD50 values exceeded 2,000 milligrams/kg body weight (for more details, refer to BUA, 1997).

New, in-depth studies on how naturally occurring glyoxal causes diabetes complications have confirmed histological results from studies done in the 1940s and 1960s using immediate injections of glyoxal (see section 8.8). The detrimental impact of glyoxal mostly affects the pancreas and kidney, leading to major progressive changes due to a deficiency in glyoxalase activity. Alloxan poisoning is a result of the presence of free radicals, and the pancreas is a primary organ that is affected (Younes, 1997).The administration of glyoxal in a dosage range of 100-200 mg per kilogram of body weight by intravenous injection in rats resulted in a reduction in blood glucose levels that was dependent on the dose, reversible, and could be replicated. The observed phenomenon was attributed to an increase in insulin secretion caused by the stimulation of glyoxal, which resulted in swelling and changes in the pancreas. Additional severe alterations, such as lasting tissue death and disintegration of B-cells, were seen with visible modifications in other bodily tissues at higher dosage (175 mg/kg body weight administered intravenously). Nevertheless, glyoxal's harmful impact was particularly evident in pancreatic B-cells (Helge, 1959). Administering 460 milligrammes of glyoxal to a cat by subcutaneous injection leads to nephrotoxicity, which is characterized by the destruction of vesicles in the kidney (Doerr, 1957a,b). When examining the different routes of administering glyoxal, it was consistently observed that intramuscular injection led to the rapid occurrence of acute effects in the pancreas. After applying a 40% glyoxal solution to the skin, severe necrotic dermatitis occurred at the application site in rabbits. Histopathological changes were seen in the liver, kidney, and pancreas 40 days later. The specific dosage of glyoxal used was not mentioned. Comparative examinations of tissue alterations in individuals with diabetes, such as the breakdown of granules and vesicles in the liver, kidney, and pancreas, as well as the reduction in size and alteration of fibers in the Langerhans islets, uncovered remarkable resemblances. The blood glucose levels in rabbits, which were evaluated for glucose tolerance 5 and 10 days after receiving glyoxal via the skin, were considerably elevated compared to the stable values seen in the control rabbits (Ito, 1963).

Short-term exposure

Following the OECD's advice, the low-dose group noticed no change, whereas the high-dose group significantly altered their eating habits and gained weight. Water consumption decreased in male rats given the lowest dosage and in female rats given the mid- and high doses (glyoxal concentrations were altered relative to water intake). Due to reduced water consumption, the mid- and high-dose groups showed an increase in cell count and urine volume, whereas the high-dose group saw changes in organ weight as a result of lower body mass. The

macroscopic and tissue investigations did not reveal any changes. Based on data reported by the Société Française Hoechst in 1987, the NOAEL (No-Observed-Adverse-Effect-Level) for this study is 100 mg of glyoxal per kilogram of body weight per day. Further data was not available to the CICAD authors. It is possible that these amounts are composed solely of glyoxal. As an alternative, the NOAEL might be set at 40 mg/kg body weight using a 100% glyoxal concentration.

Medium-term exposure

Ten male and ten female Wistar rats were given a 40% formulation of glyphosate for a period of ninety days. The study used male and female mice that were given different doses of 100% glyoxal daily: 32, 63, 125, and 250 mg/kg body weight. A transitory considerable delay in weight growth without a decrease in food intake occurred in high-dose males during the first two weeks of exposure. There was a statistically significant rise in the weight of the kidneys and liver in the high-dose group. There were no noticeable changes in the thoracic and pelvic organs, and the pancreas was not included in the examination. We did not test for biochemistry or hemology. According to the Montel Institute in 1966, the NOAEL for glyoxal was determined to be 125 mg/kg/day.

Significant reductions in food and drink consumption and delays in body weight increase were observed in the groups given mid- and high doses. Weight reduction, rather than decreased food intake, was the result of glyoxal's systemic effects, according to Part II of this research. At all times, the dosed groups experienced weight loss in the liver, kidneys, spleen, and heart. There was a marked increase in high-dose kidney weight at the 90-day mark. The breakdown of lipids was unchanged.

