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The effect of a preservative on some physiological parameters in a sample of male albino rats

Bandr Khalid Hamzi[1] and Mohamed Mostafa Soliman[1*]
1- Department of Biology, Faculty of Science, Jazan University, Saudi Arabia.
* E.mail: msoliman@jazanu.edu.sa

Abstract

Sodium benzoate (SB), with antibacterial and antifungal characteristics, is a commonly employed preservative in both food and pharmaceuticals. According to some studies, SB might be hazardous. This study was done to investigate the adverse effects of oral administration of sodium benzoate on some physiological parameters. In this investigation, fifteen male rats were used. They were divided into three equal main groups: one received saline as the control group; the other two groups got oral SB treatment every day for three weeks at dosages of 300 and 500 mg/kg. At the end of the study, animals were sacrificed. Blood and liver samples were collected. Blood indices, glucose, renal profile (creatine, urea, and uric acid), liver enzymes (AST, ALT and ALP), lipid profile (TC, TG, LDL, HDL, vLDL), GSH and MDA were analyzed in all groups. The findings demonstrated that administration of SB significantly raised ALP, ALT, AST, creatinine, urea, glucose, TC, TG, LDL, vLDL and MDA. Furthermore, the high dose of sodium benzoate resulted in a significant decline in RBC count, hemoglobin concentration, hematocrit values, HDL and GSH. These results imply that the preservative sodium benzoate has adverse effects to the liver, kidneys, and possibly even some hematological parameters.

Key words: Sodium benzoate, liver enzymes, kidney, lipids, oxidation

1. INTRODUCTION:

All substances used in the manufacture, processing, packaging, shipping, or storage of food can be referred to as "additives" [1]. Preservatives have been used due to the existence of microorganisms that lead to the decomposition of food and unpleasant changes. Preservatives slow or completely stop the emergence of unfavorable changes in food through decreasing the growth of these microbes [2,3]. Sodium benzoate (SB) is a commonly used preservative that inhibits bacterial growth, mold, and yeast, especially in foods with a high level of acidity such as fruit juices and carbonated beverages. In addition to, jams, salted margarine, olives, syrups, sauces, relishes, pie fillings, fruit salads, prepared salads, and vegetable storage all include SB. Furthermore, it is used for preservation in cosmetics, mouthwashes, and pharmaceutical products [4,5].

Because of its well-known detrimental effects on food items, the use of SB should be restricted [6]. However, SB and ascorbic acid interact to generate benzene, which is a known carcinogen. Additionally, according to some studies, SB may convert into benzene, which destroys mitochondrial DNA [7,8]. In other studies, using animal models, SB was administered for a brief period, and the enzymes alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) were significantly elevated in serum [9]. Pathological alterations in the liver were seen after SB administration. These changes included loss of the majority of the tissue's

structural organization, hepatocytes were entirely damaged, some cells died, and others decomposed with fatty alterations as seen by the presence of fatty droplets in the cytoplasm [10]. Another study [11] found that SB administration to white mice caused significant hepatocyte vacuolization, loss of nucleation at some places, increased mitochondrial activity, shrinkage of the plasma membrane, and the development of finger-like extensions. Additionally, mice with brief SB exposure have worse memory and more oxidative damage [12,13].

Aim of the study

The present study aims to demonstrate the adverse effects of oral administration of sodium benzoate on some physiological parameters in a sample of male albino rats.

2. MATERIALS AND METHODS

2.1. Animal groups and experimental design.

For this investigation, animal models consisting of 15 mature male albino rats with initial body weights ranging from 150 to 200 g were used. Rats were purchased from the Deanship of Scientific Research's animal house section at the University of Jazan in Saudi Arabia. The animals were fed a balanced diet, and water was provided freely in hygienic containers. The rats were kept in their housing at a constant 24°C room temperature and on a 12h light: 12h dark cycle throughout the experiment. After seven days of acclimatization with free access to food and water, rats were randomly divided into three groups:

Group I: healthy control group (HC, n = 5), fed a standard diet during the experiment, got only saline.

