RESEARCH ARTICLE

TOXICOLOGICAL IMPLICATION OF CONTAMINATED SOFT DRINK ON THE ACTIVITIES OF LACTATE DEHYDROGENASE (LDH) OF RAT LIVER AND KIDNEY TISSUES

SANNI Momoh^{*1}, EJEMBI Daniel¹, ABBAH Okpachi. C¹, Francis Onakpa ATANU¹, EMMANUEL T. Friday², Isaac Eleojo SHAIBU¹

- 1. Department of Biochemistry, Kogi State University. Anyigba, Nigeria.
- 2. Department of Medical Biochemistry, Kogi State University, Anyigba, Nigeria.

Correspondence author: SANNI Momoh, + 234-803965787

ABSTRACT

The present study aimed at assessing the toxicological implication of consuming experimentally contaminated soft drink on lactate dehydrogenase (LDH) activities of rat liver and kidney. Rats were fed with contaminated soft drink by gavage for a maximum of eleven days. Homogenised liver and kidney tissues were analysed for LDH activities spectrophotometrically. LDH levels decreased significantly below control values of 4.2 x 10^2 and 12.5 x 10^2 µm/min/mg protein for liver and kidney respectively. This study concluded that contaminated soft drink posed toxicity to the liver and kidney as presented by the alteration in LDH activities.

Keywords: Soft drink, Lactate dehydrogenase, Toxicity, Liver, Kidney, Rat.

INTRODUCTION

Soft drinks are most popular carbonated drink originally designed as medicine World Record, (Guiness 2006). Its popularity is displayed in high level of consumption accounting for an average of ten percent of the energy intake of teenagers. Though hygienically prepared and preserved, but prone to contamination when exposed to air and other external conditions which mav in turn have negative implications on the catalytic activities of some enzymes in vital organs. Enzymes as biological proteins are integral part of life, very sensitive and specific in their actions. Lactate dehydrogenase LDH (E.C.1.1.1.27) is

a cytoplasmic and marker enzyme present in a wide variety of organisms, including plants and animals. In animals LDH is found in many body tissues, including the liver, kidneys, serum and other biological fluids. Elevated levels of LDH may indicate liver and other tissue damage (Wilkinson, 1976). LDH catalyses the irreversible oxidation of Lactate to pyruvate, a reaction mediated by NADH as co-enzyme. The importance of this reaction is represented as disruption of the bioenergetic mechanisms for energy production when LDH levels are altered. need to investigate Hence, the the toxicological implication of the

contaminated soft drinks becomes imperative. The aim of the present work is to determine the effect(s) of contaminated soft drinks on liver and kidney lactate dehydrogenase in experimental rats.

MATERIALS AND METHODS

Materials

Bottles of soft drinks were procured from commercial shops in Anyigba, Nigeria, adult healthy albino rats were obtained from the Department of Biochemistry, University of Nigeria, Nsukka, Nigeria. Reduced Nicotinamide adenine dinucleotide (NADH), 2-oxoglutarate, pyruvic acid (sodium salt), p-nitrophenyl phosphate (disodium salt) were from Sigma Company, St. Louis Mo

U.S.A. All other reagents used were of analytical grade.

METHODS

The cork of these soft drinks were opened and poured in a larger container and exposed to air for 15 days to enhance its decarbonation, fermentation and chance of contamination from the atmosphere.

Pre-acclimatized albino rats weighing about 160±2.5 grams were divided into six groups with six rats in each group. Group, A, received distilled water and rat feed (Vital feeds, Jos, Plateau State, Nigeria) ad libitum all through the experimental period. Groups B-F received 4mls of contaminated soft drink corresponding to a dose of 25mg/kg body weight, on daily administration for 2,4,6,8 and 10 days respectively. Rats were sacrificed after 2,4,6,8 and 10 days post administration exercise. Liver and kidneys were quickly excised into ice-cold sucrose solution (0.25M), trimmed of extra tissues and homogenised in 0.25M sucrose and Triton X-100 in a ratio of 1:5 w/v and stored overnight at 0°C to equilibrate.

Lactate Dehydrogenase Activity

LDH was assayed for by the method of Wrobleski and LaDue, (1955). It involves the irreversible reduction of pyruvate to lactate using reduced nicotine adenine dinucleotide (NADH) as co-enzyme. Enzyme activity measured as function of loss of absorbance was at 400nm monitored over 5 minutes.

Protein estimation

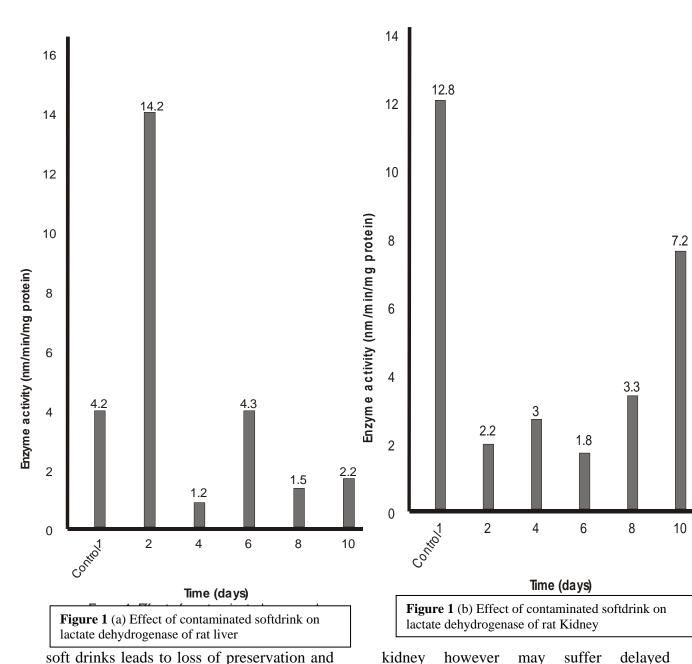
Protein content of the tissues studied was estimated by the Biuret method using Bovine serum albumin as standard (*Plummer*, 1978).

Statistical analysis

The results were expressed as means of six determinations. Significance was accepted at p<0.05.

RESULTS AND DISCUSSION

Lactate dehydrogenase is a biomarker of cell damage. It has been utilised in several experiments to deduce injury to the heart, kidney, liver etc (Wilkinson, 1976). Overall, the enzyme participates in post glycolytic metabolism in the conversion of lactate to pyruvate, a reaction whose inhibition leads to accumulation of lactate is associated with reduced tissue excitability. Relatively, increased activities of the LDH may denote an increased expression of its proteins. However, since this study does not take into full account molecular implication of contaminated soft drink consumption, it may suffice to say that LDH is either merely activated by the enzyme or as a result of increased secretion as an adaptive response. Irrespective of this parallel explanations, increases in LDH activities would likely lead to a deficit in oxidative metabolism and its attendant perturbations. Decarbonation of



soft drinks leads to loss of preservation and eventual invasion by microbes (*Mikkleson* and Mikkleson, 2004). Since the soft drink may serve as a source of nutrition, it is conceivable that waste products of invasive microorganisms could be toxic at its least. More so, increase in activities of this enzyme in the liver in contrast to the decrease in the kidney witnessed on second day of administration clearly reflects the role of the liver in xenobiotic metabolism due to its rich content of cytochrome P450. The

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damages reflected in the suppression of

LDH all through the experimental period.

Overall, the enzyme though irregular reveals that the liver tries to adjust to damages

decreases in activities of the enzyme

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