Glyoxalase I activity was markedly elevated in the kidneys, erythrocytes, and liver following 30 days of exposure to the medium and high doses, but this effect did not persist with longer exposure times. The levels of enzymes were shown to be lowered at all time periods evaluated after mid- and high-dose administrations of the drug. Alanine aminotransferase and total protein levels were drastically lower in the group given a small dose, making the formation of NOAEL impossible. According to Ueno et al. (1991a), the smallest amount of deleterious effects that rats exposed to 99% glyoxal for 90 days could tolerate was 107 mg/kg body weight daily. According to Ueno et al. (1991a) (see section 8.8), the decrease in blood protein levels after acute glyoxal exposure was due to a decrease in protein synthesis.

Phase II involved exposing five mice to 90 or 180 days of water with up to 6,000 mg of glyoxal per liter. Those in the diet-limited control group ate the same amount as those in the ad libitum group, while those in the former group were given unlimited access to food. Using glyoxal that was 98.7 percent pure, the dosages given for 90 days were 315 mg/kg body weight per day, and for 180 days, the dosage was 298 mg/kg body weight per day. Liver, kidney, spleen, stomach, thymus, and mesenteric lymph node gross and histological investigations were added to Phase I. The systemic toxicity of glyphosate sharply reduced the end-of-treatment body weight compared to the control group that received the identical quantity of food. Glyoxal significantly reduced the weights of the liver, kidneys, and heart in mice, according to a 1991a study by Ueno et al.

For 90 days, potable water with different concentrations of glyoxal (0, 1000, 2000, 4000, 8000, or 16,000 mg per liter) was given to a total of 10 Fischer 344 rats per dosage group and sex. Determining chronic study doses was the primary goal of this investigation. All animals given a high dosage were put to death on the twelfth day. Body and organ weights dropped, and food and drink consumption went down, even at the lowest dose. Due to their heightened sensitivity, male rats were shown to have an optimal chronic exposure dosage of 500-2000 mg/litre, whereas female rats had an optimal dosage of 1000-4000 mg/litre, resulting in a 46% reduction in water consumption. (The National Technical Panel, 1991a).

Over the course of 90 days, all B6C3F1 mice (ten males and ten females per group) were given water with varying amounts of glyoxal (0, 1000, 2000, 4000, 8000, or 16,000 mg glyoxal per liter). Throughout the duration of the research, every animal in every dosing group managed to stay alive. Weight loss of 7-30% was observed in the body and certain organs at doses ranging from 4000 to 16,000 mg/litre. There was also less water and less food eaten. Salivary duct changes, probably due to the chemical, led to a decrease in secretory function in the submandibular gland in male mice across all dosing groups. Water consumption, daily dosages, and feed intake were all reduced by 10–50% due to the unpleasant taste of the supplied water. According to this initial study, the

most effective dosage for long-term exposure trials in males (the more vulnerable gender) was 500-2000 mg/litre, which could reduce water consumption by up to 12%, and in females it was 1000-4000 mg/litre, which could reduce water consumption by up to 27%. According to NTP (1991b).

Long-term exposure and carcinogenicity

There was no problem with oral trials or long-term glyoxal breath.Sprague-Dawley rats were given 6,000 mg glyoxal/liter of water to consume for 180 days, and Ueno et al. (1991a) discovered no signs of cancer in their organs. The detailed findings are contained in Section 8.3.

