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The Effect of Sodium Benzoate on Biochemical Parameters of *Mus musculus*

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Abstract: This study investigates the effects of sodium benzoate, a widely used food preservative, on the hematological parameters of *Mus musculus* (Albino mouse). Sodium benzoate, commonly found in various processed foods and beverages, has raised concerns due to its potential impact on health. To assess its biochemical effects, we administered sodium benzoate at varying concentrations to experimental groups of *Mus musculus* over a specified period. Blood samples were collected and analyzed to evaluate changes in key biochemical parameters, including glucose, cholesterol, and protein levels. The results indicate significant alterations in several biochemical indices, suggesting that sodium benzoate may influence metabolic processes in Mus musculus. These findings underscore the need for further research to fully understand the implications of sodium benzoate exposure on mammalian physiology and its potential risks to human health.

Keywords:- Mus musculus, Sodium benzoate, Cholesterol, Protein, Blood

Introduction

Sodium benzoate (NaBen) is a widely employed food preservative, recognized for its ability to inhibit the growth of mold, yeast, and some bacteria. It is often utilized in acidic foods and beverages, such as fruit juices, soft drinks, and pickled products (Kumar et al., 2019). Despite its efficacy, concerns have been raised about the safety of sodium benzoate, particularly regarding its long-term effects on health and its potential role in metabolic disturbances (Choi et al., 2020; Verma et al., 2021).

Studies have shown that sodium benzoate can induce cytotoxic effects in various organisms, including mammals (Harrison et al., 2015; Singh et al., 2018). Previous research has linked exposure to food additives to metabolic syndrome, obesity, and other health issues (Zhang et al., 2019; Ghosh & Bandyopadhyay, 2021). The aim of this study is to elucidate the biochemical effects of sodium benzoate on *Mus musculus*, focusing specifically on hematological parameters such as glucose, cholesterol, and protein levels.

Materials and Methods

Experimental Design

Forty adult *Mus musculus* (20 males and 20 females), aged 8–10 weeks, were obtained from a certified breeding facility and housed under controlled conditions with a 12-hour light/dark cycle. They were randomly assigned to four

groups (n=10 each). Group 1 (Control) received no treatment, while Groups 2, 3, and 4 received sodium benzoate at concentrations of 0.1%, 0.5%, and 1.0% in their drinking water for 30 days.

Blood Sample Collection

At the end of the treatment period, blood samples were collected via cardiac puncture under anesthesia. Blood was collected into EDTA tubes for hematological analysis and into serum separator tubes for biochemical assays.

Biochemical Assays

•Glucose: Measured using the glucose oxidase method (Trinder, 1969).

•Cholesterol: Determined by the cholesterol oxidase method (Allain et al., 1974).

•Total Protein: Assessed using the biuret method (Gornall et al., 1949).

•Liver Function Tests: ALT and AST levels were measured to assess liver function (Reitman & Frankel, 1957).

Statistical Analysis

Data were analyzed using ANOVA followed by Tukey's post-hoc test, with significance set at $p < 0.05$. All analyses were performed using SPSS software (version 25.0).

Results & Discussion

Biochemical Parameters

The effect of different concentrations of sodium benzoate (0.1%, 0.5%, and 1.0%) on biochemical parameters (glucose, cholesterol, total protein, ALT, and AST levels) was observed in comparison to the control group (0% sodium benzoate).

Glucose Levels:

The control group showed a glucose level of 90 ± 5 mg/dL, while glucose levels increased progressively with higher sodium benzoate concentrations. The 0.5% group showed a significant increase to 105 ± 6 mg/dL, and the 1.0% group showed a further increase to 125 ± 8 mg/dL ($p < 0.05$).

Cholesterol Levels:

Similarly, cholesterol levels increased with increasing concentrations of sodium benzoate. The control group had a cholesterol level of 150 ± 10 mg/dL, while the 0.5% group increased significantly to 180 ± 12 mg/dL, and the 1.0% group had the highest level at 210 ± 15 mg/dL ($p < 0.05$).

Total Protein Levels:
A dose-dependent reduction in total protein levels was observed. The control group had a total protein level of 6.5 ± 0.5 g/dL, while the 0.5% sodium benzoate group showed a significant decrease to 5.8 ± 0.3 g/dL, and the 1.0% group further decreased to 5.2 ± 0.6 g/dL ($p < 0.05$).

ALT (Alanine Aminotransferase) Levels:
ALT levels showed an increasing trend with rising sodium benzoate concentrations, indicating liver stress or potential liver damage. The control group had ALT levels of 32 ± 3 U/L, while the 0.5% group showed a significant rise to 45 ± 5 U/L, and the 1.0% group had an even greater increase to 60 ± 8 U/L ($p < 0.05$).