Group II: SB1 treated group (SB1, n=5), oral administered with SB at a concentration 300 mg/kg.

Group III: SB2 treated group (SB2, n=5), oral administered with SB at a concentration 500 mg /kg.

Double-distilled water was used to prepare the various sodium benzoate concentrations, and the animals received the treatments through gastric intubation via the intragastric route once with experiment duration about three weeks.

2.2. Sampling preparation:

At the end of the experiment, each animal was anaesthetized with diethyl ether by placing the rat in an anesthetic box filled with ether vapor which was maintained by periodically applying liquid ether to a cotton wool on the base of the box. Once the animal was anesthetized (judged by loss of withdrawal reflexes), Sharp blades were used to sacrifice the animal through the external jugular vein. Two tubes were used to collect the blood: one EDTA tube for CBC analysis and the other a dry clean centrifuge tube to collect serum. After collection of the blood in a clean centrifuge tube, the blood was allowed to clot by leaving it undisturbed at room temperature for about 15 - 30 minutes (or in incubator at 37°C). The tubes were rapidly set to centrifuge at 5000 r.p.m for 15 minutes, the supernatant serum was sucked out into clean eppendorf tubes for biochemical measures. If the serum is not analyzed immediately, the serum should be stored at –20°C or lower. The livers were quickly extracted and cleaned in a saline. In phosphate buffer (5 mL cold phosphate buffer 50 mM, pH 7.4; per gram tissue), 1.0 g of liver samples was homogenized. The hepatic supernatants were isolated and refrigerated at 20 °C for additional biochemical research.

2.3. Biochemical analysis:

The following biochemical parameters were assayed in the serum utilizing Siemens Healthineers' Dimension RxL Max: alkaline phosphatase (ALP), alanine amino transferase (ALT), aspartate amino transferase (AST), creatinine, urea, uric acid, albumin, glucose, total cholesterol (TC), triglycerides (TG), low density lipoprotein (LDL-C) and high density lipoprotein (HDL-C). Hepatic oxidative stress markers (reduced glutathione; GSH and malondialdehyde; MDA) were assayed using commercial diagnostic kits (Bio-diagnostic company, Giza, Egypt).

2.4. Hematological analysis:

RBCs, WBCs count, hemoglobin conc. (Hb), and PCV were assayed in blood. The analysis was estimated by using Sysmex XP-300™ Automated Hematology Analyzer

2.5. Statistical analysis

Statistical analyses were performed using SPSS 25.0 statistical package. The results are expressed as mean ± SEM. The data was analyzed using student T- test. Any value of $P \leq 0.05$ was considered significant.

3. RESULTS AND DISCUSSION:

3.1. Liver functions, albumin and blood glucose:

Table1 showed the results of oral administration of SB at doses 300 mg/kg and 500 mg/kg on serum ALP, ALT, AST and ALB. As shown in the table, SB1 and SB2 groups showed a significant increase in ALP activity as compared to control group. Similarly, ALT and AST activities recorded a significant increase after SB administration. ALT activity recorded 120 ± 2.3 and 142 ± 3.3 U/L in SB1 and SB2 respectively as compared to 85.4 ± 6.4 U/L in control group. AST activity increased after SB administration, it recorded about 175 ± 4.9 and 179 ± 5.5 U/L in SB1 and SB2 respectively. Albumin concentration was significantly decreased after SB treatment, SB1 and SB2 recorded about 23 ± 0.6 and 20 ± 0.7 g/L respectively as compared to 32.7 ± 0.7 g/L in control group. These findings are in line with earlier studies that showed that SB damages liver tissue and has a direct toxic effect on hepatocytes, causing them to degenerate [14,15,16]. That elevation in aminotransferases activity can be related to alterations in the liver and disturbance of the hepatocytes, causing higher than normal amounts of intracellular enzyme to leak [17,18]. The extent of this leakage is a reflection of the extent of damage to body tissues, particularly the liver. This increase can also be accounted for by the generation of free radicals, which interact with polyunsaturated fatty acids in cellular membranes to rupture mitochondrial and plasma membranes and release enzymes. Hepatocellular damage caused by SB toxic activity manifested as vacuolation, swelling, and necrosis of liver cells [19]. The current investigation demonstrated that serum albumin level was reduced by benzoate. This was probably attributed to inadequate protein synthesis in many tissues of the treated rats, which was brought on by increased oxidative stress [21,22]. According to Hassan and Yousef [23], some food additives have an inhibitory effect on the synthesis of protein and albumin, suggesting that the liver cannot carry out its duties. This may be attributed to sodium benzoate's alteration of the liver's function.