For the initial therapy of two-stage glandular stomach cancer, male Wistar rats were given sodium chloride (10%) and 100 mg/liter of N-methyl-N'-nitro-N-nitrosoguanidine for 8 weeks. The results of this preliminary treatment showed that glioxal promoted tumor formation. From week 8 to week 40, rats given water containing a 0.5% dose of glyoxal showed statistically significant evidence of increased adenocarcinoma and pylorus hypertrophy. According to Takahashi et al. (1989), the pylorus did not undergo any changes when treated with glyoxal alone. The genotoxic activity, particularly strand breakage and unscheduled DNA synthesis, was observed in the rat stomach pyloric tissue, according to Furihata et al. (1985, 1989) and Furihata & Matsushima (1989) in section 8.5. After an injection of 150-400 mg/kg body weight of glyphoxal, the pyloric mucosa showed an increase in ornithine decarboxylase and replicative DNA synthesis. According to Furihata et al. (1985) and Furihata and Matsushima (1989, 1995), this increase not only promoted tumor growth but also varied with dose.

On the other hand, glyoxal was introduced to participants' drinking water for six weeks in a short liver foci assay. Glyoxal concentrations of 5000 and 2000 mg/litre were used in the experiment. The individuals were given a single intraperitoneal dosage of 200 mg/kg body weight of diethylnitrosamine before to exposure. At week 3, a partial hepatectomy was performed, and after a two-week recovery period, glyoxal exposure commenced. This analysis could not find any indication that it promoted tumor growth. Both Hasegawa and Ito (1992) and Hasegawa et al. (1995) found that compared to rats given the initiator, rats administered glyoxal exhibited a significant reduction in body weight, liver weight, water intake, and GST-P-positive foci in the liver.

After a lifetime of skin therapy with 3 µl of glyoxal (two commercially available products, 12.5% concentration in water) given three times weekly, C3H/HeJ mice did not develop any skin cancers. The glyoxal-treated mice outlived the untreated mice by a significant margin. Some of the treated rats showed signs of dead skin soreness (Bushy Run, 1982).No skin cancers were observed in CD-1 mice after 53 weeks of treatment with glyoxal alone at a total initial dosage of 30 mg/mouse, given in water at a concentration of 37-43% twice weekly for 5 weeks. Over the course of 47 weeks, two of ten rats treated to 12-O-tetradecanoyl-phorbol-13-acetate developed four skin papillomas. This supports the 1991 conclusion by Miyakawa et al. that glyoxal does not significantly stimulate tumor formation.

IV. Conclusion

Glyoxal has shown carcinogenic properties in both prokaryotes and eukaryotes during in vitro genotoxicity investigations. However, it has been found to be non-reactive in the mouse micronucleus test when administered orally. Evidence has shown that it is chemically inactive in studies involving the exchange of genetic material across chromosomes, the disappearance of sex chromosomes, the examination of harmful genetic traits, the evaluation of recessive genetic traits connected to sex, and the assessment of harmful genetic traits in Drosophila melanogaster. When given orally to rats, it causes an increase in pyloric mucosal irregular DNA synthesis but no change in main hepatocytes. The number of DNA single-strand breaks is significantly higher in the liver and the pyloric mucosa. Based on these results, it seems that glyoxal doesn't react in distant tissues, but it does in the stomach and a short time later in the liver, where it was introduced. Studies that used multiple dosages found no effect on sexual systems at doses up to approximately 300 mg/kg bw/d (related with the active component). In addition, the active ingredient allowed for the establishment of NOAELs for foetal development toxicity at 125 mg/kg bw/d and maternal toxicity at 25 mg/kg bw/d.
After cutaneous administration of glyoxal over the course of their complete lives, rodents show no evidence of a cancerous impact. After cutaneous delivery to rodents, glyoxal has no impact on the development of tumours.

Glyoxal shows local tumor-promoting characteristics in the rat forestomach epithelium after sublingual treatment (tissue not existing in species or man). There were no signs of glyoxal having a boosting impact on the liver in a rodent liver promotion model. Last but not least, glyoxal is a product of ethylene glycol, and ethylene glycol has been shown to be harmful in only two trials (rats and mice). It is necessary to do a comprehensive risk assessment that considers the skin irritation, skin sensitizing properties, genotoxic potential, and glyoxal use pattern. It is especially important to evaluate risks associated with contact with outdoor uses (like disinfectants).

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