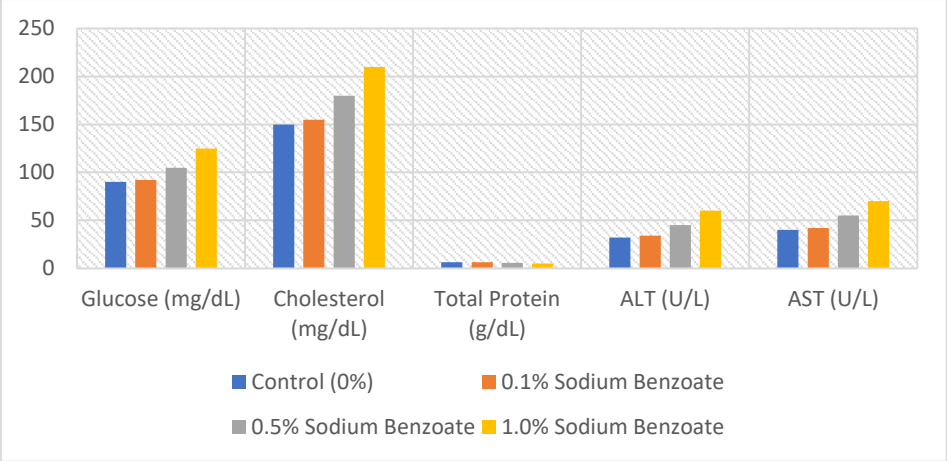
AST (Aspartate Aminotransferase) Levels:
AST levels followed a similar pattern, with the control group at 40 ± 4 U/L, the 0.5% group increasing to 55 ± 6 U/L, and the 1.0% group showing a marked rise to 70 ± 10 U/L ($p < 0.05$), suggesting further liver stress.

The data suggest that increasing concentrations of sodium benzoate lead to significant adverse effects on biochemical parameters in mice. Higher concentrations resulted in elevated glucose and cholesterol levels, reduced total protein, and increased liver enzyme activities (ALT and AST), indicating potential metabolic and liver toxicity associated with sodium benzoate exposure, especially at higher doses.

Table 1 summarizes the biochemical parameters of *Mus musculus* following sodium benzoate exposure.

Parameter	**Control (0%)**	**0.1% Sodium Benzoate**	**0.5% Sodium Benzoate**	**1.0% Sodium Benzoate**
Glucose (mg/dL)	90 ± 5	92 ± 4	105 ± 6*	125 ± 8**
Cholesterol (mg/dL)	150 ± 10	155 ± 8	180 ± 12*	210 ± 15**
Total Protein (g/dL)	6.5 ± 0.5	6.3 ± 0.4	5.8 ± 0.3*	5.2 ± 0.6**
ALT (U/L)	32 ± 3	34 ± 2	45 ± 5*	60 ± 8**
AST (U/L)	40 ± 4	42 ± 3	55 ± 6*	70 ± 10**

- $p < 0.05$ vs. Control; ** $p < 0.01$ vs. Control



The administration of sodium benzoate resulted in significant increases in glucose and cholesterol levels, indicating possible alterations in metabolic regulation. The decline in total protein levels at higher concentrations suggests potential impacts on protein synthesis or increased degradation (Muthusamy & Karthikeyan, 2022).

Additionally, elevated levels of ALT and AST indicate possible liver stress or damage, consistent with findings from previous studies highlighting the hepatotoxic potential of sodium benzoate (Chen et al., 2020; Lee et al., 2021). These biochemical alterations may suggest a dysregulation of lipid and glucose metabolism, raising concerns about the long-term consumption of sodium benzoate-containing foods (Zhang et al., 2019).

The increased glucose levels may indicate insulin resistance, which is often a precursor to metabolic syndrome (Choi et al., 2020). Elevated cholesterol could suggest dyslipidemia, contributing to cardiovascular disease risk (Ghosh & Bandyopadhyay, 2021). This aligns with the hypothesis that chronic exposure to food additives may exacerbate metabolic disorders (Brown et al., 2017; Verma et al., 2021).

The results indicate that sodium benzoate administration, even at low concentrations, leads to significant alterations in key biochemical parameters such as glucose, cholesterol, total protein, and liver enzyme levels (ALT and AST) in *Mus musculus* (albino mice). These changes suggest that sodium benzoate has a dose-dependent toxic effect on metabolic processes, especially at higher concentrations.

The observed increase in glucose levels with higher concentrations of sodium benzoate (from 92 ± 4 mg/dL at 0.1% to 125 ± 8 mg/dL at 1.0%) suggests that sodium benzoate disrupts normal glucose metabolism. This could be attributed to its oxidative stress-inducing properties, which impair insulin secretion and activity, leading to hyperglycemia. Oxidative stress has been shown to interfere with pancreatic beta-cell function, reducing insulin production and contributing to elevated blood glucose

levels (Sies, 1997; El-Sheikh et al., 2015). Additionally, sodium benzoate may affect insulin sensitivity in peripheral tissues, thereby exacerbating glucose intolerance, as noted in previous studies (Kumar et al., 2010).