Glucose concentration was elevated after SB administration, the increase recorded about 120 ± 4.03 and 129 ± 4.3 mg/dL in SB1 and SB2 respectively compared to 95.41 ± 2.21 mg/dL in the control. The activation of glycogenolysis and gluconeogenesis by the liver can be used to explain the rise in blood glucose levels [20].

3.2. Kidney functions:

As shown in table 1, SB1 and SB2 showed a significant increase in creatine level as compared to control group, it recorded about 0.73 ± 0.08 and 0.9 ± 0.07 mg/dL respectively. Similarly, urea conc. showed a significant increase after SB treatment, SB1 and SB2 recorded about 42.3 ± 1.7 and 43.3 ± 1.5 respectively as compared to 35.5 ± 1.9 mg/dL in control group. These findings were in line with [24,25,26]. This possibly as a result of the metabolites produced by the metabolism of dietary additives that affect kidney tissues. The amount of urea and creatinine that the body excretes is regulated by serum glycine levels, which are altered by SB. High sodium levels also accelerate the loss of glycine. Reduced blood glycine affects how quickly urea and creatinine are released. Lower glycine levels reduce the amount of creatinine and urea produced in the urine, which causes these compounds to build up and increase in blood levels [27]. Increased uric acid levels in rats given SB show that the kidneys are not functioning properly and that the kidney tissues have been harmed [28,29].

3.3. Lipid profile:

The impact of oral treatment for SB on lipid profile was displayed in table 2. SB treated rats had significantly higher levels of TG, TC, LDL, and vLDL ($P < 0.05$) than rats in the control group, but significantly lower ($P < 0.05$)

HDL levels. Our findings concur with those of [24,30]. The increase in total cholesterol may be caused by the release of free fatty acids from adipose tissue into the bloodstream, which raises the level of acetyl CoA and increases the synthesis of cholesterol, or it may be brought on by the peroxidation of lipids in cell membranes [31].

3.4. Lipid peroxidation and Glutathione:

As shown in table 3, SB treated groups showed a significant decrease in RBCs count, PCV, and Hb concentration as compared to the control group. Because malondialdehyde (MDA), a final metabolite of membrane peroxidation, is a frequently used to quantify lipid peroxidation. The presented data in table 4 showed MDA level was significantly increased in both groups SB1 and SB2. The corresponding value of MDA was 120.3 ± 4.4 and 144.6 ± 4.9 in SB1 and SB2 groups respectively as compared to 85.7 ± 3.5 nmol/g tissue in the control group. On the other hand, liver GSH concentration was decreased after SB administration. SB1 and SB2 recorded about 41.2 ± 3.4 and 30.9 ±2.5 µmol/g as compared to 62.8 ± 3.5 µmol/g in control group. Our experimental outcomes are in same opinion with [32- 34]. The peroxidation of biological membranes, which occurs under severe clinical situations, alters the fluidity and degrades the membrane. There is insufficient literature associated with this issue, so we were unable to find relevant information on oxidative stress induced by any food preservative.

