Vitamin E protects against acetone-induced oxidative stress in rat red blood cells

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Abstract

Acetone may induce oxidative stress leading to disturbance of the biochemical and physiological functions of red blood cells (RBCs) thereby affecting membrane integrity. Vitamin E (vit E) is believed to function as an antioxidant in vivo protecting membranes from lipid peroxidation. The aim of the present study was the evaluation of possible protective effects of vit E treatment against acetone-induced oxidative stress in rat RBCs. Thirty healthy male Wistar albino rats, weighing 200–230 g and averaging 12 weeks old were randomly allotted into one of three experimental groups: Control (A), acetone-treated (B) and acetone + vit E-treated groups (C), each containing ten animals. Group A received only drinking water. Acetone, 5% (v/v), was given with drinking water to B and C groups. In addition, C group received vit E dose of 200 mg/kg/day i.m. The experiment continued for 10 days. At the end of the 10th day, the blood samples were obtained for biochemical and morphological investigation. Acetone treatment resulted in RBC membrane destruction and hemolysis, increased thiobarbituric acid reactive substance (TBARS) levels in plasma and RBC, and decreased RBC vit E levels. Vit E treatment decreased elevated TBARS levels in plasma and RBC and also increased reduced RBC vit E levels, and prevented RBC membrane destruction and hemolysis. In conclusion, vit E treatment appears to be beneficial in preventing acetone-induced oxidative RBC damage, and therefore, it can improve RBC rheology.

Introduction

Acetone is widely used as an industrial solvent and chemical intermediate. Because of its volatility and solubility, acetone can be found in the atmosphere, in natural water bodies and it can be released to groundwater as a result of leaching from municipal and industrial landfills (EPA, 1985, Churchill et al., 2001). Exposure to acetone occurs from both natural and anthropogenic sources, and it is endogenously produced by all humans. Dermal exposure to acetone may result from skin contact with consumer products (e.g., certain nail polish removers, paint removers, cleaning agents and various pharmaceutical products) (Morgott, 1993; EPA, 1995). Current smoking and daily alcohol intake were associated with elevated...
acetone levels (Churchill et al., 2001). Its ingestion may also happen either accidentally or intentionally, mostly as a tool for committing suicide. There are several case reports in the literature about acetone poisoning in man (Gamis and Wasserman, 1988; Kostusiak et al., 2003). Acetone is one of three ketone bodies that occur naturally throughout the body. Fasting, diabetes mellitus and strenuous exercise increase endogenous generation of acetone (Lebovitz, 1995; Kalapos, 2003). It has been suggested that acetone as a lipophilic compound has the ability to pass through plasma membranes and to disturb metabolism at cellular level (Saracino et al., 1980). The metabolism of acetone has been studied extensively in laboratory animals, primarily in rats. Casazza et al. (1984) postulated two pathways by which acetol from acetone was converted to glucose; the methylglyoxal and the propapane- diol pathways. Methylglyoxal has been implicated with production of reactive oxygen species (Kalapos, 1999).

Acetone pretreatment has been shown to potentiate halogenated solvent hepatotoxicity and nephrotoxicity, and is related to the induction of microsomal enzymes that metabolize these solvents to reactive intermediates (Morgott, 1993). Systemically, acetone is moderately toxic to the liver and produces hematological effects. The mechanism by which acetone produces these effects is unknown. It is thought that this toxic effect of acetone can be related to decrease of cellular detoxification capacity or increase of generation of reactive intermediates (Hewitt et al., 1987). Red blood cell membrane is rich in polyunsaturated fatty acids which are very susceptible to free radical mediated peroxidation. Eventually hemolysis is induced by membrane lipid peroxidation (Niki et al., 1988). Lipid peroxidation is associated with a wide variety of toxicological effects, including decreased membrane fluidity and function, impaired mitochondrial and golgi apparatus functions, and inhibition of enzymes. Assesment of TBARS is probably the most commonly applied method for the measurement of lipid peroxidation (Armutcu et al., 2004). Vitamin E (α-tocopherol) is believed to be involved in a variety of physiological and biochemical functions. It is the primary liposoluble antioxidant, which may have an important role in scavenging free oxygen radicals and in stabilizing the cell membranes, thus maintaining its permeability (Navarro et al., 1999; Wang and Quinn, 1999).

The aim of this study was to prove the hypothesis that vitamin E treatment protects the rat red blood cell (RBC) against acetone-induced oxidative damage. We also studied some hematological parameters in the blood of rats treated with acetone.

Materials and methods

Design and treatment

Thirty healthy male Wistar albino rats, weighing 200–230 g and averaging 12 weeks old were randomly allotted into one of three experimental groups: Control (A), acetone-treated (B) and acetone+vitamin E-treated groups (C), each containing ten animals. Animals were housed in stainless cages and kept in a room maintained at 22 ± 3°C with 12 h light/dark cycle, relative humidity 55%. On the 1st day of initiation of treatment they received an aqueous acetone solution (5%; v/v) ad libitum as sole drinking fluid for 10 days (Dietz, 1991). The animals were given standard rat pellets (Murat Animal Food Product Co., Ankara, Turkey). Group A received only drinking water. Acetone, 5% (v/v), was given with drinking water to B and C groups. In addition, C group received vitamin E as α-tocopherol acetate (Ephynal®, Roche) dose of 200 mg/kg per day i.m. The experiment continued for 10 days. At the end of the 10th day, the blood samples were obtained for biochemical and histopathologi-