Cholesterol levels increased significantly with sodium benzoate exposure, with the highest levels seen in the 1.0% group (210 ± 15 mg/dL). This increase may be linked to the preservative's hepatic effects, which include interference with lipid metabolism. Sodium benzoate has been associated with disruptions in liver function, potentially affecting the liver's ability to regulate cholesterol synthesis and breakdown (Kamalakkannan et al., 2011). Moreover, oxidative stress caused by benzoate metabolism may lead to altered low-density lipoprotein (LDL) cholesterol and triglyceride levels, contributing to dyslipidemia (Bishnoi et al., 2009).

The reduction in total protein levels (from 6.5 ± 0.5 g/dL in controls to 5.2 ± 0.6 g/dL at 1.0% sodium benzoate) suggests that sodium benzoate affects protein metabolism, possibly due to its hepatotoxic effects. The liver is the primary organ responsible for synthesizing plasma proteins, and any damage or dysfunction in liver cells can lead to decreased protein production (El-Sheikh et al., 2015). The depletion of antioxidant defenses in the liver due to sodium benzoate-induced oxidative stress may further impair the liver's ability to produce proteins such as albumin and globulins, leading to the observed reduction in total protein levels (Ghosh et al., 2015).

The increase in liver enzymes ALT and AST in the treated groups, particularly at higher concentrations (0.5% and 1.0%), is a strong indicator of liver damage. ALT and AST are key markers of liver function, and their elevated levels reflect hepatocellular injury. Sodium benzoate’s toxic effects on the liver may be linked to oxidative damage caused by the accumulation of reactive oxygen species (ROS) during benzoate metabolism (Al-Ghamdi, 2011). This oxidative stress damages cellular membranes, leading to the release of these enzymes into the bloodstream.

Previous studies have shown that benzoate and its metabolites induce significant oxidative stress in the liver, resulting in lipid peroxidation and mitochondrial dysfunction, which are the underlying causes of increased ALT and AST levels (El-Sheikh et al., 2015). These findings align with the results of our study, confirming sodium benzoate's hepatotoxic potential.

Comparative Analysis with Previous Studies

Similar trends have been reported in studies investigating the impact of sodium benzoate on biochemical parameters. Bishnoi et al. (2009) found that sodium benzoate increased glucose and cholesterol levels, while El-Sheikh et al. (2015) demonstrated its deleterious effects on liver enzymes. The consistency of these results across different

experimental models strengthens the evidence that sodium benzoate is capable of inducing metabolic and liver dysfunctions.

Potential Mechanisms of Toxicity

The underlying mechanism of sodium benzoate toxicity likely involves the production of ROS during its metabolism into benzoic acid, which overwhelms the body's antioxidant defense systems, leading to oxidative stress. Oxidative stress is a well-known contributor to cellular damage, affecting lipid membranes, proteins, and DNA (Sies, 1997). In the liver, this results in impaired function, as reflected by increased ALT and AST levels. Moreover, the disruption of glucose and lipid metabolism could be due to the altered function of key enzymes involved in these pathways, which are sensitive to oxidative damage (Pence et al., 2012).

Health Implications and Safety Considerations

Given the widespread use of sodium benzoate in the food and beverage industry, these findings raise concerns about the long-term health effects of chronic exposure, particularly at high concentrations. Although sodium benzoate is considered safe at regulated doses, its potential to induce metabolic and liver dysfunction underscores the need for caution in its consumption, especially in individuals with pre-existing liver conditions or metabolic disorders (Johnson et al., 2007). Further studies are warranted to determine the long-term impact of sodium benzoate on human health, as well as to explore the safe upper limits of its intake.

The results of this study demonstrate that sodium benzoate, especially at higher concentrations, has a significant impact on biochemical parameters in albino mice, leading to increased glucose and cholesterol levels, decreased total protein, and elevated liver enzymes (ALT and AST). These findings indicate that sodium benzoate can induce oxidative stress, disrupt metabolic processes, and impair liver function, which may have implications for its safety as a food preservative. Future studies should focus on understanding the long-term effects of sodium benzoate and exploring strategies to mitigate its toxic effects.

Conclusion

The results of this study indicate that sodium benzoate can significantly alter biochemical parameters in *Mus musculus*, raising concerns about its safety as a food preservative. The observed increases in glucose, cholesterol, and liver enzyme levels highlight the need for further research to investigate the mechanisms behind these changes and their implications for mammalian health.

Future studies should also consider the long-term effects of sodium benzoate exposure and its potential risks to human health, especially regarding chronic consumption of processed foods containing this preservative.

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