The reaction between sodium benzoate and vitamin C results in the production of benzene, which has been linked to a number of disorders and is thought to be carcinogenic [35,36]. Reactive free radicals may develop as a result of the potential carcinogenic nature of the metabolic byproducts of sodium benzoate. As a result of the generation of free radicals, the cell's antioxidant components including GSH are consumed in order to prevent cell damage from these harmful molecules, which lowers their concentrations in the tissue. Increased free radical production and decreased antioxidant parameter activity can lead to auto-oxidation of liver tissue and significant hepatic diseases.

Table 1. Effect of 3 weeks oral administration of 300mg/kg and 500mg/kg sodium benzoate on serum ALP, ALT, AST, ALB, CREA, UREA, U. ACID, and glucose in adult male albino rats.

Groups / Parameters	Control group	SB1 group (300 mg /kg)	SB2 group (500 mg/kg)
ALP (U/L)	180 ± 20	240 ± 13.5*	265 ± 14.2**
% of change		33%	47%
ALT (U/L)	85.4 ± 6.4	120 ± 2.3**	142 ± 3.3**
% of change		40.5%	66%
AST (U/L)	150 ± 5.5	175 ± 4.9*	179 ± 5.5*
% of change		16.6%	19.3%
ALB (g/L)	32 ± 0.7	23 ± 0.6*	20 ± 0.7**
% of change		-28%	-37.5%
CREA (mg/dL)	0.49 ± 0.03	0.73 ± 0.08*	0.9 ± 0.07**
% of change		48.9%	83.6%
UREA (mg/dL)	35.5 ± 1.9	42.3 ± 1.7*	43.3± 1.5*
% of change		16.3%	19.2%
U. ACID (mmol/L)	1.3 ± 0.15	1.4 ± 0.2	1.5 ± 0.4
% of change		7.7%	15.4%
GLU (mg/dL)	95.41 ± 2.21	120 ± 4.03*	129 ± 4.3*
% of change		25.7%	35.2 %

Values represent mean ± SEM. (P* < 0.05, P*<0.05, P**<0.01 as compared to control group).

Table 2. Effect of 3 weeks oral administration of 300 mg/kg and 500 mg/kg sodium benzoate on serum TC, TG, HDL-C, LDL-C and vLDL-C in adult male albino rats

Groups / Parameters	Control group	SB1 group (300 mg /kg)	SB2 group (500 mg/kg)
Total Cholesterol, TC (mg/dl)	75.1 ± 1.6	95.5 ± 2.5*	100.2 ± 2.2*
% of change		27%	33%
Triglycerides, TG (mg/dL)	70.2 ± 1.9	100.3 ± 2.6**	104.5 ± 2.3**
% of change		43%	49%
HDL-C (mg/dL)	40.6 ± 1.5	31.2 ± 1.2*	27.5 ± 1.5*
% of change		-23 %	-32 %
LDL-C (mg/dL)	22.1 ± 1.7	35.3 ± 1.6*	39.4 ± 1.9**
% of change		60 %	78 %
vLDL (mg/dL)	14.4 ± 1.3	20.4 ± 1.8*	23.6 ± 1.7**
% of change		42%	64 %

Values represent mean ± SEM. (P* < 0.05, P*<0.05, P**<0.01 as compared to control group).

Table (3): Effect of 3 weeks oral administration of 300mg/kg and 500mg/kg sodium benzoate on RBCs, WBCs, Hb, and PCV in male albino rats.

Groups / Parameters	Control group	SB1 group (300 mg /kg)	SB2 group (500 mg/kg)
RBCs (10^6/µL)	8.21 ± 0.59	7.5 ± 0.7	6.44 ± 0.49*
% of change		-8%	-21%
WBCs (10^3/µL)	8.3 ± 0.48	7.9 ± 0.87	7.2 ± 0.39
% of change		-5%	-13%
Hb (g/dL)	11.57 ± 0.43	9.9 ± 1	8.67 ± 0.49*
% of change		-14%	-25%
PCV (%)	41.40 ± 2.75	37.6 ± 3.2	33.40 ± 2.7*
% of change		-9%	-18%

Values represent mean ± SEM. (P* < 0.05, P*<0.05, P**<0.01 as compared to control group).

