化 学 品 安 全 技 术 说 明 书

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MSDS标题

KODAK EXTACOLOR PRIME DEVELOPER/REPLENISHE MSDS报告

产品标题

二乙基羟胺

CAS号

3710-84-7

化学品及企业标识

PRODUCT NAME

KODAK EXTACOLOR PRIME DEVELOPER/REPLENISHER/RA-4 PART A

NFPA

Flammability	1
Toxicity	2
Body Contact	3
Reactivity	1
Chronic	2

SCALE: Min/Nil=0 Low=1 Moderate=2 High=3 Extreme=4

PRODUCT USE

Used according to manufacturer' s directions. Part A of a three component photographic solution.

SYNONYMS

"CIN 10095681", "PCD 6202", P-0009.740, P-0009.740

CANADIAN WHMIS SYMBOLS

EMERGENCY OVERVIEW

RISK

Risk of serious damage to eyes.
Harmful by inhalation, in contact with skin and if swallowed.
Irritating to respiratory system and skin.

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. There is some evidence to suggest that this material can cause, if swallowed once, irreversible damage of organs.

EYE

If applied to the eyes, this material causes severe eye damage. This material can cause eye irritation and damage in some persons.

SKIN

Skin contact with the material may be harmful; systemic effects may resultfollowing absorption. This material can cause inflammation of the skin oncontact in some persons. The material may accentuate any pre-existing dermatitis condition. There is some evidence to suggest that this material, on a single contact with skin, can cause irreversible damage of organs. Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

INHALED

Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. The material can cause respiratory irritation in some persons. The body's response to such

irritation can cause further lung damage. There is some evidence to suggest that this material can cause, if inhaled once, irreversible damage of organs.

CHRONIC HEALTH EFFECTS

There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There is some evidence that inhaling this product is more likely to cause a sensitization reaction in some persons compared to the general population. There is limited evidence that, skin contact with this product is more likely to cause a sensitization reaction in some persons compared to the general population. Repeated exposure to hydroxylamine and derivatives may result in respiratory sensitization with asthma-like symptoms. Prolonged or chronic exposure to alkanolamines may result in liver, kidney or nervous system injury. Repeated inhalation may aggravate asthma and inflammatory or fibrotic pulmonary disease.Results of repeated exposure tests with diethanolamine (DEA) in laboratory animals include anaemia (rats) and effects on the kidneys (rats and mice) and liver (mice). DEA produces nervous system injury in dogs and rats. Heart and salivary gland lesions have also been seen in mice treated cutaneously with DEA and in mice receiving DEA in drinking water. Rats given high doses of DEA developed anaemia and testicular lesions.Exaggerated doses of DEA produced heart and nervous system effects in other animals. Changes in other organs were judged to be secondary due to the poor health of animals subjected to extremely high doses of DEA. Rats, rabbits and guinea pigs exposed to high vapour concentrations of volatile monoethanolamine (MEA) (up to 1250 ppm) for periods of up to 5 weeks developed pulmonary, hepatic and renal lesions. Dogs, rats and guinea pigs exposed to 100 ppm MEA for 30 days, became apathetic and developed poor appetites. Animal tests also indicate that inhalation exposure to MEA may result in nervous system injury. All species exposed to airborne MEA experienced dermal effects, varying from ulceration to hair loss probably resulting from contact with the cage.An increased incidence of skeletal variations, suggestive of a slight developmental delay was seen in the foetuses of rats given 1500 mg/kg/day DEA cutaneously; this also produced significant maternal toxicity. No foetal malformations, however, were seen in rats nor in rabbits receiving identical treatment. The foetus of rats given high doses of MEA by gavage, showed an increased rate of embryofoetal death, growth retardation, and some malformations including hydronephrosis and hydroureter. The high doses required to produce these effects bring into question the relevance of this finding to humans. There is some evidence that embryofoetotoxicity and teratogenicity does not occur in rats when MEA is administered by dermal application to the mother.The National Toxicology Program (NTP) concluded that there is clear evidence of liver tumours and some evidence of kidney tumours in mice exposed dermally to DEA over their lifetime. Chronic skin painting studies in mice of both sexes produced liver tumours and an increased incidence of kidney tumours in male mice. The significance of these findings to humans is unclear as DEA is neither genotoxic, mutagenic nor clastogenic, and did not induce tumours in rats or transgenic mice similarly treated. Alkanolamines (especially those containing

a secondary amine moiety) may react with nitrites or other nitrosating agents to form carcinogenic N- nitrosamines. Alkanolamines are metabolised by biosynthetic routes to ethanolamine and choline and incorporated into phospholipids. They are excreted predominantly unchanged with a half-life of approximately one week. In the absence of sodium nitrite, no conversion to carcinogenic N-nitrosamines was observed.Diethanolamine competitively inhibits the cellular uptake of choline, in vitro, and hepatic changes in choline homeostasis, consistent with choline deficiency, are observed in vivo.Many amines are potent skin and respiratory sensitisers and certain individuals especially those described as "atopic" (i.e. those predisposed to asthma and other allergic responses) may show allergic reactions when chronically exposed to alkanolamines.In a study with coconut diethanolamide, the National Toxicology Program (Technical Report Series 479), showed clear evidence of carcinogenic activity in male B6C3F1 mice based on increased incidences of hepatic and renal tubule neoplasms and in female B6C3F1 mice based on increased incidences of hepatic neoplasms. There was equivocal evidence of carcinogenic activity in female F344/N rats based on a marginal increase in the incidence of renal tube neoplasms. These increases were associated with the concentration of free diethanolamine present as a contaminant in the diethanolamine condensate. Exposure to rats to coconut oil diethanolamine condensate by dermal application in ethanol for 2 years resulted in epidermal hyperplasia, sebaceous gland hyperplasia, hyperkeratosis and parakeratosis in males and females and ulcer in females at the site of application. There were increases in the incidences of chronic inflammation, epithelial hyperplasia, and epithelial ulcer in the forestomach of female rats. The severity of nephropathy in dosed female rats were increased. Exposure of mice to coconut oil diethanolamine condensate by dermal application for 2 years resulted in increased incidences of eosinophilic foci of the liver in males. Increased incidences of epidermal hyperplasia, sebaceous gland hyperplasia, and hyperkeratosis in males and females, ulcer in males, and parakeratosis and inflammation in females at the site of application and of follicular cell hyperplasia in the thyroid gland of males and females, were chemical related.