



Interactive effects of ethanol on ulcerative colitis and its associated testicular dysfunction in pubertal BALB/c mice



Isaac A. Adedara^{*}, Babajide O. Ajayi, Ifeoluwa O. Awogbindin, Ebenezer O. Farombi

Drug Metabolism and Toxicology Research Laboratories, Department of Biochemistry, College of Medicine, University of Ibadan, Ibadan, Nigeria

ARTICLE INFO

Article history:

Received 23 February 2017

Received in revised form

2 May 2017

Accepted 8 June 2017

Keywords:

Colitis

Ethanol

Testicular dysfunction

Spermatogenesis

Mice

ABSTRACT

Available epidemiological reports have indicated an increase in the incidence of ulcerative colitis, as well as alcohol consumption, globally. The present study investigated the possible interactive effects of ethanol consumption on ulcerative colitis and its associated testicular dysfunction using six groups of 12 pubertal mice each. Group I (Control) mice received drinking water alone. Group II mice received ethanol alone at 5 g/kg body weight. Group III mice received 2.5% dextran sulphate sodium (DSS) in drinking water followed by normal drinking water. Groups IV, V, and VI mice received DSS followed by ethanol at 1.25, 2.5, and 5 g/kg, respectively. Administration of ethanol to mice with ulcerative colitis intensified the disease-activity index with marked reduction in colon length, colon mass index, body weight gain, and organo-somatic indices of testes and epididymis when compared with the DSS-alone group. Moreover, ethanol exacerbated colitis-mediated decrease in enzymatic and non-enzymatic antioxidants but increased the oxidative stress and inflammatory biomarkers in the testes and epididymis. The diminution in luteinizing hormone, follicle stimulating hormone, and testosterone levels was intensified following administration of ethanol to mice with ulcerative colitis that were administered 5 g/kg ethanol alone. The decrease in sperm functional parameters and testicular spermatogenic indices as well as histopathological damage in colon, testes, and epididymis was aggravated following administration of ethanol to mice with ulcerative colitis. In conclusion, the exacerbating effects of ethanol on ulcerative colitis-induced testicular dysfunction are related to increased oxidative stress and inflammation in the treated mice.

© 2017 Elsevier Inc. All rights reserved.

Introduction

The World Health Organization (WHO) indicated that alcohol consumption is the fourth leading risk factor for death and disability globally, almost at par with tobacco (World Health Organization, 2002). Indeed, the most important and abundant alcohol in alcoholic beverages responsible for its characteristic taste is ethanol, which is produced during fruit or grain fermentation processes. The long-established bactericidal effects of alcohol-containing wine have been recently linked to a positive modulatory effect on inflammation and immune responses, especially at low concentrations (Biasi et al., 2014). For instance, the reduction in stool calprotectin in patients with ulcerative colitis has been related to the inhibitory effects of alcohol on the systemic immune system and neutrophil migration in ulcerative colitis. However, alcohol

could be deleterious to weak areas of the intestine, which consequently are more susceptible to the adverse effects of alcohol (Swanson, Tieu, Shaikh, Forsyth, & Keshavarzian, 2011).

Epidemiological studies showed that the incidence of ulcerative colitis has been on the increase worldwide over the past few decades just as predilection for alcohol is globally burgeoning (Bulloch, Williams, Lavorato, & Patten, 2016; Ungaro, Mehandru, Allen, Peyrin-Biroulet, & Colombel, 2016). A previous epidemiological report showed that alcohol consumption might lower ulcerative colitis incidence (Boyko, Perera, Koepsell, Keane, & Inui, 1989). The mechanism of alcohol-induced intestinal dysmotility has been related mainly to its influence on brain-gut axis neurotransmission and on the muscular layers. Furthermore, high intake of ethanol induces persistent oxidative and inflammatory reactions, which may contribute to motility disorders and mucosal injury, which are considered to be high-risk factors for colorectal cancer development (Siegmund, Spanagel, & Singer, 2003; Stermer, 2002). Additionally, young patients with inflammatory bowel disease

^{*} Corresponding author.

E-mail address: dedac2001@yahoo.co.uk (I.A. Adedara).