Table (4): Effect of 3 weeks oral administration of 300mg/kg and 500mg/kg sodium benzoate on hepatic MDA and GSH in male albino rats.

Groups / Parameters	Control group	SB1 group (300 mg /kg)	SB2 group (500 mg/kg)
MDA (nmol/g tissue)	85.7 ± 3.5	120.3 ± 4.4*	144.6 ± 4.9**
% of change		40.4 %	68.7 %
GSH (µmol/g tissue	62.8 ± 3.5	41.2 ± 3.4*	30.9 ± 2.5**
% of change		-34.4 %	-50.7 %

Values represent mean ± SEM. (P* < 0.05, P*<0.05, P**<0.01 as compared to control group).

4. CONCLUSION

The high dosage of SB has a major adverse effect that affects kidney function, antioxidant status, liver enzymes, and lipid profile. To protect human health, it is crucial to control the irregular intake of SB's preserved or processed foods and to do further research to find safer and/or natural food additives.

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الملخص العربي

تأثير أحد المواد الحافظة على بعض المعايير الفسيولوجية في عينة من ذكور الجرذان البيضاء

بندر خالد حمزي[1] و محمد مصطفى سليمان[1]

[1]قسم الأحياء بكلية العلوم جامعة جازان

بنزوات الصوديوم ، ذات الخصائص المضادة للبكتيريا والفطريات، هي مادة حافظة شائعة الاستخدام في كلاً من الأغذية والأدوية. تشير بعض الدراسات إلى خطورة أستخدام بنزوات الصوديوم كمادة حافظة. أُجريت هذه الدراسة لمعرفة

تاثير أستخدام جرعات مختلفة من البنزوات على بعض العلامات الفسيولوجية. تم في هذا البحث استخدام خمسة عشر فأراً ذكراً. وتم تقسيمهم إلى ثلاث مجموعات رئيسية متساوية: حصلت المجموعة الأولى على محلول ملحي وأُستخدمت كمجموعة ضابطة؛ تلقت المجموعات الأخرى جرعات ٣٠٠ و ٥٠٠ ملجم / كجم على التوالي يومياً لمدة ثلاثة أسابيع. تم التضحية بالحيوانات في نهاية التجربة و تم أخذ عينات من الكبد والدم. تم تحليل مؤشرات الدم: الجلوكوز، وظائف الكلى (الكرياتين، اليوريا، وحمض البوليك)، إنزيمات الكبد (أسبارتات أمينو ترانسفيريز، ألانين أمينو ترانسفيريز، الفوسفاتيز القلوي)، الألبومين، الدهون (الكوليسترول الكلي، الدهون الثلاثية، البروتين الدهني منخفض الكثافة، البروتين الدهني عالي الكثافة)، و الجلوتاثيون والمالون ألدهيد في جميع المجموعات. أظهرت النتائج أن تناول بنزوات الصوديوم أدى إلى زيادة ملحوظة في نشاط أنزيمات الكبد و زيادة فى مستوى الكرياتينين ،اليوريا ، الجلوكوز ، الدهون الثلاثية ، الكوليسترول الكلي، البروتين الدهني منخفض الكثافة و مركب المالون ألدهيد فى نسيج الكبد. علاوة على ذلك، أدت الجرعة العالية من بنزوات الصوديوم إلى انخفاض ملحوظ في عدد كرات الدم الحمراء، تركيز الهيموجلوبين و الهيماتوكريت و مركب الجلوتاثيون بالكبد. من النتايج السابقة يمكن أستنتاج أن مادة بنزوات الصوديوم الحافظة لها آثار ضارة على الكبد والكلى وربما حتى بعض مؤشرات الدموية.

الكلمات المفتاحية: بنزوات الصوديوم، انزيمات الكبد، وظائف الكلي،الدهون